STACKS

NEW YORK STATE JOURNAL OF MEDICINE

JANUARY 1987 • Volume 87, Number 1

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

LIBRARY OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA
M.D.S.
JAN 27 1987

Contents

COMMENTARIES

New York's homeless 1
PASCAL JAMES IMPERATO, MD

Homelessness in New York City: The youngest victims 3
STEN H. VERMUND, MD, MSc; ROBERTO BELMAR, MD; ERNEST DRUCKER, PhD

Implications of the recent clinical trials in hypertension 5
MARVIN MOSER, MD

Azidothymidine and human immunodeficiency virus infection 7
JACK A. DeHOVITZ, MD, MPH

RESEARCH PAPER

eproductive experience of women in hotels for the homeless in York City 10
IDY CHAVKIN, MD, MPH; ALAN STAL, MS, DrPH; CHERYL SEABRON, BS; ELA E. GUIGLI, MPH

Library of the College Physicians
of Philadelphia
19 S. 22nd St.
Phila, PA 19103
Dec. 87

Y AMERICAN MEDICINE

l and artifactual materials in the y of early American medicine 14
ERT I. GOLER, MA

n healers and domestic remedies in entury America: The recipe book of eth Coates Paschall 23
N G. GARTRELL, AM, MS

pact of smallpox on the native tion of the 18th century South 30
R H. WOOD, PhD

ns of drug usage in colonial America 37
RTH ESTES, MD

CASE REPORTS

A subglottic foreign body in an adult patient 46
RAMIRO REQUENA, MD; RAMESHBHAI PATEL, MD; INDERJIT SINGH, MD

Cystic artery pseudoaneurysm 47
JOHN W. RHEE, MD; DAVID C. BONNHEIM, MD; JAMES UPSON, MD

Mesonephric hamartoma of the seminal vesicle: A rare cause of a retrovesical mass 48
HELEN KINAS, MD, MATTHEW J. KUHN, MD

Choledochal cyst in an adult 49
NANAKRAM AGARWAL, MD; BENEDICT M. REYNOLDS, MD

LETTERS TO THE EDITOR

Cigarette advertising 52
MUHAMMAD ABU HENA, MD

Medical liability premiums in Great Britain 52
J. RICHARD TYNER, MD

Primary retroperitoneal hydatid cyst 52
AJAY K. KRIPLANI, MS

ESSAY REVIEW

The Healing Arts in Early America 54
PASCAL JAMES IMPERATO, MD

BOOK REVIEWS 57
BOOKS RECEIVED 61
NEWS BRIEFS 63
OBITUARIES 65
GUIDELINES FOR AUTHORS 68

HOUSE OF DELEGATES

Sessions, Officers, Councilors, and Trustees; Members; Nominating and Reference Committees; Report of the House Committee on Bylaws 69

MEDICAL MEETINGS AND LECTURES 4A

Annual CME Assembly, March 13–15, 1987, New York Hilton, New York City
(See December 1986 issue for Preliminary Program)

Motrin® 800 TABLETS mg

ibuprofen

Extra strength

Convenience

Economy

Upjohn

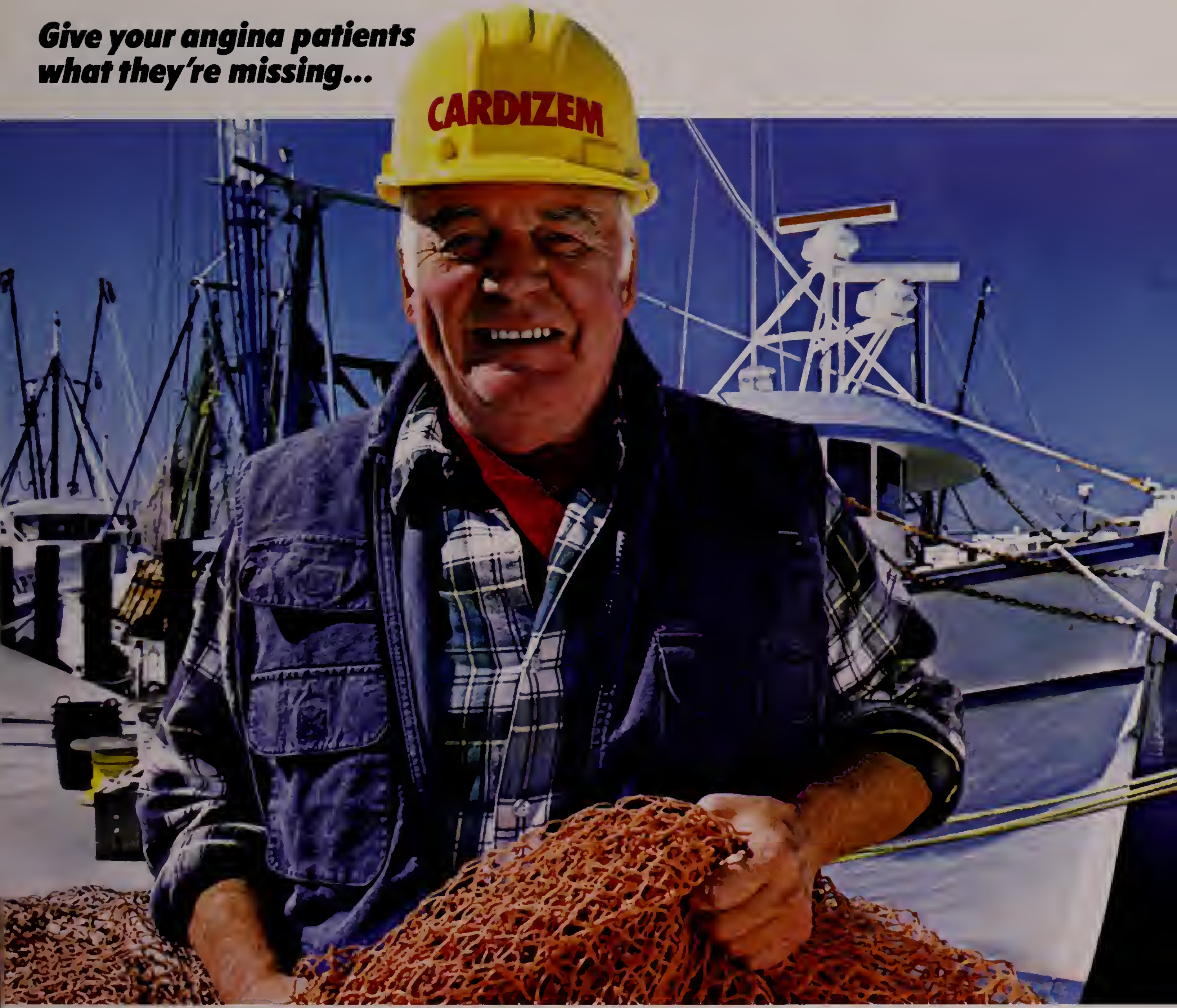
A Century of Caring

1886·1986

©1986 The Upjohn Company

J-6138 January 1986

Give your angina patients what they're missing...



CARDIZEM® : FEW SIDE EFFECTS

diltiazem HCl/Marion

- ***Antianginal action includes dilatation of coronary arteries, a decrease in vascular resistance/afterload, and a reduction in heart rate***
- ***Proven efficacy when used alone in angina[1]***
- ***Compatible with other antianginals[2,3]****
- ***A safe choice for angina patients with coexisting hypertension, asthma, COPD, or PVD[4,5]***

***See Warnings and Precautions.**

Please see brief summary of prescribing information on the next page.

CARDIZEM®
diltiazem HCl/Marion

FEW SIDE EFFECTS IN ANTIANGINAL THERAPY

60 mg tid or qid

Brief Summary
Professional Use Information

CARDIZEM®
(diltiazem HCl) 30 mg and 60 mg Tablets

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. ***Cardiac Conduction.*** *CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1,243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.*
2. ***Congestive Heart Failure.*** *Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt).*
   Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. ***Hypotension.*** *Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.*
4. ***Acute Hepatic Injury.*** *In rare instances, significant elevations in enzymes such as alkaline phosphatase, CPK, LDH, SGOT, SGPT, and other symptoms consistent with acute hepatic injury have been noted. These reactions have been reversible upon discontinuation of drug therapy. The relationship to CARDIZEM is uncertain in most cases, but probable in some. (See PRECAUTIONS.)*

PRECAUTIONS

General. *CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.*

Drug Interaction. *Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)*

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. *A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.*

Pregnancy. *Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.*

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. *Diltiazem is excreted in human milk. One report suggests that concentrations in breast milk may approximate serum levels. If use of CARDIZEM is deemed essential, an alternative method of infant feeding should be instituted.*

Pediatric Use. *Safety and effectiveness in children have not been established.*

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences as well as their frequency of presentation are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%). In addition, the following events were reported infrequently (less than 1%):

Cardiovascular:	*Angina, arrhythmia, AV block (first degree), AV block (second or third degree — see conduction warning), bradycardia, congestive heart failure, flushing, hypotension, palpitations, syncope.*
Nervous System:	*Amnesia, gait abnormality, hallucinations, insomnia, nervousness, paresthesia, personality change, somnolence, tinnitus, tremor.*
Gastrointestinal:	*Anorexia, constipation, diarrhea, dysgeusia, dyspepsia, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH (see hepatic warnings), vomiting, weight increase.*
Dermatologic:	*Petechiae, pruritus, photosensitivity, urticaria.*
Other:	*Amblyopia, dyspnea, epistaxis, eye irritation, hyperglycemia, nasal congestion, nocturia, osteoarticular pain, polyuria, sexual difficulties.*

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: alopecia, gingival hyperplasia, erythema multiforme, and leukopenia. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established. *Issued 7/86*

See complete Professional Use Information before prescribing.

References: 1. *Pepine CJ, Feldman RL, Hill JA, et al: Clinical outcome after treatment of rest angina with calcium blockers: Comparative experience during the initial year of therapy with diltiazem, nifedipine, and verapamil.* *Am Heart J 1983, 106(6):1341-1347.* ***2.*** *Shapiro W: Calcium channel blockers. Actions on the heart and uses in ischemic heart disease.* *Consultant 1984;24(Dec):150-159.* ***3.*** *Johnston DL, Lesoway R, Humen DP, et al: Clinical and hemodynamic evaluation of propranolol in combination with verapamil, nifedipine and diltiazem in exertional angina pectoris: A placebo-controlled, double-blind, randomized, crossover study.* *Am J Cardiol 1985;55:680-687.* ***4.*** *Cohn PF, Braunwald E: Chronic ischemic heart disease, in Braunwald E (ed): Heart Disease: A Textbook of Cardiovascular Medicine, ed 2. Philadelphia, WB Saunders Co, 1984, chap 39.* ***5.*** *Schroeder JS: Calcium and beta blockers in ischemic heart disease: When to use which.* *Mod Med 1982;50(Sept):94-116.*

Another patient benefit product from
M PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC.
KANSAS CITY, MO 64137

0246M6

NEW YORK STATE JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
FRANK C. MAXON, JR, MD
ELIZA H. CALDWELL, MD
JOHN T. PRIOR, MD
STANFORD WESSLER, MD

Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Consulting Editor and Book Review Editor	RICHARD B. BIRRER, MD
Consulting Editor	NAOMI R. BLUESTONE, MD, MPH
Consulting Editor	CARL POCHEDLY, MD
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Managing Editor	CAROL L. MOORE
Librarian	ELLA ABNEY
Assistant Librarian	ELEANOR BURNS
Consulting Medical Writer	VICKI GLASER
Editorial Assistant	MILDRED J. ARFMANN
Secretary	ELIZABETH J. SOMERS
Advertising Director	ROBERT MILLER

ASSOCIATE EDITORIAL BOARD 1986

MICHAEL E. BERLOW, MD
RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MYLES S. GOMBERT, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
JAMES M. MORRISSEY, ESQ
STEPHEN NORDLICHT, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. United Media Associates, Inc, 16 Bruce Park Ave, Greenwich, CT 06830. Telephone: (203) 661-9702.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

JOHN A. FINKBEINER, MD,	*President*
DANIEL F. O'KEEFFE, MD,	*Past-President*
SAMUEL M. GELFAND, MD,	*President-Elect*
CHARLES D. SHERMAN, MD,	*Vice-President*
JOHN H. CARTER, MD,	*Secretary*
GEORGE LIM, MD,	*Assistant Secretary*
MORTON KURTZ, MD,	*Treasurer*
ROBERT A. MAYERS, MD,	*Assistant Treasurer*
CHARLES N. ASWAD, MD,	*Speaker*
SEYMOUR R. STALL, MD,	*Vice-Speaker*

Councilors

Term Expires 1987

DAVID M. BENFORD, MD,	New York
DUANE M. CADY, MD,	Onondaga
JAMES M. FLANAGAN, MD,	Wayne
M. THEODORE TANENHAUS, MD,	Kings
KATHLEEN E. SQUIRES, MD,	New York

(*Councilor representing the resident physician membership*)

FRANCES HARTENSTEIN-COLLICHIO,	Monroe

(*Councilor representing the medical student membership*)

Term Expires 1988

JAMES H. COSGRIFF, JR, MD,	Erie
RICHARD A. HUGHES, MD,	Warren
ANTONIO F. LASORTE, MD,	Broome
SIDNEY MISHKIN, MD,	Nassau

Term Expires 1989

ROBERT E. FEAR, MD,	Suffolk
STANLEY L. GROSSMAN, MD,	Orange
THOMAS D. PEMRICK, MD,	Rensselaer
RALPH E. SCHLOSSMAN, MD,	Queens

Trustees

RALPH M. SCHWARTZ, MD,	Kings
EDGAR P. BERRY, MD,	New York
RICHARD D. EBERLE, MD,	Onondaga
JOSEPH F. SHANAPHY, MD,	Richmond
VICTOR J. TOFANY, MD,	Monroe
BERNARD J. PISANI, MD,	New York
DANIEL F. O'KEEFFE, MD,	Warren

Executive Vice-President	DONALD F. FOY
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Vice-President Emeritus	GEORGE J. LAWRENCE, JR, MD
Director, Division of Policy Coordination	IRMA A. ERICKSON
Director, Division of Scientific Publications	PASCAL JAMES IMPERATO, MD
Director, Division of Communications	EDWARD A. HYNES
Director, Division of Computer Services	IVAN H. NAJMAN
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Governmental Affairs General Counsel	GERARD L. CONWAY, ESQ
Director, Division of Management and Meeting Services	ROBERT MILLER
Director, Division of Membership Support Services	EUNICE M. SKELLY
Director, Division of Scientific, and Educational Activities	PETER B. FARNSWORTH, MD
Director, Division of Socio-Medical Economics	MARIO V. MENGHINI
Medical Director, Committee for Physicians' Health	BARTON A. HARRIS, MD
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly, with a special issue added in April, by the Medical Society of the State of New York.

Copyright © 1986, Medical Society of the State of New York. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meetings and lectures unless the information is submitted at least three months prior to the event.*

FEBRUARY 1987

AROUND THE STATE

MANHATTAN

Feb 7. **Pediatric Ophthalmology and Strabismus.** *Feb 13–14.* **Contact B & A Scar Ultrasonography for the Clinician.** *Feb 21.* **Basic Clinical Immunology for the Ophthalmologist.** Manhattan Eye, Ear & Throat Hospital. Contact: Martha Klapp, Manhattan Eye, Ear & Throat Hospital, 210 East 64 St, New York, NY 10021. Tel: (212) 605-3762.

LAKE PLACID

Feb 17–20. **Pediatric Orthopaedics for Primary Care Physicians.** Lake Placid Hilton. Info: Program Coordinator, Continuing Education, SUNY Health Science Center, 750 East Adams St, Syracuse, NY 13210. Tel: (315) 473-4304.

AROUND THE NATION

ARIZONA

Feb 27–Mar 1. **The American Society of Outpatient Surgeons 8th Annual Meeting.** Buttes, Tempe. Info: American Society of Outpatient Surgeons, PO Box 33185, San Diego, CA 92103. Tel: (619) 692-9918.

CALIFORNIA

Feb 2–6. **6th Annual UCSD Radiology Imaging and Intervention.** 21½ Cat 1 Credits. Hotel Intercontinental, San Diego. Contact: R. Dawne Ryals, Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

Feb 3–8. **16th Annual Pediatric Postgraduate Course.** 18 Cat 1 Credits. Rancho Las Palmas, Palm Springs. Info: Ann J. Boehme, Associate Director for Continuing Education, Schneider Children's Hospital of Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

Feb 11–13. **Recent Advances in Neurology.** 15½ Cat 1 Credits. Fairmont Hotel, San Francisco. *Feb 28.* **Major and Minor Hand Problems in the Emergency Dept.** 7 Cat 1 Credits. University of California, San Francisco. Info: University of California, Extended Programs in Medical Education, Room U-569, San Francisco, CA 94143. Tel: (415) 476-4251.

COLORADO

Feb 1–6. **Diagnostic Radiology Seminars.** 24½ Cat 1 Credits. The Gant, Aspen. Info: Radiology Postgraduate Education, University of California, Third & Parnassus Ave, Room C324, San Francisco, CA 94143. Tel: (415) 476-5731.

Feb 2–7. **Winter Skin Seminar.** 20 Cat 1 Credits. Sheraton at Steamboat. *Feb 14–18.* **Frontiers in Cardiac Care: Arythmias and Coronary Artery Disease.** 14 Cat 1 Credits. Marriott's Mark Resort at Vail. *Feb 15–20.* **New Approaches to Clinical Problems in Internal Medicine.** 24 Cat 1 Credits. Snowmass Conference Center. *Feb 22–28.* **Infectious Disease/Pulmonary Medicine.** 30 Cat 1 Credits. Given Institute, Aspen. *Feb 23–27.* **Family Practice Review.** 35 Cat 1 Credits. Denver. *Feb 28–Mar 7.* **Total Knee-Hip Arthroplasty.** 27 Cat 1 Credits. Snowmass Conference Center. Info: University of Colorado School of Medicine, Office of Continuing Medical Education, 4200 E 9th Ave, Box C-295, Denver, CO 80262. Tel: (303) 394-5195.

Feb 7–14. **Vail Symposium in Intensive Care.** 25 Cat 1 Credits. Marriott's Mark Resort in Vail. Info: Professional Seminars, PO Box 012318, Miami, FL 33101. Tel: (305) 547-6411.

Feb 15–20. **13th Stanford-Research Institute, Palo Alto Medical Foundation Winter Course in Infectious Diseases.** 23¼ Cat 1 Credits. Continental Inn, Aspen. *Feb 16–20.* **8th Annual Conference on Problems in Gastroenterology: A Clinical and Pathological Approach.** 24½ Cat 1 Credits. Keystone Resorts, Keystone. Contact: American College

(*continued on p 8A*)

LIBRIUM®
chlordiazepoxide HCl/Roche Ⓒ
5-mg, 10-mg, 25-mg capsules
Before prescribing, please consult complete product information, a summary of which follows:

Indications: Management of anxiety disorders; short-term relief of anxiety symptoms, acute alcohol withdrawal symptoms, preoperative apprehension and anxiety. Usually not required for anxiety or tension associated with stress of everyday life. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended.

Contraindications: Known hypersensitivity to the drug.

Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage. Withdrawal symptoms (including convulsions) reported after abrupt cessation of extended use of excessive doses are similar to those seen with barbiturates. Milder symptoms reported infrequently when continuous therapy is abruptly ended. Avoid abrupt discontinuation; gradually taper dosage.

> ***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically. Due to isolated reports of exacerbation, use with caution in patients with porphyria.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral—Adults:* Mild and moderate anxiety disorders and symptoms, 5 or 10 mg *t.i.d.* or *q.i.d.;* severe states, 20 or 25 mg *t.i.d.* or *q.i.d.* *Geriatric patients:* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)

Supplied: Librium® (chlordiazepoxide HCl/Roche) *Capsules,* 5 mg, 10 mg and 25 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in boxes of 4 reverse-numbered cards of 25, and in boxes containing 10 strips of 10. Libritabs® (chlordiazepoxide/Roche) *Tablets,* 5 mg and 10 mg—bottles of 100 and 500; 25 mg—bottles of 100. With respect to clinical activity, capsules and tablets are indistinguishable.

ROCHE®
Roche Products Inc.
Manati, Puerto Rico 00701

Do not substitute

Librium®

brand of

chlordiazepoxide HCl/Roche Ⓒ IV

Nobody does it better.

ROCHE®

Copyright © 1986 by Roche Products Inc. All rights reserved.

Please see adjacent page for summary of product information.

Classified Advertising

PHYSICIANS WANTED

ARIZONA BASED PHYSICIAN RECRUITMENT firm has opportunities Coast to Coast. "Professionals working with Professionals". Over 13 years experience. Call (602) 990-8080, or send CV to: Mitchell & Associates, Inc., P.O. Box 1804, Scottsdale, AZ 85252.

PRACTICE OPPORTUNITIES for full time and part time FP/IM/ER physicians in modern ambulatory care centers in Metropolitan, Buffalo. Competitive salary plus incentives and malpractice coverage. Replies: Eugene Turchetti, M.D., c/o Medical Center Consultants, 1875 Grand Island Blvd., Grand Island, NY 14072.

EMERGENCY MEDICINE POSITIONS. Full/part time emergency medicine physicians sought by multi-state professional association. Openings in metropolitan NY, NJ, PA, MD, DC, FL and throughout U.S. Contact or send CV to Liberty Healthcare Corporation, 399 Market Street, Suite 400, Philadelphia, PA 19106, (215) 592-7400 or outside PA (800) 331-7122.

GENERAL SURGERY—Solo General Surgical Practice looking for a young associate to begin Spring/Summer 1987. General and some vascular surgery. Delightful upstate location. Prefer candidates completing residency or with recent training. Send confidential letter and C.V. to P.O. Box 505, New Hartford, New York 13413.

DO YOU WANT TO SHARE A THRIVING DERMATOLOGY PRACTICE: Do you enjoy year around outdoor recreation? You should contact us—a progressive multi-specialty group of 68 physicians in a pleasant, growing community. We offer a liberal financial package and outstanding benefits. Send curriculum vitae and references to: Michael T. Anderson, M.D., Willmar Medical Center, 101 Willmar Ave. SW, Willmar, MN 56201.

PHYSICIANS needed for 75 children's private residential camps July and/or August. Contact: Association of Independent Camps (JM), 60 Madison Avenue, New York, NY 10010, (212) 679-3230.

BE A "WINTER TEXAN" INTERNIST. Enjoy the warm, beautiful Rio Grande Valley while practicing internal medicine with an internist. Texas license essential. Salary, living accommodations, and malpractice insurers. Send curriculum vitae to Luis F. Arango, M.D., P.A., 104 South Bryan Road, Mission, Texas 78572 or call (512) 585-2783 for more information.

PRACTICES AVAILABLE

GYN AND FERTILITY PRACTICE—Long established prime Nassau County office. Refers out 50+ OBs annually. Backlog of surgery for purchasing physician. Doctor will remain with practice. Countrywide, Inc., (212) 686-7902.

PRACTICES AVAILABLE—CONT'D

"SELLING PRACTICES IS OUR ONLY BUSINESS"

Want to maximize the return on your investment? Before you buy or sell, call for an appraisal to assure the best financial and transfer terms. We guide you through the entire sales process from initial meeting to closing. Serving NY, CT, NJ and MA.

Countrywide Business Brokerage, Inc.
319 East 24th St., Suite 23-G
New York, NY 10010
(212) 686-7902 (203) 869-3666

FAMILY PRACTICE AND OFFICE BUILDING in $3 million, 5 acre professional complex for sale. 1800 square feet, fully equipped, 5 exam rooms, beautifully landscaped. Room for two physicians. Located in Modesto with easy access to San Francisco. Available now. Contact Emile Lengyel, M.D., 4141 Carver Road, Modesto, CA 95356, (209) 522-2544.

MEDICAL PRACTICE SALES AND APPRAISALS. We specialize in the valuation and selling of medical practices. Listed are a few available practices: ALLERGY—Philadelphia (2) and Kansas; DERMATOLOGY—Pennsylvania and Kansas; FAMILY PRACTICE—Ohio, New Jersey, Virginia and Delaware; INTERNAL MEDICINE—Pennsylvania, Maryland and Arizona; OPHTHALMOLOGY—Illinois; PEDIATRICS—Pennsylvania; RADIOLOGY—Pennsylvania (2); UROLOGY—Arkansas. If interested in buying or selling a medical practice, contact our brokerage division at The Health Care Group, 400 GSB Bldg., Bala Cynwyd, PA 19004 or call (215) 667-8630.

PRACTICES WANTED

FAMILY PHYSICIAN, interested in purchasing practice on Long Island. Reply Dept. #430 c/o NYSJM.

EQUIPMENT

DISCOUNT HOLTER SCANNING SERVICES starting at $35.00. Spacelabs holter recorder (cassette) available from $1,275.00. Cardionostic holter recorder (cassette) available from $1,125.00. Smallest and lightest holters update. Fast service (24–48 hours) turn over. Hookup kits starting at $4.95. Special introductory offer of three free tests with any purchase or lease of the recorder. Cardiologist overread available for $15.00. If interested call (301) 870-3626.

USED MEDICAL EQUIPMENT FOR SALE. All in excellent condition: Labophot biological Microscope with video camera and monitor $2500; Nucleus Gamma Counter, 40-tube carosel $4000; Burdick electrocardiograph EK/5A with stand $1250; Burdick EKG EK/5A $950; Pharmacia Diagnostics Phadezym Analyzer $3500; Lyte-Tech Sodium-Potassium Analyzer $1200; Clay Adams Enzak Analyzer $1750. Call (914) 278-2121.

REAL ESTATE FOR SALE OR RENT

OFFICE SPACE AVAILABLE TO SHARE—1,500 sq. ft. in Medical Service Building at St. Agnes Hospital, White Plains. Call (914) 428-7480.

REAL ESTATE FOR SALE OR RENT—CONT'D

FREEPORT, SOUTH, NY Colonial adjacent to shops, banks, other professionals and waterfront. Ideal home/professional potential, 70′ x 160′, 3 bedrooms, 1½ baths, kitchen/nook, DR, L/R with fireplace, 2 small dens, basement, oil steam heat and garage. Principles only, asking $219,000. Mrs. Smith (516) 228-5771/5, evenings: (516) 741-4799.

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

EAST SIXTIES, PROFESSIONAL BUILDING. The preferred location for internists, cardiologist, rheumatologist. 7 treatment rooms, 2 reception areas to share with three other internists. Share NYC licensed lab, radiology, thermography. Full or part time. (212) 838-2860.

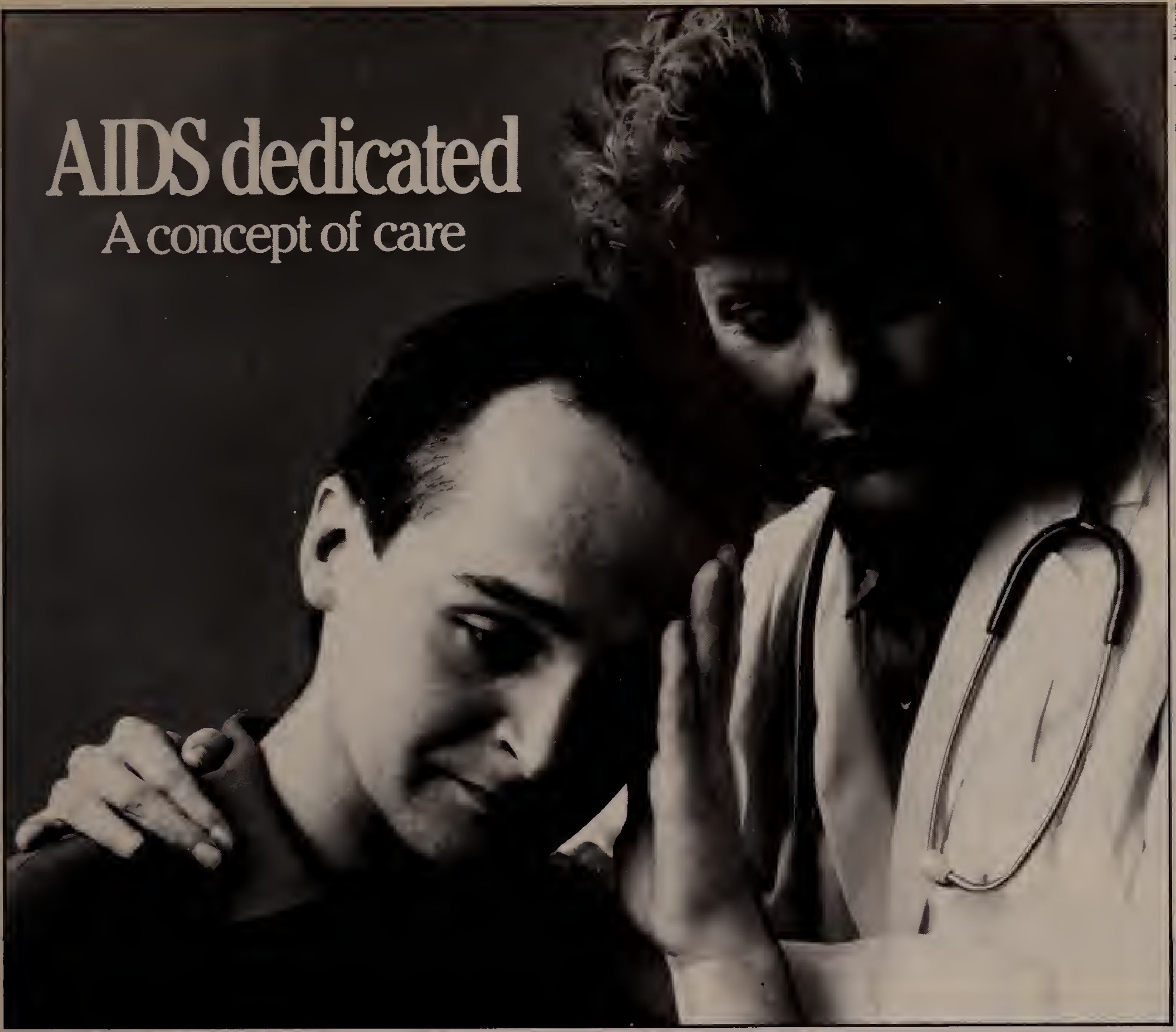
SCARSDALE—Georgian brick, acre+, built 1982, oak floor, 4 bedrooms, 6 bathrooms, indoor pool, maid quarters, 3 zone gas heat and central air conditioning, courtyard and patio, oak kitchen cabinets, 2 dishwashers, 2 Jenn-Air ranges, Thermador wall oven. A must see. Call Broker (914) 273-8259 or (212) 994-5007.

MISCELLANEOUS

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

ARE YOU HAVING A PROBLEM RELATING TO CHEMICAL DEPENDENCE? If your answer is affirmative, contact Susan Kaplan, Attorney-at-Law, (212) 877-5998. Practice limited to legal problems directly involving your license to practice medicine—with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professionals), and as an Assistant District Attorney in Nassau County. Susan Kaplan, Esq., 165 West End Avenue, Suite 27P, New York, N.Y. 10023.

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuarial certification and employee statements of participation—partnership agreements and professionals incorporation—No insurance required—references upon request—Wachstock and Dienstag Attorneys at Law, 122 Cutter Mill Road, Great Neck, NY 11021 (516) 773-3322.



Photography by Ken Nahoum

AIDS is a complex disease, assaulting the psyche as well as the body. The Spellman Center for the Treatment of Persons With AIDS treats both. A comprehensive medical facility, Spellman is devoted to the care of those suffering from Acquired Immune Deficiency Syndrome.

Respect for individual dignity guides the Spellman program. Staffed by trained personnel dedicated to working with AIDS patients, the Center includes family and friends in the management of the patient's health crisis. Spellman also operates a 24-hour information hotline at 1-800-433-AIDS.

For criteria on admission and referrals, please contact the Medical Director, at **1-212-307-0735**. Spellman is a division of St. Clare's Hospital and Health Center, designated the first AIDS treatment center by New York State.

The Spellman Center
for the Treatment of Persons With AIDS
St. Clare's Hospital and Health Center

415 W 51st St
New York, NY 10019

An Affiliate of New York Medical College

An AZT Clinical Site

Medical experts,
providers of care

PHYSICIANS RECIPROCAL INSURERS

Offering New York State Physicians the Broadest
Claims-Made Professional Liability Coverage

OUR UNIQUE PROGRAM INCLUDES...

■SELECTIVE UNDERWRITING	A preferred risk group
■STATEWIDE DISTRICT REPRESENTATIVES	Knowledgeable and sensitive to physicians' concerns
■GROUP PRACTICE COVERAGE	Additional layer of protection at nominal cost
■NEW DOCTOR & PART-TIME RATES	Aware of the special needs and unique situations of our fellow professionals
■AND MORE....	

FOR ADDITIONAL INFORMATION CONTACT
PHYSICIANS RECIPROCAL INSURERS
111 East Shore Road, Manhasset, New York 11030
212/936-3050 • 516/365-6690 • TOLL FREE 800/632-6040

of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175. Tel: (800) 523-1546.

FLORIDA

Feb 5–7. **The 21st Annual Symposium on Cosmetic Surgery.** Hyatt Regency Miami. Contact: Thelma MacGregor, Cedars Medical Center, 1400 NW 12 Ave, Miami, FL 33136.

Feb 12–14. **Perspectives in Allergy and Rheumatology.** 10½ Cat 1 Credits. Colony Beach and Tennis Resort, Longboat Key, Sarasota. *Feb 15–18.* **Critical Care Medicine and Update.** 24 Cat 1 Credits. Americana Dutch Resort Hotel, Lake Buena Vista. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175. Tel: (800) 523-1546.

Feb 14–16. **The 4th Annual Mohs Surgery Conference.** The Buena Vista Palace, Orlando. Info: Mitzi Moulds, Executive Director, The Skin Cancer Foundation, 475 Park Ave South, New York, NY 10016. Tel: (212) 725-5176.

Feb 15–19. **Thoracic Imaging 1987.** 32 Cat 1 Credits. Wyndham Hotel Sea World, Orlando. Contact: R. Dawne Ryals, Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

Feb 16–20. **The Physician in Management.** 31 Cat 1 Credits. The Sheraton Sand Key, Clearwater Beach. Contact: Sherry Mason, American Academy of Medical Directors, 4830 W Kennedy Blvd, Suite 648, Tampa, FL 33609. Tel: (813) 873-2000.

Feb 26–Mar 1. **Flexible Fiberoptic Sigmoidoscopy in the Diagnosis of Colorectal Diseases.** 15 Cat 1 Credits. Wyndham Hotel Sea World, Orlando. Info: Seminars & Symposia, Inc, 132 East 70th St, New York, NY 10021. Tel: (212) 517-7520.

HAWAII

Feb 7–14. Office Pediatrics for Primary Care Physicians. 20 Cat 1 Credits. Kona Surf Hotel, Kona. Info: University of Colorado School of Medicine, Office of Continuing Medical Education, 4200 E 9th Ave, Box C-295, Denver, CO 80262. Tel: (303) 394-5195.

KENTUCKY

Feb 22–27. **18th Family Medicine Review.** Hyatt Regency Hotel in Lexington. Info: Joy Greene, Continuing Medical Education, 132 College of Medicine Office Building, University of Kentucky, Lexington, KY 40536-0086. Tel: (606) 233-5161.

LOUISIANA

Feb 1–4. **American Society for Parenteral and Enteral Nutrition, 11th Clinical Congress.** New Orleans Convention Center. Info: Kelly G. Tilton, Conventions & Exhibits, ASPEN, 8605 Cameron St, Suite 500, Silver Spring, MD 20910. Tel: (301) 587-6315.

TEXAS

Feb 23–28. **Doppler and 2-D Echocardiography.** 40 Cat 1 Credits. San Antonio. Contact: Lisa Krehbiel, Institute for Medical Studies, 30131 Town Center Dr, Suite 215, Laguna Niguel, CA 92677. Tel: (714) 495-4499.

UTAH

Feb 5–7. **Colposcopy: Basic and Advanced.** Yarrow Hotel, Park City. Info: ACOG Registrar, 600 Maryland Ave,

(*continued on p 15A*)

FOR SEVERE COUGHS, CAN YOU REMEMBER THESE LETTERS?

You know Robitussin®, and the "DAC" formula gives you just what you need to manage those severe coughs complicated with nasal congestion . . . Decongestant And Codeine!

Robitussin®-DAC Ⓒ

Each 5 ml (1 teaspoonful) contains:

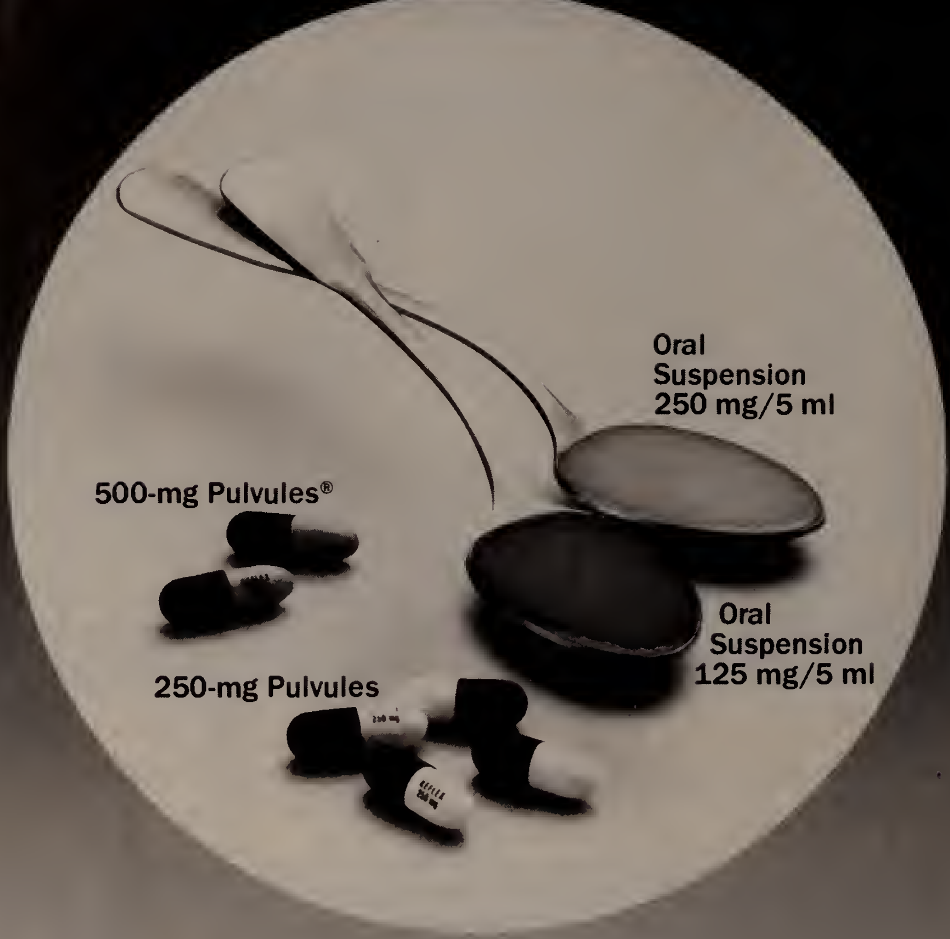
Guaifenesin, USP	100 mg
Pseudoephedrine Hydrochloride, USP	30 mg
Codeine Phosphate, USP	10 mg

(Warning: May be habit forming)
Alcohol 1.4 percent

℞ ONLY

A·H·ROBINS
Pharmaceutical Division
Richmond, Virginia 23261-6609

Easy To Take



Keflex®
cephalexin

Additional information available to the profession on request.

DISTA

420113

Dista Products Company
Division of Eli Lilly and Company
Indianapolis, Indiana 46285
Mfd. by Eli Lilly Industries, Inc.
Carolina, Puerto Rico 00630

NEW YORK STATE
JOURNAL OF MEDICINE

January 1987 • Volume 87, Number 1

COMMENTARIES

New York's homeless

The plight of the homeless in New York State comes under intensified societal scrutiny during the winter months. The press and media almost routinely carry stories dramatized by shocking photographs of people sleeping on heating grates or taking shelter under boardwalks or in abandoned cars. Yet homelessness is a year-round problem and is on the increase throughout the country.[1(p1)] National estimates of the numbers of homeless range from 250,000 to two million.[2(pp18-19),3] In New York State it is estimated that there are 50,000 homeless, 85% of them located in New York City.[4(pp6-8,10)] A survey of 250 shelter providers conducted by the New York State Department of Social Services revealed that in 1983, 20,200 persons used shelters on an average night in New York State, of whom 9,013 were single; the remaining 3,170 were families.[4(pp6-8,10)]

Counting the homeless is not an easy task, because only a percentage of them stay in shelters; locating the others is extremely difficult. Thus, various studies conducted either nationally or in localities frequently produce broad ranges of census data. While the census data from diverse studies often conflict, most show that homelessness is on the increase.[1(pp10-13)]

A multiplicity of societal factors and public policies have worked in concert to create the homelessness problem of the 1980s. For many decades, the homeless in New York State and elsewhere in the United States were single men suffering from alcoholism or drug addiction, or else they were transients.[5] Members of either of these groups frequently wandered the country, and the latter often engaged in seasonal work. They were associated with bars, pool halls, flop houses, and the skid rows of major cities, of which the Bowery in New York City is the best known. These groups have not disappeared, but their numbers among the homeless have been proportionately reduced by the addition of other categories. Among these are chronic mentally ill persons who have been deinstitutionalized without benefit of community-based services, and those displaced from homes because of personal crises, unemployment, cuts in public assistance programs, and the conversion of low-cost rental housing into expensive condominiums, cooperative apartments, and office space.

Between 1955 and 1980, the population of state mental institutions throughout the country decreased 75%, from 559,000 to 138,000.[6] This process of deinstitutionalization was brought about by both federal and local legislation, the intent of which was to return patients to community-based mental health centers, most of which were never built. The Hospitalization Act of 1964 in New York State effectively stripped courts of the power to issue involuntary commitments to mental hospitals and made it easier for state mental hospitals to reject patients.[7] Gruenberg[7] makes a cogent point when he characterizes the intended community care as de facto community neglect and deinstitutionalization as abandonment. This is a widely shared view.[8]

At present, patients discharged from New York's state mental hospitals are frequently lost to follow-up or else move through the so-called "revolving door" in and out of the service delivery system.[9] Many of the chronic mentally ill never get into state hospitals because of admission diversion policies. A number of studies have been conducted to determine the proportion of the homeless who are suffering from chronic mental disorders. A National Institute of Mental Health analysis of various surveys found that an average of 50% of the homeless have severe and persistent mental disorders, 10–15% abuse drugs, and 40–45% abuse alcohol, with a great deal of overlap between categories.[1(p21)] Surveys of shelter populations are biased, because residents may not be a true reflection of the universe. The more severely mentally ill and those with specific mental disorders may avoid shelters.

In 1983, the American Psychiatric Association set up a task force to study the homeless mentally ill. They concluded that a substantial proportion of the homeless are mentally ill men and women who, in previous years, would have been long-term residents of state mental institutions. In addition, they found that in states with admission diversion policies, a significant proportion of the homeless mentally ill had never had psychiatric care.[10]

The essential problem of the chronic mentally ill homeless is not homelessness but mental illness. Attempts to address their homelessness are at best palliative, because they fail to deal with the basic problem. Providing these homeless with shelter is laudable. However, it does not in and of itself alter the public policies and the regulations spawned by those policies that put "bag ladies" on the streets and simultaneously deprived them of access to psychiatric care.

The American Psychiatric Association has recommended a reexamination of the closing of state mental hospitals and a loosening of involuntary commitment rules.[10] These efforts, coupled with an expansion of short- and long-term mental health care and social services, would move many of the homeless mentally ill into safer and healthier living environments. Failure to implement these measures will only result in a worsening of the problem, as the baby boom cohort of the population, with its larger numbers of mentally ill, reaches young adulthood. In places like New York City, the conversion of single room occupancy hotels (SROs) into condominiums, or the demolition of these hotels to make way for expensive housing or office space, will only aggravate the problem, as a burgeoning population of chronic mentally ill is pushed out onto the streets.

Unemployment is frequently cited as a major cause for homelessness.[11] However, unemployment cannot be viewed as an isolated variable. The homeless chronic mentally ill are often unemployed, as are those suffering from drug addiction and alcoholism. Shelter-based surveys that try to determine whether or not unemployment is responsible for homelessness are prone to producing unreliable results, since the basic problem of those being surveyed is not unemployment but rather mental illness.

A significant group of homeless consists of those who have experienced personal crises. Among this group are runaways, persons recently released from prison, refugees from domestic violence, and those who have been evicted from their homes by either fire or landlord action.[2(pp22-26)] A survey conducted by the New York City Human Resources Administration on a single night in June 1984 found that 25% of 791 men and 131 women interviewed in three city shelters said that they had come either because they were no longer welcome at their previous lodging place or because they had nowhere to go.[12] The results of such studies must be viewed in light of the nature of the shelters surveyed and the type of individuals they attract.

Reductions in public assistance have been cited by some as a cause for increased homelessness among the poor.[1(p23)] During the 1980s, a number of federal domestic programs that provided different forms of assistance to the poor were curtailed. State programs were also reduced, with new eligibility standards being established. In Pennsylvania, for example, the number of monthly general assistance payments to able-bodied persons under the age of 65 years was reduced to three months from 12 in any one year.[11(p24)] Many of the people who receive these payments turn to shelters during the three quarters of the year that they do not receive payments.[11(p24)]

The decline in the low-income housing supply is another major cause of homelessness, particularly among families. This is an emerging and increasing problem in New York City, where condominium conversion, gentrification of low-income neighborhoods, and redevelopment play an important role in reducing the stock of low-cost housing. In addition, rent control is providing slim or no profits to landlords faced with increasing maintenance costs, and income tax incentives for abandonment have led many owners of low-cost housing to walk away from it. Arson of inner city housing stock by both landlords out to recoup insurance costs and tenants desirous of relocation and relocation allowances greatly reduced the number of apartments in the city during the 1960s and 1970s. Meanwhile, New York City, lacking in sufficient capital funds, has not been able to undertake the construction of large numbers of low-cost housing units. The combination of increased demand from higher income renters and decreased construction is held by many as the major factor for the decreasing supply of housing for low-income renters.[1(p25)] Since 1980, the federal government has shifted its low-income housing aid away from providing subsidies for the construction and operation of public housing to providing people with vouchers to find existing housing in the private market.[13]

In New York City, the last hope for many people before becoming completely homeless is often the single room occupancy hotel. Yet between 1978 and 1984, New York City lost 32,000 SRO units.[4] Those persons most affected by this loss are the chronic mentally ill.[1(p25)] However, families living in such housing are affected as well.

In this issue of the *Journal*, Chavkin et al[14] present the results of a survey of 401 women who delivered babies while residing at New York City hotels for the homeless. Although the data Chavkin et al present have certain limitations, they clearly portray the plight of these women, who are often single-parent heads of households. Compared to women living in low-income housing projects and women citywide, the homeless women were younger, received significantly less prenatal care, and gave birth to babies whose birth weights were lower.[14] In addition, infant mortality was highest for the children born to mothers living in hotels.[14]

Anyone who has visited these hotels does not need statistical data to convince them that such housing is unsuitable for pregnant women and children. The hotels are crowded and grim, and are often compressed into busy business districts on Manhattan's west side, far from hospitals and primary care services, but near the drug pushers, hustlers, and video arcades. The plight of these women and children will only grow worse unless strong action is taken to provide both immediate and long-range relief. As the study by Chavkin et al shows, the provision of shelter alone does not solve any of the other problems faced by the homeless.

Short-term solutions, such as placing homeless families in midtown Manhattan hotels with flagging occupancies, are popular with politicians.[15] This solution takes homeless families off the streets and, therefore, out of the news. However, it solves none of the basic problems and does not even represent a step towards a needed long-term solution. Approximately 40% of New York City's homeless families are now housed in a half-dozen hotels between 23rd Street and 32nd Street.[15] As the number of homeless families in this business district has increased, so too has opposition to their presence from a coalition of real estate interests, businessmen, regular residents, and clergymen. Self-interest and concern about the welfare of these families have galvanized these diverse groups—some of which filed a suit in state supreme court in September 1986 asking that the city stop placing homeless families in hotels in the area and reduce the numbers already there.[15] If the suit is successful, it could force the city to place these families in hotels in other boroughs, a move that is likely to meet with resistance in those areas for much the same reasons that midtown groups want the families relocated.

Homelessness is a long-term problem in both New York and the United States. In large urban centers such as New York City, homelessness will escalate in magnitude in the years ahead unless the forces creating it are stopped and creative long-term interventions are instituted. The interventions put forth through the consensus of those who have most studied homelessness include more permanent low-income housing, comprehensive and ongoing mental and physical health care, the expansion of community-based services, employment and training programs, and efforts to help the homeless gain access to available programs and benefits.[1(p58)] In New York City, the municipal administration could move forward on one front by adopting proposals such as that of the Partnership for the Homeless to spend $320 million over two years to rehabilitate some 20,000 city-owned apartments. This action would provide some of the homeless with permanent housing at affordable rents.[16] Unless these and other bold measures are taken, the dark silhouettes of the homeless stretched out across heated grates on frigid winter nights will haunt America's horizon into the 21st century.

PASCAL JAMES IMPERATO, MD
Editor

Professor and Chairman
Department of Preventive Medicine
and Community Health
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

1. *Homelessness: A Complex Problem and the Federal Response.* US General Accounting Office publication No. GAO/HRD-85-40. General Accounting Office, 1985.
2. *A Report to the Secretary on the Homeless and Emergency Shelters.* US Dept of Housing and Urban Development, 1984.
3. Hombs ME, Synder M: *Homelessness in America: A Forced March to Nowhere,* ed 2. Washington, DC, The Community for Creative Non-Violence, 1983, p xvi.
4. *Homelessness in New York State,* vol 1. Albany, New York State Department of Social Services, 1984.
5. Leepson M: The homeless: Growing national problem. *Editorial Research Reports* 1982; 11:796-802.
6. Goldman H: The demography of deinstitutionalization, in Bachrach LL (ed): *Deinstitutionalization.* San Francisco, Jossey-Bass, 1981, pp 34-35.
7. Gruenberg EM: Mental disorders, in Last JM (ed): *Maxcey-Rosenau Public Health and Preventive Medicine,* ed 12. Norwalk, Conn, Appleton-Century-Crofts, 1986, pp 1378-1379.
8. Abandoned: Well meant reforms have shattered our system for treating the chronic mentally ill and left thousands of them to scrape by in the streets. *Newsweek,* January 6, 1986, pp 14-20.
9. Bachrach LL: Continuity of care for chronic mental patients: A conceptual analysis. *Am J Psychiatry* 1981; 138:1449-1456.
10. *The Homeless Mentally Ill: A Task Force Report of the American Psychiatric Association.* Washington, DC, American Psychiatric Association, 1984.
11. Robertson MJ, Ropers RH, Boyer R: *The Homeless of Los Angeles County: An Empirical Evaluation.* Los Angeles, Basic Shelter Research Project, 1985.
12. *Correlates of Shelter Utilization: One-Day Study.* New York, New York City Human Resources Administration, August 1984.
13. Struyk RJ, Tuccillo JA, Zais JP: Housing and Community Development, in Palmer JL, Sawhill IV (eds): Washington, DC, The Urban Institute Press, 1982, pp 406-415.
14. Chavkin W, Kristal A, Seabron C, et al: The reproductive experience of women living in hotels for the homeless in New York City. *NY State J Med* 1987; 87:10-13.
15. Basler B: Organizing to block the homeless: In midtown groups oppose Koch's policy. *New York Times,* October 31, 1986, pp B1-2.
16. Smith PP: Homelessness in New York isn't inevitable. *New York Times,* September 20, 1986, p 27.

Homelessness in New York City: The youngest victims

American families are becoming homeless at an alarming rate, due in part to the rising proportion of families falling below the poverty line[1] and to the shortage of low cost housing. The important paper in this issue of the *Journal* by Dr Wendy Chavkin and her colleagues[2] at the New York City Department of Health (DOH) should serve to alert the New York State medical community to some grim, if not surprising, news. Pregnant women who are homeless are significantly less likely to receive adequate prenatal care. They are also more likely both to deliver a low birthweight baby and to have a child die in the first year of life, when compared either to women in public housing projects (persons similar to the homeless in sociodemographic background) or to women in the general New York City population.

In the best tradition of policy oriented epidemiology, the authors of the DOH study sought to discover the health consequences of homelessness for pregnant mothers. Routine data were used in a creative and economical quasi-experimental design.[3] Birth and death certificates from January 1982 through June 1984 served to identify persons stating their addresses as one of the principal hotels used to house the homeless, as well as to identify women living in any of the over 200 low income public housing projects in New York City. This strategy permitted comparisons of birth and death outcomes in the group of homeless mothers, the public housing project dwellers, and the entire New York City population delivering a baby during the two-and-a-half-year study period. Some of the limitations of the methods used are carefully outlined by the authors themselves. It would have been useful if the authors had included confidence intervals[4] for their risk ratios.[5] In addition, the lack of statistical control for the probability that a difference between groups on one or more of multiple comparisons might have been due to chance alone introduces the possibility that the authors made a type I statistical error,[6] though the very large sample sizes and the highly significant *p* values are reassuring in this regard.

An important observation in this study is that sociodemographic differences between homeless mothers and mothers living in city housing projects are small. This suggests that for comparisons between these groups, the state of homelessness is the critical predictor variable associated with differences in the outcome variables of infant mortality, prenatal care, and birthweight. While prior mental illness may sometimes predispose individuals to homelessness,[7,8] a recent family-based study in New York City found that the immediate causes of homelessness for 196 surveyed families were linked more to poverty and the shrinking low cost housing market than to causes plausibly related to mental illness. Twenty-nine percent of those surveyed had been forced out by a primary tenant; 28% by fire or other disaster; 20% by eviction by landlord for nonpayment of rent; 17% by "bad" housing conditions (eg, no heat, water, or security); and 11% by "vacate" orders (eg, condemned property)[9] (some families reported more than one precipitating factor). Given this list of causal circumstances, one might surmise that homelessness itself may lead to poor mental health in the highly stressed family member.[8]

The prenatal period, the moment of birth, and the first year of life are the most critical times in human development. Research into the health problems of homeless men demonstrates that the social and psychological stresses associated with homelessness can exacerbate the risk of adverse health outcomes from conditions both coincident with and a consequence of the homeless condition.[10] The major biological risks associated with pregnancy, childbirth, and early infancy are well known. Clinicians can attest to the value of early prenatal care in recognizing and ameliorating many of these hazards, particularly in the high risk pregnancy. The current DOH study reveals the extent to which our present health services fail to protect homeless women from adverse reproductive outcomes.

While not the specific subject of the DOH study, the association of homelessness with adverse outcomes in other areas of maternal and child health is the concern of all clinicians and other health care workers or social workers caring for homeless families. For example, while a free child health service and a school monitoring program help to maintain moderately high rates of immunization in children from poorer families in New York City, homeless children were disproportionately affected in the 1986 measles outbreak (New York City Department of Health, unpublished data, 1986). Surveys of the immunization status of homeless children compared to children of comparable age and socioeconomic background are in progress at Bellevue Hospital (A. Fierman, personal communication, October 1986) and St Luke's/Roosevelt Medical Center (G. Alperstein, personal communication, November 1986) in Manhattan. Such studies will help determine whether a higher incidence of vaccine-preventable infection in the homeless is likely to be a result of greater delay in obtaining immunization or whether other factors, such as increased crowding and contact with potentially infected persons, are responsible. In either case, special attention to vaccination in homeless children is indicated until permanent housing is made available to them. In addition, the low participation rate of homeless children in preschool programs (an estimated 15% of eligible homeless preschoolers attend preschool[11]) means that most homeless children under the age of five will not have gone through the school-mandated process of screening for full immunization coverage. The health impact of homelessness on families needs more attention, as it represents an epidemic of preventable and curable stress on the poorest segment of society, which is least prepared to cope with such upheavals.

Of course, it is safe, low cost housing and not improved health care that represents the ultimate cure for homelessness. In 1981 federal funding provided more than $30 billion for low income housing.[12] In 1986, as the tragedy of homelessness reached levels not seen since the Great Depression, funding was reduced to under $10 billion.[12] Advocates for the homeless continue to pressure government and the real estate industry to house homeless families. The DOH study indicates the urgency of this agenda, while challenging the medical, nursing, hospital, and public health communities to address the immediate needs of homeless families for ready access to health care.

STEN H. VERMUND, MD, MSc
Assistant Professor
Department of Epidemiology and
Social Medicine
Department of Pediatrics

ROBERTO BELMAR, MD
Professor

ERNEST DRUCKER, PhD
Associate Professor
Department of Epidemiology and
Social Medicine
Albert Einstein College of Medicine/
Montefiore Medical Center
Bronx, NY 10467

1. Sidel R: *Women and Children Last: The Plight of Poor Women in Affluent America*. New York, Viking-Penguin Inc, 1986.
2. Chavkin W, Kristal A, Seabron C, et al: The reproductive experience of women living in hotels for the homeless in New York City. *NY State J Med* 1987; 87:10-13.
3. Cook TD, Campbell DT: *Quasi-Experimentation: Design and Analysis Issues for Field Settings*. Boston, Houghton Mifflin Co, 1979.
4. Simon R: Confidence intervals for reporting results of clinical trials. *Ann Intern Med* 1986; 105:429-435.
5. Kelsey JL, Thompson WD, Evans AS: *Methods in Observational Epidemiology*. New York, Oxford University Press, 1986.
6. Godfrey K: Comparing the means of several groups. *N Engl J Med* 1985; 313:1450-1456.
7. Bassuk EL, Rubin L, Lauriat A: Is homelessness a mental health problem?

Am J Psychiatry 1984; 141:1546–1550.

8. Holden C: Homelessness: Experts differ on root causes. *Science* 1986; 232:569–570.

9. *Longitudinal Study of Homeless Families: Return to Permanent Housing.* New York, New York City Human Resources Administration, December 1985.

10. Brickner PW, Scanlan BC, Conanan B, et al: Homeless persons and health care. *Ann Intern Med* 1986; 104:405–409.

11. *7000 Homeless Children: The Crisis Continues.* New York, Citizens' Committee for Children of New York, Inc, October 1984.

12. O'Connor J: Homeless face 'national crisis,' need help now. *Psychiatric News* (newspaper of the American Psychiatric Association), October 17, 1986, pp 1, 16.

Implications of the recent clinical trials in hypertension

Several long-term clinical trials relating to the treatment of hypertension have recently been reported. These have important implications for the practicing physician.

The European Working Party Study on High Blood Pressure in the Elderly (EWPHE)[1] has helped clarify questions regarding the treatment of hypertensive patients 60 years of age or older. In this study, 840 people with an average age of 70–71 years and average pretreatment blood pressures of 182–183/101 mm Hg were placed at random in a treatment group or a placebo group. The investigators found that one capsule a day containing 25 mg of hydrochlorothiazide and 50 mg of triamterene was sufficient to lower blood pressure to goal levels in 51% of the subjects; 45% of the participants required two or more capsules, and 35% required the addition of a second drug, alpha methyldopa. While levels of serum creatinine, glucose, and uric acid rose in the treatment group compared to the placebo group, the serum potassium level decreased by only 0.1–0.2 mEq/L—presumably because of the use of the potassium sparing agent—and side effects were generally not significant.

Blood pressures were reduced by 10/6 mm Hg in the placebo group and by 32/13 mm Hg in the treatment group at the end of the first year, with only a slight additional decrease in blood pressure in the treatment group reported during the next four to five years.

Decreases of 27% in total cardiovascular mortality, 38% in cardiac mortality, and 32% in deaths from cerebrovascular disease were noted in the treatment group compared to the placebo group (differences in deaths from cerebrovascular disease did not achieve statistical significance, presumably because of the small number of cases involved). Mortality was reduced among patients in the treatment group whose pretreatment diastolic pressures were either above or below 100 mm Hg. Mortality data are not available regarding the benefit of treatment for patients with pretreatment diastolic pressures of 90–95 mm Hg.

These data appear to confirm previous impressions that therapy for elderly patients with both systolic and diastolic hypertension is beneficial, that it can lower blood pressure, and that it will result in reduced morbidity and mortality. The data are consistent with the findings in the 60–69-year-old cohort in the Hypertension Detection and Follow-up Program (HDFP) and Australian trials.

The EWPHE study provides important information to physicians, who now should be able to treat elderly patients with systolic and diastolic hypertension with some conviction that the treatment will be beneficial. It also confirms the efficacy and relative safety of diuretic therapy in this group of patients. It is of interest that a one-year study on systolic hypertension in the elderly in the United States has also demonstrated the effectiveness of low-dose diuretic therapy in this group of patients.

The Medical Research Council (MRC) trial,[2] involving more than 17,000 patients during a five- to six-year period, showed that 18% of patients with mild hypertension became and remained normotensive on placebo therapy during the first three years. The phenomenon of normalization usually occurred within three months. Between a third and a half of those patients receiving a placebo had diastolic pressures below 90 mm Hg on annual examinations; different people made up the total at each examination. Since these patients had already been screened on several occasions prior to entering the study, it is probable that even a greater number of unselected patients with mild hypertension would become normotensive without specific therapy. For this reason, the 1984 Joint National Committee Report on Detection, Evaluation, and Treatment of High Blood Pressure[3] suggested that observation of patients with mild hypertension (diastolic pressures between 90 and 104 mm Hg) for three to six months is a reasonable first-step approach to management.

However, in a large number—1,011, or about one eighth of the total—of patients in the placebo group in the MRC trial, severe hypertension developed (blood pressures above 200/110 mm Hg), compared to only 76 patients in the treatment group. This finding is consistent with other studies and serves as an argument for careful follow-up and early treatment of patients with mild hypertension.

Although total cardiovascular events were substantially reduced by beta adrenergic inhibitors or diuretics, compared to a placebo, the effect of treatment on fatal coronary events was minimal. This finding was disappointing but is not unexpected in a relatively short-term trial, in which individuals with measurable evidence of pretreatment cardiovascular disease were excluded from the randomization process. The decrease in coronary deaths noted in the HDFP and the EWPHE trials may be accounted for by the inclusion of more elderly patients or patients with pretreatment end-organ damage.

The MRC results also confirm the efficacy and relative safety of diuretics, even in high dosages (bendrofluazide, 10 mg/day—approximately equivalent to 100 mg of hydrochlorothiazide). Treatment resulted in lower blood pressures, fewer strokes and other cardiovascular events, and reduced stroke death rates when compared to the findings for the placebo group. There was no association between diuretic use and increased coronary deaths.

While stroke death rates were reduced among both smokers and nonsmokers in the diuretic treated group, only nonsmokers benefited in the beta blocker group. Coronary death rates were only reduced to a measurable degree among the nonsmokers receiving beta blockers; no reduction was seen for either smokers or nonsmokers receiving diuretics. These findings emphasize the importance of reducing as many risk factors as possible to prevent heart disease.

A third study, The International Prospective Primary Prevention Study in Hypertension (IPPPSH),[4] was a complicated multinational trial involving more than 6,000 patients. One group of 3,172 patients was treated with no medication or with a diuretic plus any other drugs, other than beta blockers, that were necessary to lower blood pressures to goal level (<95 mm Hg). The other 3,185 patients received beta blocker therapy plus any other drugs that were necessary to lower blood pressures, including diuretics. Overall cardiovascular mortality was lower than original estimates in both groups, and there was a direct correlation between blood pressure and the cardiac event rate. The authors concluded that the lowering of blood pressure, rather than the type of medication administered, resulted in the reduced mortality and morbidity, and that beta adrenergic inhibitors were not more effective than diuretics, except among nonsmoking men. Again, no deleterious effect of diuretics on cardiovascular mortality was noted; in this study, too, more than 40% of patients received potassium sparing agents or potassium supplements. Although there are problems with the design of this trial, the conclusions are of interest.

The MRC, EWPHE, and IPPPSH trial results appear to be consistent with previous studies, such as the HDFP[5] and Australian trials.[6] The findings appear to vindicate the position that specific therapy and lowering of blood pressure in patients with mild hypertension, even among the elderly, is justified before the onset of end-organ impairment or more severe disease. However, specific answers to the dilemma of treating patients with diastolic pressures between 90 and 94 mm Hg were not given in any of these trials except the HDFP. These studies also appear to reestablish the efficacy and relative safety of diuretic therapy.

In the MRC trial, a reduction in the cardiovascular event rate of 1.6/1,000 patient years and in the stroke events of 1.2/1,000 patient years translates to 32,000 and 23,500 fewer events per year, respectively, in a population of approximately 20 million persons with mild hypertension in the United States. The EWPHE study, in which a higher overall mortality rate was expected, reported a 13/1,000 patient year reduction in cardiovascular deaths (translating to a potential of 200,000–260,000 lives saved per year among the approximately 15–20 million elderly persons with hypertension in the United States). The 2.3/1,000 patient year reduction in death rates reported in the HDFP study can be translated to 46,000 fewer deaths per year, if patients with mild hypertension are effectively treated. These benefits are not inconsequential and can probably be achieved by the use of one medication or small doses of two different drugs, requiring only two to three physician visits per year and little disturbance of quality of life.

The challenge remains to select individuals with mild hypertension who might benefit the most from therapy, so that the fewest possible number of people will be subjected to long-term medical treatment. If periodic readings are taken to detect patients whose pressures decrease to normal without specific intervention, and if nonpharmacologic approaches (which may be effective in about 20–30% of cases) are tried before drug treatment is begun, excessive treatment may be minimized. If blood pressures remain elevated (>140/90–95 mm Hg), however, specific therapy is indicated.

The results of these recent studies, coupled with those previously published, should serve as guidelines to physicians in planning long-term treatment programs for patients with hypertension.

MARVIN MOSER, MD
Senior Medical Consultant
National High Blood Pressure
Education Program
National Heart, Lung, and
Blood Institute

Clinical Professor of Medicine
Yale University School
of Medicine
New Haven, CT 06510

1. Amery A, Birkenhager W, Brixko P, et al: Mortality and morbidity results from the European Working Party on High Blood Pressure in the Elderly trial. *Lancet* 1985; 1:1349–1354.
2. MRC trial of treatment of mild hypertension: Principal results. Medical Research Council Working Party. *Br Med J* [*Clin Res*] 1985; 291; 97–104.
3. The 1984 Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. *Arch Intern Med* 1984; 1045–1057.
4. The International Prospective Primary Prevention Study in Hypertension (IPPPSH). Cardiovascular risk and risk factors in a randomized trial of treatment based on the beta blocker oxyprenolol. *J Hypertens* 1985; 3:1–13.
5. Five-year findings of the hypertension detection and follow-up program. I. Reduction in mortality of persons with high blood pressure, including mild hypertension. *JAMA* 1979; 242:2562–2571.
6. The Australian Therapeutic Trial in Mild Hypertension. Report by the Management Committee. *Lancet* 1980; 1:1261–1267.

Azidothymidine and human immunodeficiency virus infection

The National Institutes of Health in association with the Burroughs Wellcome Company has recently announced that azidothymidine (AZT) will be made available to patients with acquired immunodeficiency syndrome (AIDS) who have had an episode of *Pneumocystis carinii* pneumonia.[1] AZT is an antiviral agent that has been shown in a double-blind trial to temporarily decrease morbidity and mortality in patients with AIDS and AIDS-related complex (ARC).[2] Accordingly, the Food and Drug Administration has placed the drug on a fast-track approval process, so that it may be available by prescription to all physicians by early 1987. Aside from critical questions of both efficacy and long-term toxicity, it is clear that the availability of antiviral therapy for human immunodeficiency virus (HIV, formerly known as HTLV-III/LAV) infection will dramatically affect the provision of care to patients with this disease.

The identification of HIV as the etiologic agent of AIDS, and the subsequent publication of numerous seroepidemiologic studies, have made it clear that patients infected with this retrovirus present with a variety of manifestations. These range from asymptomatic infection to severe immunodeficiency and life-threatening secondary infectious diseases and cancer. The initial surveillance definition of AIDS was extremely effective in both the clinical and the epidemiologic study of this disease. However, this definition measured only the most severe manifestations of HIV infection. With the need for a more inclusive definition becoming apparent, the Centers for Disease Control (CDC) has developed a classification system that groups patients infected by the virus according to the clinical expression of disease.[3] The availability of antiviral therapy will certainly accelerate clinicians' acceptance of this scheme as the application of such therapy is inevitably broadened beyond those patients who have AIDS. Although AZT is currently approved only for use in patients who fulfill the clinical definition of AIDS, it may be used for less severe manifestations of HIV infection as clinicians become more comfortable with its use. Some physicians, for example, may feel that the benefits will outweigh the risks of AZT therapy for patients with severe ARC or dementia. Indeed, if AZT (and/or other antiviral interventions) is found to be effective we may find such therapy used progressively earlier in the course of HIV infection. This will encourage physicians to follow closely such immunologic parameters as the T helper (T4) lymphocyte count and the presence of delayed hypersensitivity skin reactions.

The availability of AZT will likely accelerate the already changing focus of the treatment of this disease from the inpatient service to the ambulatory care setting. One of the major goals of clinicians dealing with AIDS patients is to keep them at home, a commitment that fulfills both social ideals and economic needs. Currently, because of the severity of HIV infection, many patients require frequent follow-up from a variety of health care providers, including physicians and social workers. The use of AZT will place an additional strain on the outpatient service. While morbidity may be reduced for HIV-infected patients receiving AZT, they will require careful monitoring. Patients on AZT are at risk of developing severe anemia[2,4] and will initially require at least monthly physician visits and biweekly complete blood counts. Thus, physicians and institutions dealing with large numbers of these patients will have to develop monitoring programs for patients receiving AZT therapy. In addition, third party payors should be aware of the impact that frequent visits will have on the cost of providing care.

With the availability of antiviral therapy, we may also see increased utilization rates in the intensive care unit (ICU). In the early years of the epidemic, many AIDS patients received terminal care in the ICU. As the dismal prognosis for such patients became increasingly clear, many of them, after discussion with their physicians, decided to forgo ICU intervention. Although it is too soon to gauge adequately the impact of antiviral therapy on prognosis, its very availability may again increase ICU utilization rates. For example, if a patient survives a complication requiring ICU utilization, with AZT therapy there may be an increased chance of long-term survival. Physicians, patients, and hospital administrators should keep abreast of the results of efficacy studies as they become available. These may dramatically affect both the quality of life of AIDS patients and the cost of their medical care.

Will primary care practitioners need to become comfortable with antiviral therapy? Given the presence in this country of at least one million individuals who are infected with HIV, there will be few physicians who will *not* see such patients. But it is clear that even if AZT is effective, it will not be the last word on therapy. In all likelihood, patients with HIV-caused disease will require some combination of immunostimulation and effective antiviral therapy. Although many primary care physicians have become comfortable with the use of antiviral agents such as acyclovir, the complexity of HIV therapy may require that such treatment be carried out in specialized centers.

Although the potential of therapy for HIV infection ushers in a new and optimistic era for clinicians dealing with this disease, it also raises many new issues. Each of these issues must be dealt with as we begin to develop an effective therapeutic armamentarium for the ravages of this virus.

JACK A. DeHOVITZ, MD, MPH
Assistant Professor
Department of Preventive Medicine
and Community Health
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

Assistant Medical Director
Spellman AIDS Program
St Clare's Hospital and
Health Center
New York, NY 10019

1. Barnes DM: Promising results halt trial of anti-AIDS drug. *Science* 1986; 234:15–16.
2. Azidothymidine for AIDS. *Med Lett* 1986; 28:107–109.
3. Classification system for human T-lymphotropic virus type III/lymphadenopathy-associated virus infections. *MMWR* 1986; 35:334–339.
4. Yarchoan R, Klecker RW, Weinhold KJ, et al: Administration of 3′-azido-3′-deoxythymidine, an inhibitor of HTLV-III/LAV replication, to patients with AIDS or AIDS-related complex. *Lancet* 1986; 1:575–580.

FROM THE LIBRARY

HEALTH AND THE ECONOMIC DEPRESSION

There has been much discussion in periodicals, both medical and lay, regarding the effect of the economic depression on public health and death rates. The prevailing opinion seems to be that the rates for either sickness or death have not increased; or, if they have, the increase is regional and accidental. On the other hand, some studies have revealed an increase in the proportion of undernourished children discovered in public schools, and on this basis predictions of later ill health are made.

This general maintenance of a high standard of health during times of depression was unexpected, because poverty has previously been recognized as a leading cause of sickness, especially the more chronic forms such as tuberculosis. The reasons for the apparent anomaly are plain, and are a justification of the methods adopted throughout the nation for the distribution of medical services to all who need them.

Poverty is a potent cause of ill health because it prevents the very poor from obtaining those things which are essential for healthful living. It is highly to the credit of citizens generally that they have willingly supported public measures for providing the poor with necessities which they would not otherwise have obtained, and thereby they have neutralized the effects of poverty on health. Public officials and welfare organizations have provided the hungry with not merely bread, but with milk and green vegetables which are essential for health, and which were formerly considered to be luxuries. No one in these modern days sees a child clothed in the rags which were the classic badge of poverty a half century ago; and no community tolerates tumble-down tenements, with their lack of sanitation. Finally there has come the realization that medical attention is as essential as food; and the services of a doctor with hospitalization and nursing, if necessary, are available at public expense. Medical service is available more readily and abundantly than ever before. Another factor is the activities of health departments both in carrying on their old lines of work and also in entering new fields, such as giving immunizations, child welfare, maternal health, and school examinations. Some public health nurses have been laid off, but the amount of preventive work done by departments of health has been increased during the depression. All these activities have had a sure effect in lessening morbidity and mortality.

What the future may bring forth is uncertain; but there is abundant evidence that the economic depression has stimulated all forms of health work to such an extent that living conditions for the poor are now guarded and controlled more carefully than ever before.

EDITORIAL
(*NY State J Med* 1933; 33:1395)

The reproductive experience of women living in hotels for the homeless in New York City

WENDY CHAVKIN, MD, MPH; ALAN KRISTAL, MS, DRPH; CHERYL SEABRON, BS;
PAMELA E. GUIGLI, MPH

ABSTRACT. The reproductive experience of women living in hotels for the homeless was compared to that of women living in low-income housing projects and that of all other New York City residents. Birth and linked death certificates for the singleton births during the period January 1982 through June 1984 were used to ascertain maternal characteristics, birth weight, and infant death rate. The relative risks of receiving no prenatal care for residents of hotels compared to those of housing projects and all other New York City residents were 2.5 and 4.12, respectively; the relative risks of low birth weight among these groups were 1.4 and 2.18, respectively; and the relative risks for infant mortality were 1.5 and 2.07, respectively. These data suggest that special efforts should be focused on women living in hotels for the homeless.

(NY State J Med 1987; 87:10–13)

In New York City, attention has recently been focused on the plight of the growing population of homeless families with young children.[1] The number of these families has risen dramatically over the past few years, straining the resources available to shelter such families.[2] In December 1982, there were 1,280 families with 3,187 children in temporary housing facilities consisting of four shelters run by the Department of Housing, Preservation and Development—and managed by nonprofit community agencies—and ten privately owned and managed hotels.[3] By June 1984, the city was using seven shelters and 48 hotels to house 2,889 families (3,498 adults and 6,789 children). This striking trend toward growing numbers of homeless families continues.[4]

Most of these families are single-parent households headed by women.[5,6] The New York City Department of Health established the Family Shelter Program in 1984 to assist these families in meeting their health needs. In a door-to-door survey of one of the larger hotels, the program staff found that in approximately 16% of families there was a pregnant woman.* The public health nurses who comprised the program staff reported that the stressful conditions encountered in these quarters negatively affect the women's utilization of prenatal care. The purpose of the study was to describe the reproductive experience of the homeless population residing in hotels, to aid in future development of the Family Shelter Program.

METHODS

Birth certificate files were used to select three groups of women for this study: the "hotel" group included the 401 women who delivered live-born singleton children in New York City between January 1982 and June 1984, and whose address, as recorded on the birth certificate, corresponded to one of the principal hotels used to house the homeless. The "project" group included the 13,247 women who delivered live-born singleton children in the same period, and whose address on the birth certificate corresponded to one of 219 low-income housing projects sponsored by the city. This population was expected to resemble the hotel population in demographic and socioeconomic characteristics. The second comparison group consisted of all other New York City residents (241,558) who delivered live-born singleton children in New York City during the study period. Data on birth weight, maternal age, parity, race, and prenatal care were gathered from the New York City birth certificate, and subsequent infant deaths were determined from the linked birth/death certificates.

Pairwise comparisons were performed using the chi square test of significance. Multiple regression analysis was also performed to compare birth weight among the three groups.

RESULTS

The number of births to women living in hotels increased from approximately seven per month in 1982 to more than 23 per month in 1984 (Fig 1). Demographic characteristics of the mothers in the three groups are shown in Table I. The racial and ethnic distributions of mothers in the hotel and project populations were similar; about two thirds were black and one quarter were Hispanic.

In contrast, the citywide mothers were evenly divided among the three major racial/ethnic groups. The age distributions among all three groups were dissimilar. Overall, mothers in the hotel and project groups were concentrated in the younger age groups (with more than 50% under 25 years of age), with the proportion of teenage births higher in the project group (30%). Conversely, the citywide group had the highest proportion of births to women over age 30 (30%). The parity distributions within the project and citywide

---

From the New York City Department of Health, New York, NY.

Address correspondence to Dr Chavkin, Director, Bureau of Maternity Services and Family Planning, New York City Department of Health, 280 Broadway, Room 303, New York, NY 10007.

* Nutritional Patterns of Families in a Welfare Hotel, Family Shelter Program, New York City Department of Health, August 1984.

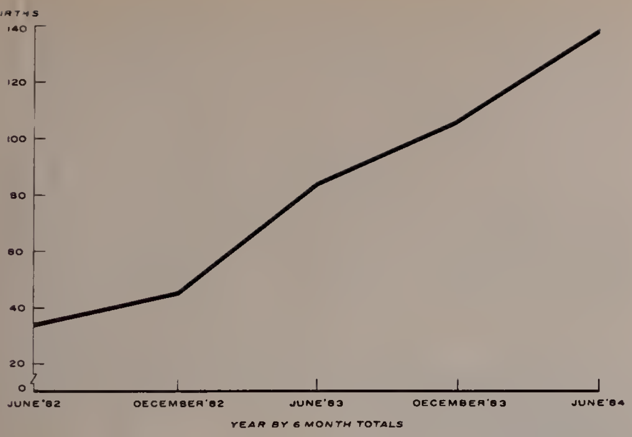


FIGURE 1. Number of births to residents of family shelter hotels in New York City, June 1982–June 1984.

groups were similar. In marked contrast, the proportion of nulliparous women was much lower (20% compared to 40%) in the hotel group and the proportion of women of high parity (three or more children) was much larger (44% compared to 18%) than in the citywide group.

The hotel group had the highest proportion of unmarried women receiving Medicaid who had completed 12 or fewer years of school. The project group was ranked next, and the citywide group had the lowest proportions regarding these parameters.

The birth certificates record the date of the first prenatal visit and the total number of prenatal visits. As these followed similar patterns, only data on the total number of visits are shown here. The hotel mothers reported significantly less prenatal care than did women in the comparison groups (Table II). More than half of this group had no or minimal care (0–3 visits) compared to 22% of the project mothers and 15% of the other mothers in the city. Sixty-eight percent of the citywide group had seven or more visits, whereas only 30% of the hotel women received this level of care.

Mean birth weight in the hotel group was 2,979 g—lower than the project group by 149 g and lower than the citywide group by 276 g. To control for confounding factors due to maternal characteristics, multiple regression analysis was used to compare birth weight across the three groups (Table III).[7] Two covariates, race and prenatal care, were the most significant factors in explaining the lower birth weights among hotel residents. Although this was only an exploratory model, which did not include all possible variables, there was, nevertheless, an estimated reduction of birth weight of 125 g associated with hotel residence and a reduction of 48 g associated with project residence (both $p < 0.001$) compared to an adjusted mean birth weight of 3,253 g in the citywide group.

TABLE I. Characteristics (%) of Mothers of Live-born Singleton Children by Place of Residence, New York City, January 1982–June 1984

Characteristic	Hotel	Low-Income Housing Project	All Others
Age			
<20 yr	17.4	29.8	12.3
>30 yr	19.2	19.8	29.6
Race			
White	11.9	5.8	33.5
Black	63.8	63.5	30.9
Hispanic	22.4	29.4	30.4
Asian and other	1.7	1.2	5.0
Parity			
% Nulliparous	19.7	37.3	38.7
% Parity ≥ 3	44.1	24.0	18.2
Marital status			
% Unmarried	83.3	69.7	37.4
Education			
% >12 years	6.4	12.6	27.3
Medical coverage (Medicaid)	81.7	61.3	34.7
Total live births	401	13,247	241,558

TABLE II. Prenatal Care (%) by Place of Residence

	Hotel	Low-Income Housing Project	All Others
Prenatal Visits			
None	39.7	14.5	9.0
1–3	16.7	8.0	6.0
4–6	13.9	19.8	16.7
7+	29.6	57.5	68.1
Mean number of prenatal visits (if woman had at least one prenatal visit)	6.2	8.2	8.7

The rate of low (< 2,500 g) birth weight delivery was highest in the hotel group and lowest in the citywide group (Table IV). The relative risks for low birth weight, comparing the hotel group to the project and citywide groups, were 1.4 and 2.18, respectively (both $p < 0.01$).

Infant mortality was highest in the hotel group and lowest in the citywide group (Table V). There were ten infant deaths in the hotel group, for a rate of 24.9 per 1,000 single live births—significantly higher than in the project group (16.6 per 1,000) and in the citywide group (12.0 per 1,000). The corresponding relative risks, comparing the hotel group to the project and citywide groups, were 1.5 (not significant) and 2.07 ($p < 0.01$), respectively.

DISCUSSION

There are several limitations to the analyses presented here. The number of hotel births (401) does not reflect the total number of women who lived in a hotel during some part of their pregnancies. Only those women who reported the hotel address on the birth certificate were included. This bias leads to underreporting of hotel births and underestimates of the differences between the hotel and citywide populations. Nevertheless, we consider the birth certificate to be a useful tool for preliminary investigation. Birth certificates provide reliable data for selected param-

TABLE III. Multiple Regression Analysis of Birth Weight Differences (grams) Comparing Residents (234,752) of Hotels and Public Housing to All Others in New York City, January 1982–June 1984*

Control Variables in Model	Hotel	Low-Income Housing Project	All Others
None	−251g	−125g	
Age, infant sex	−233g	−98g	
Race (black, Hispanic, other)	−181g	−49g	
Parity (0, 1–2, 3+)	−182g	−49g	
Prenatal visits	−125	−48g	
Adjusted birth weight (g)	3,128	3,205	3,253

* All effects were significant at the $p < 0.001$ level. Numbers slightly different from tabular analyses, due to exclusion of observations with missing data.

TABLE IV. Birth Weight (%) by Place of Residence

	Hotel	Low-Income Housing Project	All Others
Birth weight			
<2500 g	16.3	11.4	7.4
≥2500 g	84.0	88.0	92.0
Mean birth weight (g)	2,979.5	3,128.8	3,255.8

eters, such as birth weight and maternal age, and thus they enable routine surveillance of the reproductive experience of a population without extensive survey research.

Another limitation to these analyses is that they use only data available in New York City vital records. Important predictors of pregnancy outcome, such as cigarette smoking, are poorly recorded.[8] The reliability of items relating to prenatal care has also been questioned.[9] It has been speculated that reports of no prenatal care may be erroneous when, in fact, a woman received care at an institution other than the one where she delivered.[10]

The low proportion of births to nulliparous women in the hotels reflects the requirements for hotel acceptance. These rooms are reserved for families with young children and so, by definition, these heads of households must already have children. We infer, both from this and from the high proportion of high parity births, that it is primarily these female heads of households who are pregnant while in the hotels, and not their teenage daughters.[11]

Families arrive in the hotels as a result of some catastrophic disruption of their lives, usually fire or eviction.[12] They have often lost their possessions and are frequently lodged in a new borough, far from friends, any source of income maintenance and food stamp offices, the children's schools, and any familiar sources of health care.[13] The mothers spend long hours traversing the city, taking children to school, picking up Aid to Dependent Children (ADC) checks or food stamps, and trying to buy moderately priced food (often daily when there is a lack of refrigeration).

There is barely time available for the most pressing goal—finding permanent housing; much less can time be spared traveling to and then waiting in a prenatal care clinic. We infer that these stressful and difficult circumstances contribute to poor utilization of prenatal care, because the economically disadvantaged residents of housing projects obtained considerably more prenatal care.[14]

It is difficult to care for a new baby while living in one of these hotels for the homeless. There are usually other children in the room and often no basic amenities, such as cribs or facilities for refrigerating formula.[15] An April 1984 survey revealed that only 8% of families had refrigerators; subsequently, official policy stated that pregnant women and families with newborns should have refrigerators.[16] A low-birth-weight infant is at a disadvantage under the best of circumstances, and is in double jeopardy under these conditions.[17]

TABLE V. Infant Mortality Rate by Place of Residence

	Hotel	Low-Income Housing Project	All Others
Number of infant deaths	10	220	2,901
Infant mortality rate (deaths/1,000 live births)	24.9	16.6	12.0

Are the reproductive outcomes in hotels for the homeless poor because exceptionally high-risk women are concentrated there?[18] These data demonstrate that women with multiple risk factors are disproportionately represented among the hotel group.[19] Yet, multiple regression analysis revealed a reduction in birth weight associated with hotel living after controlling for certain maternal risk characteristics. While it is not possible to make conclusions about causal relationships based on these data, there is sufficient foundation for planning the future of the program.[20]

The large differences in utilization of prenatal care suggest that programs designed to improve birth outcome among hotel residents should focus on making prenatal care readily accessible.[21] On-site health education, followed by on-site provision of care or a Department of Health program coordinated with local providers, are some short-term options to consider.[22] Other options might include on-site provision of social services, such as income maintenance and nutritional supplementation, as recent research indicates that the physical fatigue of commuting may be associated with preterm delivery.[23,24] In the long run, solutions must include the provision of permanent and adequate housing.

Acknowledgments. The authors thank Dr Karen Benker, Dr Margaret Grossi, Jean Lee, Ellen Rautenberg, and Dr Alex Tytun for their assistance.

REFERENCES

1. Bassuk EL: The homelessness problem. *Sci Am* 1984; 251(1):40–45.
2. Baxter E, Hopper K: *Private Lives/Public Spaces: Homeless Adults on the Streets of New York City.* New York, Community Service Society, 1981.
3. *Monthly Report: Crisis intervention services.* Human Resources Administration, May 1984.
4. *Homelessness in America.* Testimony before the Subcommittee on Housing and Community Development of the Committee on Banking, Finance, and Urban Affairs, House of Representatives publication No. 97-100, 1983.
5. Slavinsky AT, Cousins A: Homeless women. *Nurs Outlook* 1982; 30:358–362.
6. Sebastian JG: Homelessness: A state of vulnerability. *Fam Community Health* 1985; 3:11–24.
7. Kleinbaum D, Kupper L: *Applied Regression Analysis and Other Multivariable Methods.* Boston, Duxbury Press, 1978.
8. Lincoln R: Smoking and reproduction. *Fam Pl Perspectives* 1986; 18(2):79–84.
9. *Reliability of Statistical and Medical Information Reported on Birth and Death Certificates.* New York State Dept of Health monograph No. 15. Albany, NY, 1979.
10. Friedman E: On the street. Hospitals wrestle with the problem of homeless patients. *Hospitals* 1983; 57:97–98, 102, 105.
11. Kelly E: Homeless young people in cities. *Midwife, Health Visitor and Community Nurse* 1978; 10:357–358.
12. Cousins A: Profile of homeless men and women using an urban shelter. *J Emergency Nurs* 1983; 9(3):133–137.
13. Fennelly C, Klopfleisch S, Hombs ME, et al: The vexing problem of street people. *Public Welfare* 1979; 37:7–63.
14. Young CL, Hall WT, Collins J: Providing health and social services to illegal alien families: A dilemma for community agencies. *Soc Work Health Care* 1979; 4:309–318.
15. Hornbake HF: The migrant child: Coping with a life style. *Children Contemp Soc* 1980; 13:38–41.
16. Simpson J: *Struggling to Survive in a Welfare Hotel.* New York, Community Service Society, 1984.
17. Wingert A, Halfman P: Migrant health question. *Pediatr Nurs* 1979; 5(6):19–20.
18. Blackburn MC: A health profile of homeless vs non-homeless families in Central London. *Health Visit* 1981; 3:300–309.
19. *No One's in Charge: Homeless Families with Children in Temporary*

Shelter. New York, Citizen's Committee for Children of New York Inc, 1982.

20. Marwick C: The sizable homeless population: A growing challenge for medicine. *JAMA* 1985; 253:3217–3219, 3223–3225.

21. Jamieson K, Spencer S: How doctors can help the homeless [letter]. *Lancet* 1978; 2:1308–1309.

22. Fisher E: Homeless families: A scheme of notification to ensure effective care. *Nurs Times* 1980; 76(suppl):77–80.

23. Saurel MJ, Kaminski K: Pregnant women at work. *Lancet* 1981; 1:475.

24. Mamelle N, Lauman B, Lazar P: Prematurity and occupational activity during pregnancy. *Am J Epidemiol* 1984; 119:309.

See also pp 1–3 and 3–5.

Abused Women, Lucille Friedland (Richard Rodamar, photographer), bonded aluminum relief, 16″ × 24″, 1984 (reproduced courtesy of the artist).

EARLY AMERICAN MEDICINE

Visual and artifactual materials in the history of early American medicine

ROBERT I. GOLER, MA

Addressing the need for anatomical experiments five centuries ago, Leonardo da Vinci underscored the importance of nonverbal communication in the study of medicine:

> Dispel from your mind the thought that an understanding of the human body in every aspect of its structures can be given in words; for the more thoroughly you describe, the more you will confuse. . . . I advise you not to trouble with words unless you are speaking to a blind man.[1]

This paper examines attitudes toward disease and treatment in 17th and 18th century America. It emphasizes the importance of the surviving visual and historical materials and suggests how they can improve the understanding of medicine. The materials that hold special significance for this approach are paintings, prints, books, documents, and historical artifacts such as medical instruments and household objects related to medical care. In order to understand American history in its totality it is essential to go beyond documentary sources. By relying solely upon documentary sources researchers may actually lose sight of some of the important components of early American medicine.

What can be learned about American medical history from visual and artifactual materials? If the proper questions are framed of the available evidence, it should be possible to augment our knowledge of history through the study of "things." Given the overall paucity of original source materials for the early American period, the study of material culture should be considered by historians of medicine as an important, largely untapped, resource for examination. In short, material culture can provide insight into the workings of everyday society.[2]

On another level, it is important to recognize the significant dramatic appeal of historical objects. The fascination with George Washington's false teeth (Fig 1) and the Shroud of Turin, to name only two examples, testifies to the extraordinary interest in relics. Whether relics, in and of themselves, constitute "good" history may be questioned, but there can be little doubt of their role as touchstones to the past.

This article focuses on some issues concerning the interpretation of medical practice in American history. These issues represent popular beliefs about colonial medicine and about the role of artifacts in historical study that should be considered by those who investigate such material. While these reflections admittedly are subjective, together they may help to reveal attitudes toward, and prompt new interpretations of, health care in early America.

THE POWER OF THE OBJECT

Few objects have as much presence and immediacy as medical instruments. For most viewers, they trigger the memories of pain and suffering so commonly associated with sickness and disease. It is not always easy

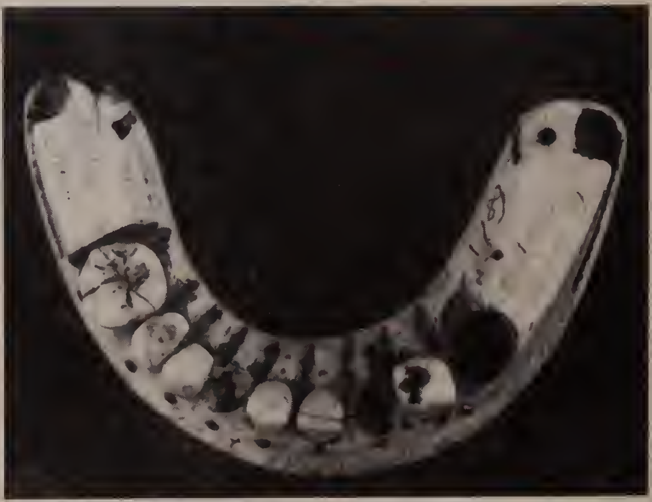


FIGURE 1. *Denture (worn by George Washington)*, John Greenwood (1760–1819), bone, ivory and metal, New York, 1789 (courtesy of The New York Academy of Medicine).

This article is based on a presentation at the symposium entitled, "Interpreting Medical History and Understanding Its Importance in the Study of Early American Life," held April 18, 1986, at Fraunces Tavern Museum, New York, NY. Funding for the symposium was provided, in part, by the New York State Council on the Arts and the New York Council for the Humanities. Additional funding was provided by Patricia Kind and two anonymous donors.

Address correspondence to Mr Goler, Acting Director and Curator of Collections, Fraunces Tavern Museum, 54 Pearl St, New York, NY 10004-2429.

Copyright © 1987, Sons of the Revolution in the State of New York (Fraunces Tavern Museum). Fraunces Tavern Museum is owned and operated by, and FRAUNCES TAVERN® is a registered service mark of, the Sons of the Revolution in the State of New York, Inc, a not-for-profit corporation instituted in 1876 and incorporated in 1884. Funding is provided by corporations, foundations, individuals, state and federal government agencies, and the Sons of the Revolution in the State of New York.

to interpret such instruments, or to portray their importance as historic objects and representations of the technology of the age, and their direct relationship to men, women, and children. As an example, consider the practice of bloodletting, the most misunderstood practice of colonial medicine.

Bloodletting dates from antiquity and probably was the single most continuously employed medical technique in human history.[3] The popular response to its role in the repertoire of past treatments, however, is generally one of disparagement. Can the instruments of bloodletting portray the practice for which they were employed? How was bloodletting actually performed? What instruments were used? How was the patient positioned? Was the procedure painful? The standard use of bloodletting in the 18th century prompted Benjamin Bell, a prominent Scottish physician and author of the influential *A System of Surgery*, to remark, "I can with freedom say, that I have seldom seen bloodletting with the lancet done very correctly."[4(p50)]

Special care was needed to maintain equipment and,

> . . . as the prevention of pain is with most patients a matter of no small consequence, I think it ought to be laid down as a fixed rule never to use the same lancet twice, without putting it into the hands of a cutler."[4(p66)]

One such cutler was William Jasper, who advertised his services in an 18th century New York City newspaper as one who "makes all kinds of Surgeons instruments, and grinds and cleans them."[5]

Blood was taken from virtually every part of the body; hands, tongue, legs, and anus could be tapped. The patient generally was to assume "a horizontal position, either upon a bed or on a couch." (This is shown in the historical print depicting George Washington's death in 1799 (Fig 2), when some 96 ounces of his blood were removed, an amount equal to approximately half of his total blood volume.) The available light was to "fall directly" on the desired site, with candles to be lighted if it were night or the daylight were too dim. Throughout the procedure, Bell remarked, the practitioner "ought always to be seated. . . . When properly performed it is really a neat operation."[4(pp50,52–53)]

The placement and positioning of the healer and the healed were important elements of the procedure, just as they are in surgical and diagnostic activities today. Historical materials can potentially provide the viewer with visual representations of the nature and substance of medical care as practiced in a past generation.

THE ROLE OF THE COMMUNITY IN EARLY AMERICAN MEDICINE

The popular view of early American medicine is heavily influenced by portrayals of the well-appointed doctor's office and apothecary shop of the 19th and 20th centuries. Regrettably, historians generally have given short shrift to the experiences of the 17th and 18th centuries, in part because little artifactual and documentary evidence survives and because the achievements of the colonial period pale in comparison with the "successes" of the 19th century. In addition, medicine in 18th and early 19th century America was a less formalized institution than in the era following the development of the "doctor's office." Doctors in the 18th and 19th centuries spent much of their time visiting patients at home. For example, it was not unusual for Peter Middleton, a prominent New York physician, to visit ten to 12 patients in a single day.[6] Thus, the physical setting for medical treatment, which in the late 19th and 20th centuries was so firmly located in offices and hospitals, was far more informal in early America.[7]

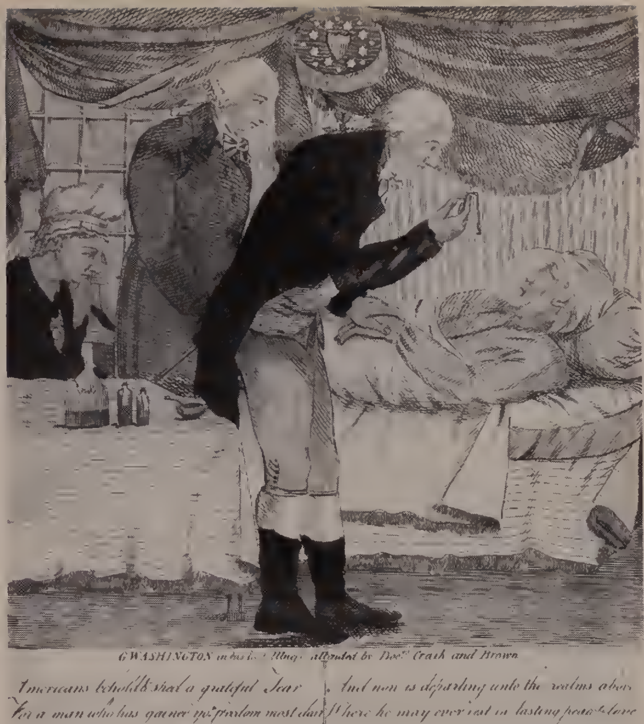

G WASHINGTON in his last Illness attended by Docrs. Craik and Brown
Americans behold! & shed a grateful Tear
For a man who has gained yr. freedom most dear
And now is departing unto the realms above
Where he may ever rest in lasting peace & love

FIGURE 2. *G. Washington in his last Illness attended by Docrs. Craik and Brown*, unknown artist, etching with hand coloring, America, 1800 (courtesy of the Museum of Fine Arts, Boston).

With very few exceptions, healing in the 17th, 18th, and early 19th centuries was practiced in the home. There were few, if any, trained physicians in colonial communities. Home remedies were the mainstay of most family treatments, often gleaned from available publications such as Buchan's *Domestic Medicine*[8] or Wesley's *Primitive Physick*,[9] or from the advice of neighbors and relatives. Cary Carson has remarked on the central importance that domestic materials, especially those from "the hearth," can play in the representation of historical lifestyles.[10] Artifacts from the home are most likely to impart an accurate appreciation of colonial medical practices (Fig 3).

The majority of early Americans lived in rural areas where access to educated practitioners was limited. Medical care was largely a collective effort of families, neighbors, relatives, and local healers, with the occasional participation of doctors. On the morning of August 6, 1793, for example, a doctor named Samuel Adams, who had been trained as an apprentice and had been a surgeon in the Revolution, rode to see "the wife of Isaac Stanwood in travail with her 7th Child" in rural Massachusetts. Following his visit he "called on Mrs. Hodgkins & [Mrs] Pindar to come to Mrs. Stanwood's" to care for the expectant mother during the day, probably so that Adams could re-

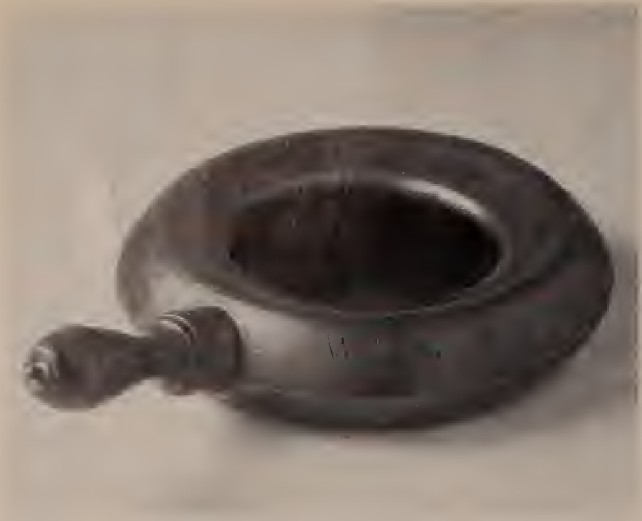


FIGURE 3. *Bedpan*, William Will (1742–1798), pewter, Philadelphia, ca. 1764–1798 (courtesy of the Henry Francis du Pont Winterthur Museum).

turn to his home which he operated as a tavern. When he returned later that day, Adams

> . . . found Mrs. Stanwood's pains encreased [sic], and soon after my return while she was standing on the floor and taking a Pain in that position, the membranes burst and I delivered her the same pain & in that position of a fine daughter.[11]

This complementary relationship between Adams, Mrs Hodgkins, and Mrs Pindar reflected the common need for ordinary people to provide primary medical care to others in their community. Neighbors and friends, artisans such as the cutler who sharpened lancets, women who collected leeches (Fig 4), and the merchant who sold toothbrushes were important participants in the health care networks of early American communities.

Another example comes from the diary of Lewis Miller, which offers a delightful account of daily life in the rural community of York, Pennsylvania. In one incident described by Miller, Old Schrenck, one of the town's residents, borrowed a dovetail saw from the local carpenter in order to remove his wife's overgrown toe nail which, he remarked, had grown "So long and horny two inches long" (Fig 5). To the delight of Miller, who must have seen Schrenck returning from the carpenter's shop with a saw and who perhaps watched through the window at just the right instant, Mrs Schrenck let out a shriek of laughter when her husband grabbed her foot since "it tickled her almost to death. . . ."[12]

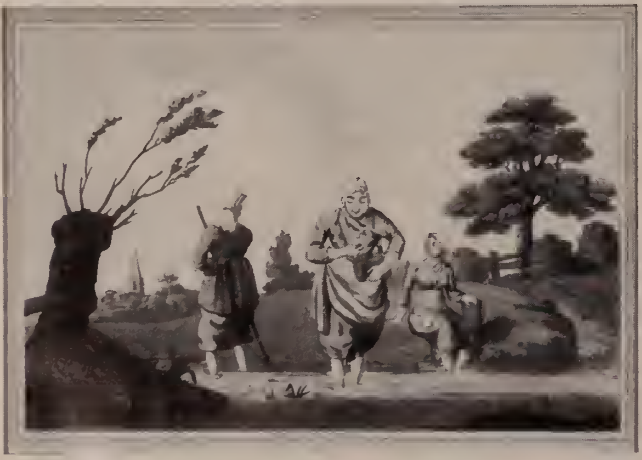


FIGURE 4. *The Leech Finders*, R. Havill after George Walker, color lithograph, published by Robinson & Son, Leeds, England, 1814 (courtesy of the National Museum of American History, Smithsonian Institution).

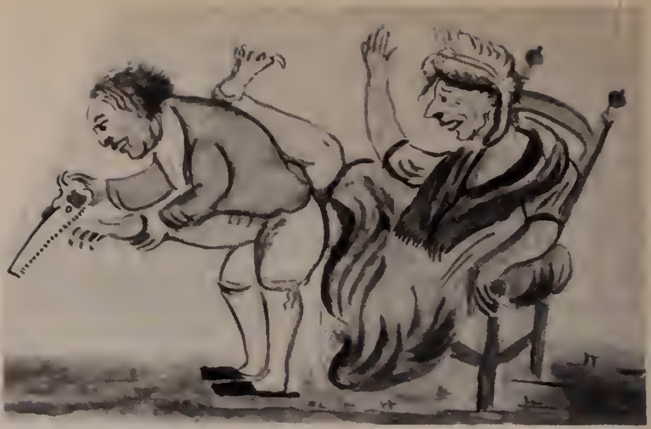


FIGURE 5. *Old Schrenck and His Wife, 1799*, Lewis Miller (1796–1882), watercolor, York, Pennsylvania, early 19th century (courtesy of The Historical Society of York County).

The practice of healing in America was not a uniform practice, for the practitioners did not share common educational backgrounds, experience, or access to therapeutic substances. For the most part, medical practitioners (whom we might more accurately call healers) were not professionals at all. They had no journal, and maintained virtually no common standards for proper education and practice. Only a fraction of the approximately 4,000 "doctors" in America at the time of the Revolution had received medical degrees. Most had apprenticed with surgeons or other healers, while many possibly had no training at all. "In general," summarized the 18th century physician William Douglass,

> the physical practice in our colonies is so perniciously bad, that, excepting in surgery, and some very acute cases, it is better to let nature under a proper regimen take her course (naturae morborum curatrices) than to trust to the honesty and sagacity of their practitioner; our American practitioners are so rash and officious. . . Frequently there is more danger from the physician, than from the distemper;. . . but sometimes notwithstanding of male practice, nature gets the better of the doctor, and the patient recovers."[13]

Medical education in the colonies was a loosely organized enterprise and generally was not lucrative. "A young man without any liberal education," commented William Douglass, "by living a year or two in any quality with a practitioner of any sort, apothecary, cancer doctor, cutter for the stone, bone-setters, tooth-drawer, &c. with the essential fundamental of ignorance and impudence, is esteemed to qualify himself for all the branches of the medical art. . . ."[13] Even those few who were able to travel abroad for study at the intellectual centers found it necessary to compete with apprentice-trained practitioners upon returning from Europe with medical degree in hand. For example, after returning from study in Edinburgh to his native New York where his father was a prominent doctor, Samuel Bard, Jr, confided to a classmate that only

> my connection with my Father greatly lessens the confinement and alleviates the anxiety naturally attendant on our Profession.

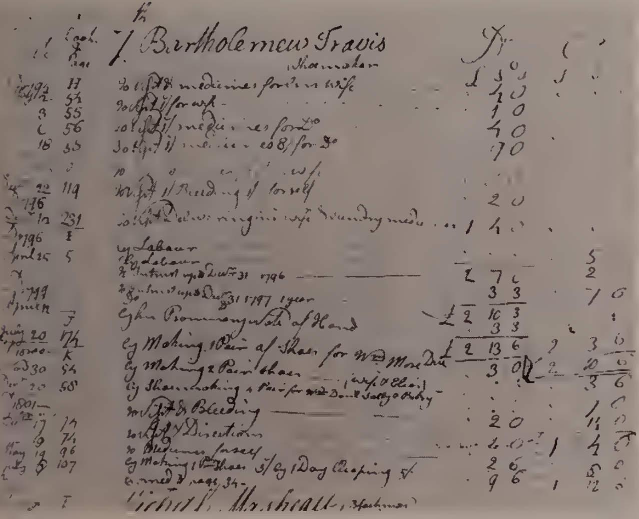
> There is I own one Dissagreeable [sic] Circumstance attending it, that of keeping a shop.[14]

Many doctors found it necessary to do much more than to operate an apothecary shop to maintain their practices. Samuel Adams, for example, a doctor in Ipswich, Massachusetts, turned to tavern keeping (diary entries of March 24–July 12, 1792).[11] And, because of the general shortage of hard currency, most practitioners found it necessary to barter their services. Elias Cornelius of Somers, New York, for example, accepted 15 pounds of veal, one day of repairing a rail fence, and another day of raking hay—over a period of more than a decade—in return for having inoculated seven members of a family against smallpox[15] (Fig 6).

The image of the colonial doctor dressed in "a Scarlet camblet Surtout . . . large overcoat without a cape . . . hair Dress'd in a [wig] bag . . . and at publick assemblies [appearing] with a Sword" exemplified the style sought by many of America's most celebrated practitioners.[16] In Philadelphia, for example, John Redman was renowned for his elegant cloak,[17] and Benjamin Rush carried a gold-tipped cane.[18] These descriptions, though, represent only the most prominent and illustrious members of the medical profession. For the most part, doctors were not a particularly prosperous group during the 18th century. Recent research by art historians indicates that the relatively small number of 18th century portraits of doctors suggests that doctors had little disposable income.[19] Indeed, with the exception of the few hundred individuals who traveled abroad for formal medical education, healers did not generally come from wealthy families.[20]

HISTORICAL MATERIALS ARE NOT NEUTRAL

One historian of medicine has described the 18th century as "the Golden Age of duplicity and charlatanism."[21] This era might be characterized more usefully as exhibiting a tension between rhetoric and science. This may be illustrated by an anecdote from popular literature that appeared in a New York City magazine of 1796:



Bartholemew Travis
Shoemaker
Dr

FIGURE 6. *Account for Bartholemew Travis* (*Shoemaker*), Dr Elias Cornelius (1758–1823), *Ledger Book D* (detail), Somers, New York, 1781–1803 (courtesy of The New York Academy of Medicine).

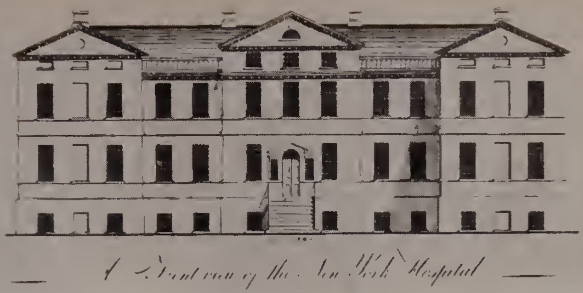

A Front view of the New York Hospital

FIGURE 7. *A Front View of the New York Hospital*, [William] Rollinson (1762–1842) after John Crommelin Lawrence (d. 1791), engraving, New York, ca. 1791 (courtesy of The New-York Historical Society and the Medical Archives, New York Hospital-Cornell Medical Center).

> A Bad Painter, who could never hit off a good likeness, and whose pictures were always returned upon his hands, went off to another country and turn'd physician. One who went that way knew him again, and asked him for what reason he went in the habit [dress] of a physician? "I have a mind (said he) to profess an art, where in all mistakes are covered by earth."[22]

The depiction of the doctor as a dissembling, pretentious fake has a solid tradition in the West. William Schupbach, for example, has ingeniously demonstrated the allegorical meaning of "The Cure of Folly" scenes in 16th and 17th century Europe.[23] This same spirit pervades much of the visual material of the 18th century, particularly in the works of James Gillray, William Hogarth, and Thomas Rowlandson which circulated in America. There often was an element of gallows humor in the characterizations of physicians in early America.

By contrast, the 18th century also saw efforts to improve medical treatment and scientific understanding. Through the colonial period, those who needed medical care but who had no alternative entered almshouses, which were, without exception, institutions of last resort for the poor, the homeless, and the outcast. The overseers of the almshouse in Boston, for example, lamented that "the sick are disturbed by the noise of the healthy, and the infirm rendered liable to the vices and diseases of the diseased, and profligate."[24] Hospitals provided the first medical alternative to the almshouse and were established in Philadelphia and New York during the colonial period. These hospitals were striking structures; their neoclassical designs embodied the community's aspirations toward social and moral order (Fig 7). Those whom medicine could not cure nor architecture inspire were safely confined, sometimes in chains, within the solid walls surrounding the hospital. The practice of visiting the insane wards—lampooned by William Hogarth in his depiction of London's Bethlem (Bedlam) Hospital (Fig 8)—also occurred in America.

The establishment of American medical schools in the third quarter of the 18th century prompted a desire for anatomical study. Dr John Fothergill, a prominent English physician and corresponding member of the American Philosophical Society, had taken an early interest in the improvement of medical education in America. In 1762, he sent to the Pennsylvania Hospital a set of 16 anatomical watercolors by Jan van Rymsdyck "to be under the care of the physicians & to be by some of them explained to the students or pupils who may attend the Hos-

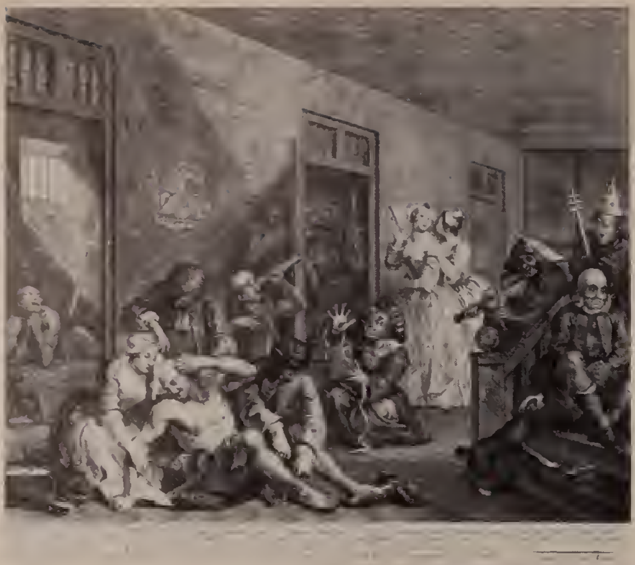


FIGURE 8. *In Bedlam*, William Hogarth (1697–1764), engraving, London, 1735/1763, from *The Rake's Progress* (courtesy of Yale Center for British Art).

pital."[25] Since they were intended for instructional use, these studies were stored in the hospital, where medical students gained practical experience (Fig 9). Only in the 19th century did life drawing attain respectability in America.

Efforts to describe and analyze disease in a scientific manner also emerged in the latter decades of the 18th century. In the 1790s, Valentine Seaman's spot maps pinpointed the outbreaks of yellow fever along the docks of New York, and meteorological tables were published along with accounts of epidemic diseases.[26] These attempts to quantify experience represented a new attitude toward medicine, an attitude that reflected the emergence of a medical profession. This did not mean that popular criticism of doctors stopped with the introduction of professionalism or empirical discoveries—as evidenced by Gillray's parody of vaccination (Fig 10)—but it created a new framework for medical practice that promulgated progress.

INTERPRETATION OF ARTIFACTUAL MATERIALS

> We know it is impossible to transmit behaviour and artifacts from one generation to another, apart from the ideas which give them context and meaning; all that can be conveyed or communicated are ideas through which behavior patterns and artifacts can be manufactured, employed, or valued.[27]

Artifacts are a major aspect of the evidence of past societies and peoples, but they are not infallible records. Significant elements of the past have not survived in material culture, and this affects how the remaining articles are interpreted. Researchers must also take care in the interpretation of visual material to avoid the conscious (and unconscious) distortions that are inherent in historical ob-

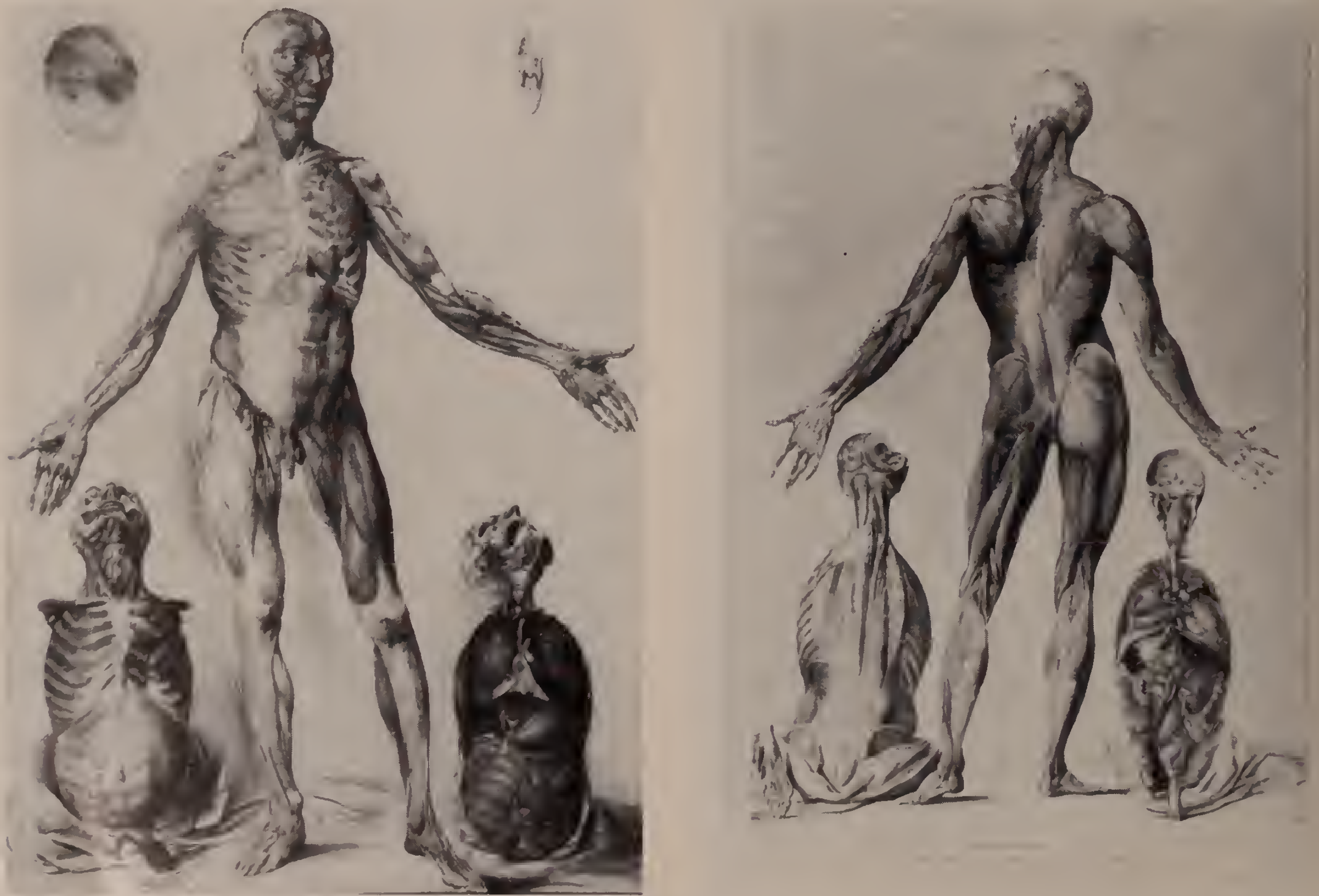


FIGURE 9. *Pair of Anatomical Figures*, Jan van Rymsdyck (fl. 1760–1780), watercolor, London, ca. 1760 (courtesy of Pennsylvania Hospital).

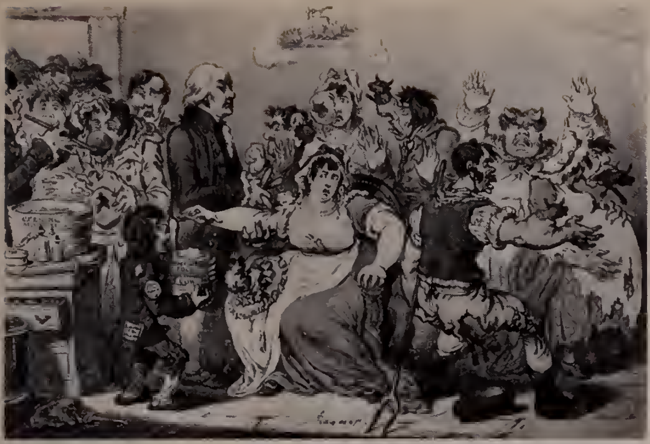


FIGURE 10. *The Cow-Pock, or The Wonderful Effects of the New Inoculaticn!*, James Gillray (1757–1815), engraving with hand coloring, published by H. Humphrey, London, 1802 (courtesy of Yale Medical Library).

jects. Portraits, for example, number among the most misleading sources for exactly the same reasons that they are such powerful records. While a portrait may contain the likeness of an individual, it may also contain inaccuracies. For example, nearly one out of every five individuals who survived smallpox, including George Washington, was scarred by pock marks. Yet portraits of Washington rarely depicted him with pock marks. Caesar Rodney, a signer of the Declaration of Independence from Delaware, was so badly disfigured by cancer that he generally wore a cloth over his face to cover his disfigurement; he was never accurately portrayed, either. The extent to which the marks of disease were visible, however, is suggested in the 1770 notice of "...a stout well-made, very black NEGRO [runaway] man, named WALLEY, about 5 Feet, 9 Inches high, much pitted with the Small-pox."[28]

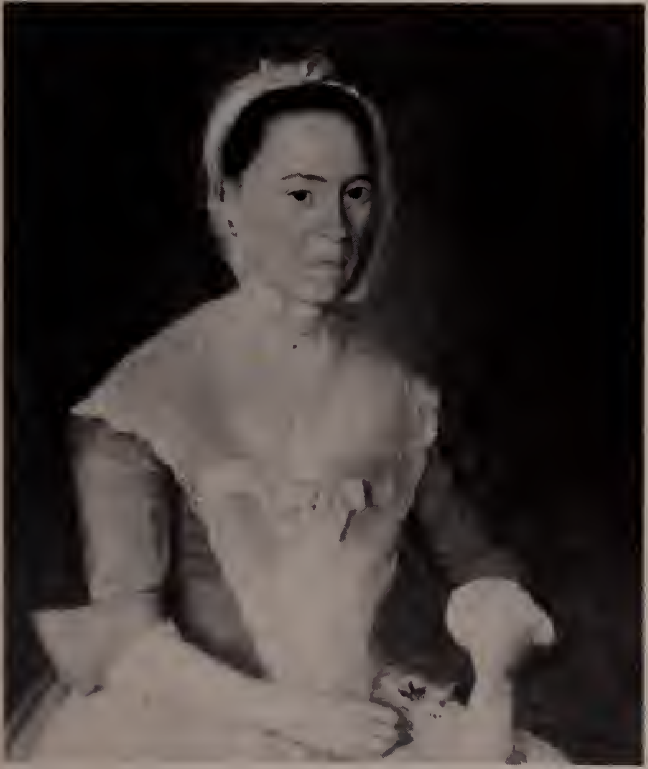


FIGURE 11. *Elizabeth Stiles* (*d. 1775*), Samuel King (1749–1819), oil on canvas, America, 1771 (courtesy of Yale University Art Gallery).

Just as significant as these omissions were the socioeconomic factors that governed the making of pictorial images in early America. For example, since many of the women who were known to have participated in the medical arts were not wealthy, portraits of these female healers were rarely painted in the colonial era. One exception is the portrait of Elizabeth Hubbard Stiles (Fig 11), of Newport, who

> had considerable medical & chirurgical Skill by which she was enabled to do much good. Indeed when she went among the sick she relied more on her Skill than on many of the Physicians—for the latter might be interested, she not.[29]

Yet when Samuel King painted her image in 1771, it was as the wife of a prominent minister and scholar. Except for the possible medicinal use of the flower in her hand, Stiles' portrait little suggests her medical abilities.

As with women, the experiences of children often are overlooked in interpretations of colonial and federal period medicine. Children left few records of their lives and, since they generally were treated in their homes, most of their illnesses cannot be traced. Certainly it was difficult (especially by colonial standards) to accurately determine the nature and severity of a child's ailment since infants and toddlers were unable to describe their complaints.[30] Pewter nursing bottles, made from a lead alloy, may well have contributed to the high infant mortality rates of the colonial era (Fig 12). The constant threat of death may

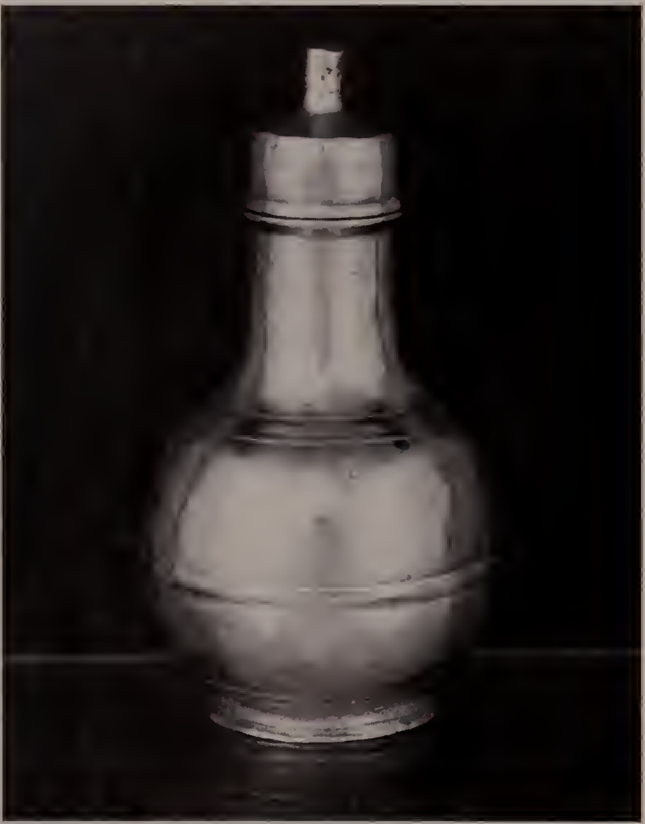


FIGURE 12. *Nursing Bottle*, Frederick Bassett (ca. 1761–1800), pewter, Hartford or New York, late 18th century (courtesy of Mabel Brady Garvan Collection, Yale University Art Gallery).

have conditioned the attitudes of parents and society toward children, as was indicated by Charles Willson Peale when one of his newborn children "had like to have lost her Life & continued excessive ill for several weeks."[31]

For information about the behavior and experiences of children, we have been forced to rely almost exclusively on historical documents. Historians have noted that, in many important respects, the attitude towards children in the 17th and first half of the 18th centuries was mean and "ferocious." One mid-17th century author reflected that children commonly were bound tightly in swaddling clothes to underscore "the stains and pollutions of sin which it [the child] inherits from our first parents through our loins." The harsh reality and strict discipline of the 17th century are foreign to our worldview; yet the vast majority of tracts on child rearing prior to 1700 recommended that fathers beat their children. William Byrd recorded another example of such extraordinary behavior in his remarkable diary: a punishment of drinking "a pint of piss" for a young dependent.[32] Such attitudes, however, are difficult to glean from the surviving artifacts.

Compare the attitudes projected by the contrasting images of a portraitist and a caricaturist. Figure 13 is a portrait of a successful physician, Dr Amos Throop, first president of the Rhode Island Medical Society. In this portrait the viewer can recognize Throop's scholarship and education; his pose and direct gaze, his clothes and his wig, are each artifices intended to reinforce his self-assurance and position in society. Compare Dr Throop's portrait with a series of four dental scenes engraved only a few years earlier (Fig 14). Unlike the portrait—an art form that contained its references internally—these prints were accompanied by satiric verse which helps focus their intent:

[A] A Doctor once much puzzl'd was
To find out ways and means
How teeth to draw of ev'ry class
Without such wracking pains.

A packthread string he ty'd in haste
On tooth, wich sore did wring.
He pull'd, the patient follow'd fast,
Like Towzer in a string.

[B] He miss'd at first, but try'd again,
Then clap'd his foot o'th' chin;
He pull'd—the patient roar'd with pain,
And hideously did grin.

But lo!—capricious fortune frown'd,
And broke the clewkin string,
And threw him backward on the ground,
His head made floor to ring.

[C] An old wife next, with wrapt-up jaw,
And her last tooth, did come:
This tooth, thought he, I soon can draw,
And gain some credit from.

So he the pinchers took in hand,
And pull'd with might and main,
But these slipp'd off, we understand.
Which much'd increas'd the pain.

This made the doctor cast about,
And muse—in doleful dumps:
If fast with large teeth drawing out,
What must I do with stumps?

He puzz'ling star'd; next man, thought he,
I'll try to string again;
The knack I've found most certainly
To do't with little pain.

[D] Now string's put fast on tooth that aches,
Which round his hand he wraps,
A glowing coal i'the tongs he takes,
And to his nose he claps.

The fight and smell of fire drove back
The patient's head in fright,
Who drew his own tooth in a crack,
And prov'd the doctor right.[33]

Which of these images is more accurate: the verisimilitude of the portrait, or the bumbling efforts of the itinerant dentist? Which is more revealing: the medical society's scholarly library, or the dentist's foot against his patient's jaw? In truth, it is not possible to cite one to the exclusion of the other, for they both contain elements of truth and artifice. It is almost as if both sides of the medical visit are depicted here: The portrait shows the doctor as he would like to be seen, while the engravings depict patients as they no doubt felt, grimacing with pain. Together, these images reveal more than the sum of their individual parts.

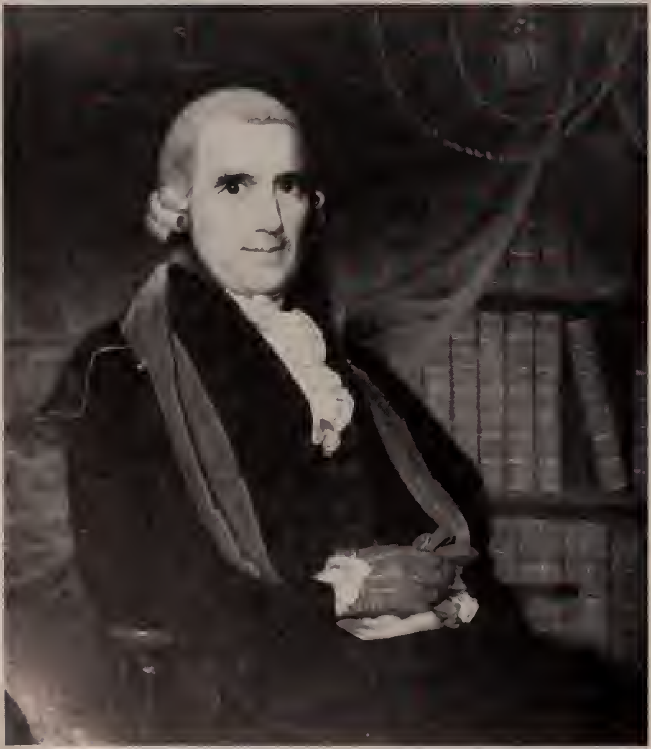


FIGURE 13. *Dr Amos Throop (1735–1814)*, unknown artist, oil on canvas, Rhode Island, ca. 1790 (courtesy of Rhode Island Medical Society).

CONCLUSION

In recent years, historians have made substantial contributions to our understanding of the history of medicine, especially with regard to social history. Yet, for all of this work, remarkably little attention has been given to the role of artifacts and visual sources. I would like to suggest two avenues that medical historians can pursue to augment our understanding of American history. These suggestions build upon the existing strengths of the history of medicine and, if properly implemented, would bring the excellent work in this field to a wider audience.

First, we must assess the role of historical exhibitions

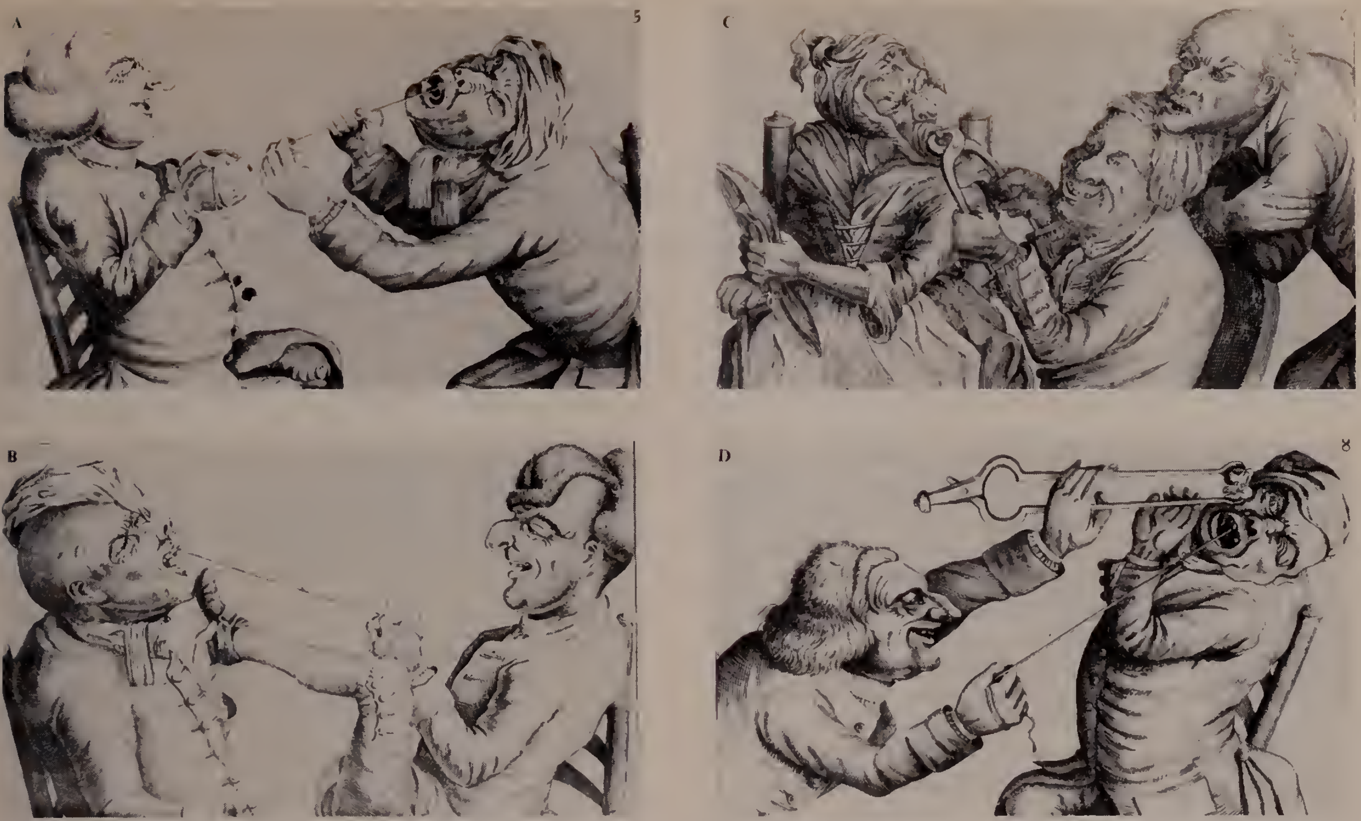


FIGURE 14. *Four Dental Scenes*, T. Sanders after Timothy Bobbin (pseud.), engravings from *Human Passions Delineated*, England, 1773 (courtesy of National Library of Medicine).

and collections management. In general, medical history has been approached as an "internal" discipline. By this I mean that its emphasis has been on how medical knowledge was developed, used, and expanded; perhaps more than other types of historical collections, medical history museums comprise specialized groups of objects, such as pathological or anatomical specimens. Objects that represent technological advancement, as well as associations with famous individuals, also have been important to the growth of collections. Interpretations of medical history from an "external" point of view have begun to provide important additions to general investigations of American history.[34] Detailed studies of medical account books, day books, and inventories would give a better foundation for interpreting the daily life and activities of colonial healers. A clearer perspective on the relations of climate, clothing, and diet to medical treatment would be of immense value. To some extent the material culture of 18th century American medicine, including imported drugs and instruments (Fig 15), can be seen as components of the "consumer revolution" that seems to have motivated much of that era.[32,35]

Second, it would be useful to improve the documentation of medical artifacts. In general, medical artifacts have been preserved and interpreted as examples of technology and medical progress. Medical history traditionally has developed in an opposite fashion from that of the decorative arts, which has emphasized the point of view of connoisseurship. Stylistic developments, requiring detailed examination of and specific attention to makers' marks and materials, have been the guiding forces in art history. By contrast, medical history has been approached from a technical and historical perspective and those objects and materials to be preserved generally have symbolized "progressive" changes or associations with important individuals or events. Perhaps these two approaches can converge.

For this to happen, medical objects need to be approached as artistic creations. By examining the objects of

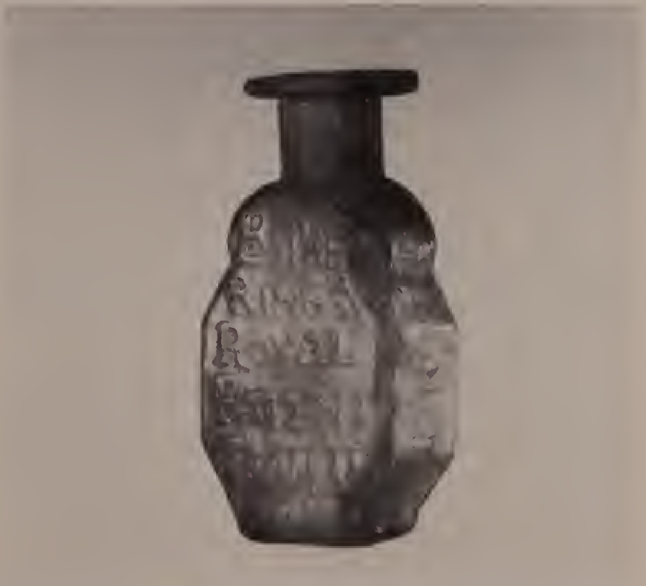


FIGURE 15. *Balsam Bottle*, Robert Turlingham, blown and molded glass, England, ca. 1755 (courtesy of Corning Museum of Glass).

medical history as "creations" rather than as "technology," it may be possible for them to serve as sources for new ideas rather than as illustrations for documentary studies. Standard cataloging procedures need to be adopted to assemble the rough data needed for in-depth study: makers' marks, materials, and provenance. The directory of makers in Elizabeth Bennion's[36] recent study of European medical instruments provides another useful starting point, but it is incomplete and largely ignores American makers. Some projects currently underway to document American makers of medical instruments may help in this endeavor.[37]

Expanding and improving cataloging procedures also would increase the utility and interpretations of medical history collections. For example, a cataloging system devised by the Dittrick Museum is modeled after the National Library of Medicine's Medical Subject Headings indexing classifications (MeSH) used in medical bibliography and promises to provide new points of access to collections.[38] Such information might better enable us to understand instruments and related materials as well as the complex commercial networks between, on one side, doctors and dentists, and, on the other, apothecaries, carpenters, cutlers, merchants, and gold, silver, and whitesmiths.

To garner a wider audience, medical historians should consider process in addition to progress, emphasizing the experiences of people, not only individuals. Such an effort often requires the cooperation of historians knowledgeable about medical and social history, as well as curators and educators. In a sense, this is an extension of the discussion of whether medical history should be written by physicians or historians.[39] A vivid example of the growing appeal of such an approach is the recent recreation and installation of the public hospital at Colonial Williamsburg.[40]

The major challenge before us is the study of the history of American medicine, but even more importantly, it is the study of medicine in American history.[41]

REFERENCES

1. Karp DR, et al: *Ars Medica: Art, Medicine, and the Human Condition.* Philadelphia, Philadelphia Museum of Art, 1985, xi.

2. On material culture studies see Schlereth TJ: Material culture studies in America, 1876–1976, in Schlereth TJ (ed): *Material Culture Studies in America.* Nashville, American Association for State and Local History, 1982, pp 1–75; Kouwenhoven JA: *Half a Truth is Better Than None: Some Unsystematic Conjectures about Art, Disorder, and American Experience.* Chicago, University of Chicago Press, 1982, p 25.

3. Davis A, Appel T: *Bloodletting Instruments in the National Museum of American History and Technology.* Washington, Smithsonian Institution Press, 1979.

4. Bell B: *A System of Surgery* (4 vols). Worcester, Isaiah Thomas, 1791, vol 1.

5. *The New York Gazette*, August 29, 1763.

6. Middleton P: Ledgers B and C, in Middleton papers, Historical Documents Collection, Queens College, The City University of New York; Hershkowitz L: Powdered tin and rose petals: Myer Myers, goldsmith and Peter Middleton, physician. *Am Jewish Hist* 1981, 70:462–467.

7. Flexner JT: *Doctors on Horseback: Pioneers of American Medicine.* New York, Garden City Publishing Co, 1939.

8. Buchan W: *Domestic Medicine: or, the Family Physician.* Philadelphia, printed by John Dunlap for R. Aiken, 1772. From 1769 to 1871 at least 142 editions of Buchan's work were published. Rosenberg CE: Medical text and social context: Explaining William Buchan's *Domestic Medicine. Bull Hist Med* 1983; 57:22–42.

9. Wesley J: *Primitive Physick.* Philadelphia, printed by Pritchard & Hall for Dickens, 1789.

10. Carson C: Doing history with material culture, in Quimby IMG (ed): *Material Culture and the Study of American Life.* New York, WW Norton & Co, 1978, p 59.

11. Adams S: Diary, in Samuel Adams papers, Manuscript Room, The New York Public Library. Dr Adams was not related to the revolutionary politician from Boston with the same name. I am grateful to Kym S. Rice for bringing this source to my attention. (For a discussion of the relationship between rural midwives and doctors, see Ulrich LT: Martha Moore Ballard and the medical challenge to midwifery, unpublished manuscript.)

12. Turner R (ed): *Lewis Miller, Sketches and Chronicles: The Reflections of a Nineteenth Century Pennsylvania German Folk Artist.* York, Pa, The Historical Society of York County, 1966, p 46.

13. Blake J: Diseases and medical practice in colonial America, in Marti-Ibanez F (ed): *History of American Medicine: A Symposium.* New York, MD Publications, 1959, p 43.

14. Bard S: Letter to John Morgan, January 21, 1767, in Bard papers, Historical Collections, College of Physicians of Philadelphia.

15. Cornelius E: Ledger D, account of Frederick Devoo Sr, p 75, in Cornelius papers, Rare Book Room, The New York Academy of Medicine.

16. Bard J: Letter to Samuel Bard, Jr, January 13, 1765, in Bard papers, Rare Book Room, The New York Academy of Medicine.

17. Bell WJ: John Redman, Medical Preceptor, 1722–1808, in *The Colonial Physician and Other Essays.* New York, Science History Publications, 1975, p 27.

18. Novotny A, Smith C (eds): *Images of Healing: A Portfolio of American Medical & Pharmaceutical Practice in the 18th, 19th, & Early 20th Centuries.* New York, Macmillan Publishing Co, Inc, 1980, p 23.

19. Gerdts WH: *The Art of Healing: Medicine and Science in American Art.* Birmingham, Ala, Birmingham Museum of Art, 1981, pp 4–5.

20. Malloch A: *Medical Interchange Between the British Isles and America Before 1801* (publisher unknown), 1947.

21. Saffron MH: The eighteenth century, in Lyons AS, Petrucelli RJ (eds): *Medicine: An Illustrated History.* New York, Harry N. Abrams Inc, 1978, p 485.

22. Anonymous anecdote. *The Time Piece; and Literary Companion*, May 26, 1796.

23. Schupbach W: A New Look at *The Cure of Folly. Med Hist* 1978; 22:267–281.

24. Grob GN: *The State and the Mentally Ill.* Chapel Hill, University of North Carolina Press, 1966, p 6.

25. Scott JA: Concerning the Fothergill pictures at the Pennsylvania Hospital. *Univ Penn Med Bull* 1903–1904; 16:388–393.

26. Seaman V: An Inquiry to the Cause of the Prevalence of the Yellow Fever in New-York. *The Medical Repository* 1798, 1:315–332; Stevenson LG: Putting disease on the map: The early use of spot maps in the study of yellow fever. *J Hist Med* 1965, 20:226–261. I would like to thank Ramunas Kondrates for this reference.

27. Becklow SM: Culture, history and artifact, in Schlereth TJ (ed): *Material Culture Studies in America.* Nashville, American Association for State and Local History, 1982, p 116.

28. Spruill JC: *Women's Life and Work in the Southern Colonies*, 1938, reprint ed. New York, WW Norton & Co, 1972, p 207.

29. Stiles E: *Literary Diary of Ezra Stiles, 1727–1795* (3 vols). New York, Charles Scribner's Sons, 1901, vol 1, p 565. I am indebted to Laurel Thatcher Ulrich for this reference.

30. Caulfield E: Some common diseases of colonial children, in: *Publications of the Colonial Society of Massachusetts.* Boston, Colonial Society of Massachusetts, 1951, pp 4–65; George MD: *London Life in the Eighteenth Century.* New York, Capricorn Books, 1965, p 51.

31. Miller LB (ed): *The Selected Papers of Charles Willson Peale and his Family.* New Haven, Yale University Press, 1983, vol 1, p 27. For more detailed examinations of the attitudes toward death in early America see Hijiya JA: American gravestones and attitudes toward death: A brief history. *Proc Amer Philosophical Soc* 1983, 127:339–363; Ludwig AI: *Graven Images: New England Stonecarving and Its Symbols, 1650–1815.* Middletown, Wesleyan University Press, 1966; Rumford BT: *The Role of Death as Reflected in the Art and Folkways of the Northeast in the Eighteenth and Nineteenth Centuries*, MA thesis. SUNY College at Oneonta, Museum Studies Program, Cooperstown, NY, 1965.

32. Plumb JH: Commercialization and society: The new world of children, in McKendrick N, Brewer J, Plumb JH: *The Birth of a Consumer Society: The Commercialization of Eighteenth-Century England.* Bloomington, Indiana University Press, 1985, p 287.

33. [Collier J]: *The Human Passions Delineated.* London (publisher unknown), 1773, plates 5–8.

34. On the issue of medicine and social history see Reverby S, Rosner D (eds): *Health Care in America: Essays in Social History.* Philadelphia, Temple University Press, 1979.

35. Hoffman R (ed): *Of Consuming Interest: Style in the Eighteenth Century.* Charlottesville, University of Virginia Press, forthcoming.

36. Bennion E: *Antique Medical Instruments.* London, Sotheby Parke Bernet, 1979, pp 304–342.

37. Davis A, Dreyfuss M: *The Finest Instruments Ever Made: A Bibliography of Medical, Dental, Optical and Pharmaceutical Company Trade Literature, 1700–1939.* Arlington, Mass, The Printers' Devil, forthcoming; Edmondson J: Surgical instrument makers in 19th century America (in preparation); Goler RI: Medical personalities in 18th century New York City (in preparation).

38. Gerstner P, Chelnick J: The classification of artifacts at the Howard Dittrick Museum. *Caduceus, A Museum Quarterly for the Health Sciences* 1986, 2(2):39–43.

39. For example, see Grob GN: The social history of medicine and disease in America: Problems and possibilities. *J Social Hist* 1977, 10:391–409; Numbers RN: The history of American medicine: A field in ferment. *Rev Amer Hist* 1982, 10:245–263; Tomes N: The social transformation of American medicine: An historical perspective. *Sociology of Health & Illness* 1985, 7:248–259.

40. Zwelling SS: *Quest for a Cure: The Public Hospital in Williamsburg, 1773–1885.* Williamsburg, Colonial Williamsburg Foundation, 1985.

41. For a discussion of a similar situation in a parallel discipline, see Hounshell D, Stapleton DH: The discipline of the history of American technology: An exchange. *J Amer Hist* 1982, 68:897–900.

Women healers and domestic remedies in 18th century America: The recipe book of Elizabeth Coates Paschall

ELLEN G. GARTRELL, AM, MS

Sometime in the middle of the 18th century Elizabeth Coates Paschall of Philadelphia recorded the following medical advice in a small suede-covered volume:

> For an Inward Fever
>
> make a Whey of Boyld Milk with a Large head of house Leek Boyld in it. pound or bruise the house Leek first & putt it into a pint of Boyling hot milk & Lett it Simmer a few minutes over the fire . . . then Strain it & drink halfe a pint Every Night when you Go to Bed. I advised my Neighbour Vernon to Give this to her Eldest Son, who was Reduced that he Could Scarce Creep 200 foot with a stick to Support him & this quickly Coold & abated the Inward Fever & Cured him, tho the Docter Could not help him & it has Cured Henry Hartleys wife & Severall others. [Recipe #44; numbering supplied by E. Gartrell][1]

This recipe is one of over 200 that Mrs Paschall wrote in her notebook. In doing so she participated in the long tradition of lay persons' receiving, recording, using, and sharing medical information. Over the centuries in Europe and America most medical care has taken place in the home, and women have played a major role in it, caring for family, friends, neighbors, and servants or slaves. Oral tradition was the repository of most advice, but the literate of the 17th and 18th centuries sometimes consulted published guides or wrote down favored remedies for personal use or for sharing with others.

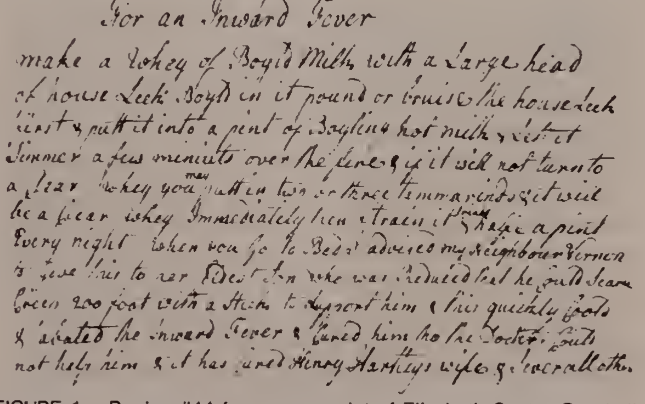
By far the predominant role of 18th century women in medicine was in domestic practice, caring for members of their own households and sharing in the care of members of the community. From studying diaries and letters mainly from Philadelphia, it is clear that women provided both medical and emotional support to those they cared for. The mainly female activity of visiting sick neighbors and attending at births and deaths was part of a network of social interactions that helped cement family and community ties.[2]

In the 18th century American colonies, lay persons' involvement in diagnosis and treatment of major and minor medical problems was routine at all levels of society, whether or not physicians were available. This fact is widely acknowledged by historians but has been little studied until recently. Research interests traditionally have been focused on the development of the medical profession, institutions, and scientific breakthroughs rather than on the experiences of ordinary people.[3-6] And when women were mentioned at all, sometimes the view was disparaging. Thus one historian wrote as follows about European domestic practitioners:

> Innumerable ladies of the manor, squires' or ministers' wives, went around trying to help the sick. Some were well motivated, with fine humanitarian impulses, others were officious busybodies who enjoyed imposing their will and their remedies on hapless parishioners. These well-meaning people knew little, and their practice was the crudest, but at least there was no financial gain involved.[7]

Usually the care was offered on an exchange basis or as charity, with no money changing hands. Published works for lay persons might exhort Christian gentlewomen to "help the Poor in their Afflictions," by which acts they might "gain Good-Will and Wishes, entitle themselves to their Blessings and Prayers, and also have the Pleasure of seeing the Good they do in this world, and have good Reason to hope for a Reward in the World to come."[8]

A few women entered into medical roles for pay, serving as midwives, nurses, herbalists, faith healers, or even—rarely—as physicians. Only midwifery and nursing were



For an Inward Fever
make a Whey of Boyld Milk with a Large head
of house Leek Boyld in it pound or bruise the house Leek
first & putt it into a pint of Boyling hot milk & Lett it
Simmer a few minutes over the fire & if it will not turn to
a clear whey you may putt in two or three Tammarinds & it will
be a clear whey Immediately then Strain it & take a pint
Every night when you Go to Bed I advised my Neighbour Vernon
to Give this to her Eldest Son who was Reduced that he Could Scarce
Creep 200 foot with a Stick to Support him & this quickly Coold
& abated the Inward Fever & Cured him tho the Docter Could
not help him & it has Cured Henry Hartleys wife & Severall others

FIGURE 1. Recipe #44 from manuscript of Elizabeth Coates Paschall (courtesy of The College of Physicians of Philadelphia).

This article is based on a presentation at the symposium entitled, "Interpreting Medical History and Understanding Its Importance in the Study of Early American Life," held April 18, 1986, at Fraunces Tavern Museum, New York, NY. The research for this paper was supported in part by the National Endowment for the Humanities through a summer stipend, 1985.

Address correspondence to Ms Gartrell, Manuscript Department, Duke University Library, Durham, NC 27706.

Copyright © 1987, Sons of the Revolution in the State of New York (Fraunces Tavern Museum).

the near-exclusive domain of females in the colonial period, though by the 1760s male midwives or accoucheurs were beginning to attend at births in urban areas.[9] Good midwives commanded considerable respect but many were probably not literate and so left few personal records of their work. The occasional exception is prized by historical researchers, such as the diary of Martha Moore Ballard of Maine, whose diary documents her attendance at 996 births over a period of 27 years.[10] Even less information can be gleaned about nurses, who also tended to be women of little education and were of lower status than midwives.[11] Most glimpses of the work of midwives and nurses come from passing references to their activities in the surviving letters and diaries of their patients, a limited and skewed sampling at best.

Women sometimes turned their gardening, cooking, and distilling skills to profit by selling herbs or medicines. Some of them advertised in newspapers. The pages of the *Pennsylvania Gazette*, for example, contain advertisements for a gout ointment offered for sale by one Barbara Grant (July 31, 1746) and a cure for the "Scald head" (cradle cap) by Hannah Pearson (February 1, 1759). A few popular patent medicines bore women's names, such as Mary Bannister's drops, which were marketed into the 19th century. Occasionally evidence other than advertising documents a woman's activities as a vendor of medicinals. The will of one Grace Lloyd of Chester County, Pennsylvania, for example, lists several stills and apothecary jars and vials in quantities that seem to indicate that she prepared medicines for sale.[12]

A few women had reputations as physicians, though like most of their male contemporaries they undoubtedly had limited training. One intriguing example is that of Martha Brand, who was listed in Philadelphia business directories between 1796 and 1814 as a "doctoress," but about whose practice only fragmentary evidence has turned up.[13]

SOURCES FOR STUDY OF LAY MEDICAL ACTIVITIES IN THE 18TH CENTURY

In an age of limited understanding of disease, medical concerns were prevalent in the daily life of 18th century society. Letters and diaries graphically illustrate how much time people spent worrying about illness, injury, and death, though these sources still reveal but little about the behavior of the unlettered people who made up the majority of the population. Another difficulty with using letters and diaries is that the medical information they contain—though it may be abundant and extremely illuminating—often appears in very low density, thus requiring the reading of an enormous amount of material to locate pertinent information. Some diaries and correspondence are very rewarding, however; the 49-year diary of Elizabeth Sandwith Drinker of Philadelphia is a well-known example which has been published in part.[14,15] In this diary, interspersed with many other topics, the reader finds detailed information about Mrs Drinker's care of her family, her preparation of medicines, and her interaction with prominent physicians. Men's diaries and letters can also be illuminating about women's roles. The diary of Pennsylvania farmer Benjamin Hawley, for example, notes in 1770 that he had a doctor lance his hand and then "got Some Salve & Pills of Rachel." The next year he had the same Rachel bleed a servant and paid her six pence.[16]

Remedy books such as that of Elizabeth Coates Paschall are potentially very rich information sources, but they, too, present problems for the researcher. Though handed down carefully from generation to generation, rather few have survived to the present time. They suffered wear and tear from heavy use, and as their precious advice became outmoded, most were probably discarded. Such books often contain only recipes—mere lists of ingredients with directions for the preparation and administration of medicines. While this information can be of great interest for the study of, for example, herbalism or therapeutics, it is rarely revealing of the context in which the information was gathered, evaluated, and used, for frequently even the compiler's name is unknown, let alone the place, date, and circumstances of compilation. This is where Elizabeth Paschall's manuscript stands out. From her we learn also about sources of medical information and something about its application, in a well-defined social context: the well-to-do Quaker society of colonial Philadelphia. A detailed look at Mrs Paschall's book and the environment in which she created it is very revealing about medical care among the city dwellers of the mid-18th century colonies.

MRS PASCHALL AND HER RECIPE BOOK

Frustratingly few details of Elizabeth Coates Paschall's life are known. She was born in 1702 to Thomas and Beulah Coates. Her father was a Quaker born in England who emigrated to Pennsylvania in 1683. He became a highly successful merchant in Philadelphia and in 1696 married Beulah Jacques (d.1741), a woman of French Huguenot descent. There were ten Coates children, of whom five grew to adulthood.[17] A surviving family ledger shows that Elizabeth and her siblings received some formal education, probably from a private tutor.[18] Upon her father's death in 1719, the 17-year-old Elizabeth inherited £50 in trust and one of his houses on High (now Market) Street, where she later lived.[19]

In 1721 Elizabeth married merchant Joseph Paschall, one of 11 children born to Thomas and Margaret (Jenkins) Paschall.[20] Like the Coates family, the Paschalls were respectable members of the prosperous Quaker establishment in Philadelphia. Joseph, for example, was a member of the Common Council, a justice of the peace, and, with Benjamin Franklin, one of the founders of the Union Fire Company in 1736.[21] Among her friends and neighbors Elizabeth counted the noted botanist John Bartram and members of the prominent Pemberton, Deshler, Mifflin, Norris, and Wister families. Elizabeth and Joseph Paschall had nine children between 1723 and 1740, but only Isaac (1728–1773), Beulah (1732–1793), and Joseph (1740–1795) lived more than two years. Little more is known of the family's life during the years of the marriage; Joseph died at age 43 in 1742, leaving his widow with three children aged 14, 10, and 2. His estate, including the inventory of his shop, was valued at over £3900.[22]

At some point, presumably after she was widowed, Elizabeth took over her husband's business, selling general merchandise. She mentioned the shop several times in her medical manuscript. Some evidence of her business activity survives in two manuscript receipt books[23] and at least one newspaper announcement.[24] She evidently prospered, for in 1748 she built a summer house at Frankford, four miles outside of Philadelphia. (The nucleus of this house, known as Cedar Grove,[25] which survives today, was moved in the 1920s to Philadelphia's Fairmount Park.) When she died in 1767 at age 65, Elizabeth Paschall's estate was valued at over £5,000.[26]

It is not possible to date Mrs Paschall's recipe book precisely, but it appears that she compiled it over a number of years between about 1740 and 1765, as she ministered to her family and community. The small suede-covered volume has 90 pages and contain 203 recipes, of which 12 are for cookery and several are for other household needs such as stain removal; all the rest are medical.

As reflected in the volume, Elizabeth Paschall's medical beliefs were in line with the dominant and conservative tradition based upon classical humoralism and variant theories. Very briefly stated, the central theme of the explanatory models of disease was balance versus imbalance in the body. Constantly interacting with the environment, the body struggled to maintain equilibrium (health) in a complex system affected by factors such as climate, constitution of the individual, and the body's intake and outgo. When something in the bodily system was out of balance, disease resulted. Too much of a particular "humor," for example, might result in an inflammatory disorder. Thus, Elizabeth Paschall attributed bad scabs on her infant brother's face to "Sucking his Mother when She was in a Surfeit of heat" (Recipe #9). Curing disease generally meant restoring the body's balance, often by "regulating the secretions" by pharmacological or mechanical means.[27]

The humorally based medical system, much elaborated, dominated medicine until well into the 19th century. The important point here is that 18th century laymen shared the general belief system with physicians. There was a shared vocabulary and understanding of how diseases came about and how medicines "operated" to "cure" or "give ease." And as historian Charles Rosenberg has emphasized, "A good portion of the actual therapeutic practices of formally-trained physicians overlapped with those employed by untutored lay persons—perhaps not in the consistency and sophistication of the rationalizations that justified them, but in the particular drugs, dosage levels, and the social settings in which their remedies were administered."[28] The vast majority of Mrs Paschall's remedies reflect her implicit acceptance of the rudiments of the prevailing medical and pharmacological theory. Her activities show that the system of medical practice was a fluid one. Through the interaction of oral and written tradition, high culture and low, practitioners and patients, elements were constantly blending and changing to meet perceived needs, as, for example, in the adaptation of European medicine to the climate and resources of America and to the particular medical concerns of the age.

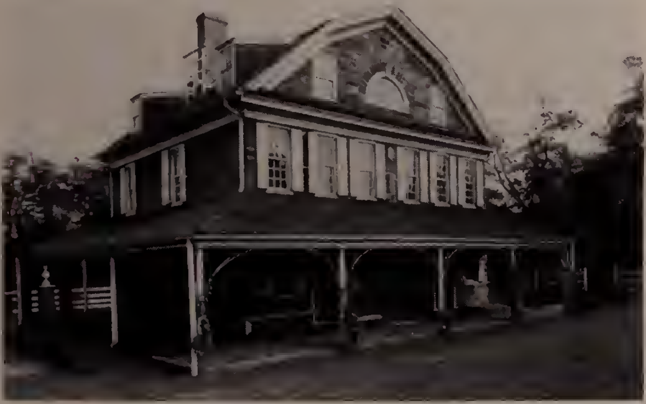


FIGURE 2. Cedar Grove, begun by Mrs Paschall in 1748; left-hand side of house is original section (courtesy of Philadelphia Museum of Art).

Like most personal remedy collections, Elizabeth Paschall's recipe book is not a treatise on health and disease. It is a collection of practical solutions to prevalent problems. Mrs Paschall used 97 different terms to name those problems. The fact that she rarely described or defined the ailments she named suggests that they were familiar to her. A small number of disorders account for over half the total number of medical recipes. The most frequently mentioned were felons, rheumatism, and flux (ten times each). Among others mentioned four or more times were colic, fever, stone, abortion, costiveness, dropsy, and smallpox. The number of cures for felon—an inflammation on the fingertip—is unusually large compared to other contemporary collections, but otherwise the list includes most of the ailments commonly described in the mid-18th century.[29]

It is perhaps surprising to find no references to broken bones or to yellow fever. Fractures must have been a common problem, but many medical guides and recipe books omit them. Yellow fever was mildly epidemic at least three times in mid-18th century Philadelphia,[30] but perhaps it did not strike close enough to Mrs Paschall's family to cause her to secure information about it for her recipe book. She did include a few recipes for two problems that caused great concern in the 18th century: the bites of rattlesnakes and of mad dogs. Many published medical writings also dealt with these afflictions, but the large number of proposed remedies probably reflects the fear they evoked rather than the actual numbers of venomous snakes and mad dogs encountered.

In her remedies, Elizabeth Paschall often used multiple ingredients, usually from two to six items. This polypharmacy, much simplified from practices a century earlier, was fairly typical of both domestic and professional prescriptions of the time. Most frequently she compounded the ingredients herself to make teas, clysters, poultices, or ointments, and she used her concoctions in the usual ways, for example, to cool in fevers, to build up in weakness, to draw out the bad humors of an abscess.

Plant materials accounted for over two thirds of the different substances used. The most frequent were camomile (used ten times in the 191 recipes), pennyroyal (seven times), hyssop and mustard seed (six times each), elder, nettles, and wormwood (five times each). All of these were plants native to Europe and North America that were commonly used in therapeutics.[31] Mrs Paschall used over 70 other plant products. Most of them she listed in her recipes without comment, suggesting that the plants were well known to her. In a few cases, though, she seemed to be adding new plants to her repertoire, as when she described in some detail how to identify or prepare them:

> Take Black berry Bryer Roots that Bares the Large Black black Berrys, the upright Sort not the Creeping Dewberry Kind. . .& white Beach Bark, not the Button wood Sort, . . . John Bartram Sayes there is but one Sort of beech in the Country Besides the water Beech or Buttonwood so that you cannot Mistake the Sort. [Recipe #24]

That sort of note to herself also makes clear that she gathered at least some herbs herself. Some items she must have purchased (eg, imported spices like cinnamon, and possibly some patent medicines), but she made no specific reference to apothecaries or other suppliers.

Although Mrs Paschall used a greater number of different botanicals than any other type of ingredient, she used some nonplant materials with greater frequency. The most common were animal parts (27 times), milk products (24 times), alcohol (22 times), beeswax and honey (14 times), and soap (ten times). In some cases these items clearly had a therapeutic intent; in others, the milk, wax, or alcohol appears to have been the vehicle for the therapeutic agent. As with plant products, it is notable that the majority of the substances were readily available, part of the usual household stores. Similarly, but not surprisingly, there is no evidence that Mrs Paschall had any special equipment for medical use. She measured with teacups and kitchen spoons, prepared medicinals in cooking pots, bandaged with rags and cloths.[32] The equipment used for medicine, like the herbs that served for cooking and therapeutics, had multiple household functions.

Some therapeutic agents and methods usually associated with 18th century medicine are strikingly absent from Elizabeth Paschall's compilation. There are virtually no references to diet or regimen in disease, and none to mercury, laudanum, or inoculation against smallpox. There is not a single reference to bloodletting, and only one each to cupping, issues, and blisters. The reasons for these omissions are open to conjecture. Perhaps Mrs Paschall disapproved of some of them; perhaps their practice was left largely to physicians or other practitioners, and thus had no place in her personal armamentarium. The only invasive measure mentioned regularly was the lancing of felons and other inflammations.

Nine of the remedies called for the use of feces or urine of humans or animals. This was a practice that was gradually dying out in the orthodox medicine of the 18th century. The *Dreckapotheke* of Christian Franz Paullini (1643–1712) had its last edition in 1748, and by mid-century filth remedies were being removed from the pharmacopoeias,[33] yet the substances continued to show up in popular medicine, most often for external applications, for bathing or poulticing wounds. The reasons for their use may have included their ready availability, and possibly the idea of drawing out bad humors with bodily substances that had already been removed—in a natural way—from the body.[34]

There are only four remedies in the manuscript that could be called magical: They rely on a curative method that does not fit the prevailing view. This number of magical recipes is very small, representing only about 2% of the total. And unlike many of the other recipes, there is no evidence that Mrs Paschall actually tried them herself or recommended them to others. In fact, three of the magical recipes are crossed out in the original manuscript, which suggests that someone disapproved of them. All four of the recipes involve some form of "transference" of disease, one of the most common forms of magical cure.[35,36] Recipe #21, for example, is a transference cure for fever and ague (malaria):

> I was informd by an Intimate Acquaintance of mine of their own Experience, that take the Urin of the person when the Ague fitt is on them & mix it with milk, Sugar & Butter or any Clean fatt & flour & Bake a Cake of it under the ashes & Give it to a Dog. Certainly Cures the person, But take Care that no Child or any person Eates it, for I Beleive the Consequence would be bad & it may Give the Dog the mange.

Only one recipe, copied from a British magazine of 1752, makes any reference to astrology. That recipe (#173) makes the following recommendation in treating the bite of a mad dog: " . . . the Patient Should be kept warm & the Medicine to be Repeated Every New & full moone for two or three times . . ." This advice is typical of the simplistic remnants of advice that survived from the complex intellectual system of astrology that had formerly held a prominent place in academic medicine. Most astrological remedies in circulation in the mid-1700s were for mysterious ailments such as epilepsy and mad dog bites, for which no good explanation was available and which were traditionally associated with the lunar cycles.[37]

SOURCES OF MRS PASCHALL'S MEDICAL KNOWLEDGE

Elizabeth Paschall obtained the medical knowledge which she recorded from a number of different sources: physicians, various lay persons, and a variety of publications. She received information from physicians both directly and through third parties. A page in the manuscript daybook of Philadelphia physicians Thomas and Phineas Bond lists one visit and 20 prescriptions for "The Widow Paschall" in a seven-month period in 1755.[38] The recipe book records 38 encounters with doctors by Mrs Paschall or her acquaintances. In almost half the instances, the doctor's advice was accepted and proved satisfactory to the patient. Mrs Paschall mentioned seven different doctors by name, but only in cases where his remedy succeeded. Dr John Kearsley, Sr (1684–1772), a prominent Philadelphia physician, was mentioned four times, more often than any other. The doctors who—in her word—"faild" remained anonymous, but Mrs Paschall expressed no ill feeling toward the unsuccessful practitioners. In 18 instances, she reported on a home remedy that succeeded where one or more doctors had failed to effect the desired cure. These cases included ailments she labelled as piles, "inward fever," "Violent Pain in the Head," ingrowing toenail, and a bad bruise. There were also three cases where doctors advised surgery that the patient refused. Philadelphians clearly preferred to exhaust other possible cures before submitting to the knife.

Other medically knowledgeable people besides physicians also contributed to Mrs Paschall's store of information. Notable among these was botanist John Bartram, a personal friend. He helped her to identify medically useful plants and also shared several recipes. Bartram's sons Moses and Isaac, both apothecaries in Philadelphia, also made suggestions. A newspaper advertisement in 1740 showed that Mrs Paschall's brother Samuel Coates sold drugs at his shop,[39] so he may also have advised his sister or provided her with medicines. Her brother-in-law John Paschall, who was "known as a doctor,"[20(pp224-225)] also played at least an occasional role in approving medicines for particular uses. Another brother-in-law, John Reynell, a merchant by profession, was active in the management of the Pennsylvania Hospital for nearly 30 years.[40] On at least one occasion, he passed on to Elizabeth an observation he had made at the hospital. There is only one brief reference in the manuscript to a midwife, in attendance on the occasion of a miscarriage.

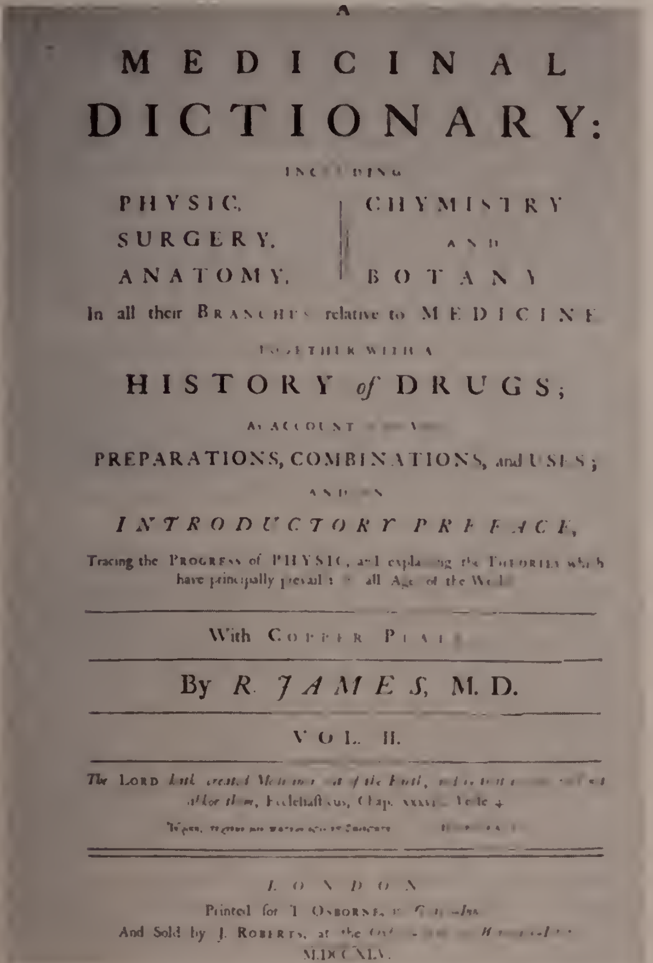
Mrs Paschall also exchanged information, hearsay, and advice with a large number of other people: relatives, neighbors, friends, servants, patrons in her shop, tavernkeepers, Germans, Indians, Negroes, and what she called "antient" women—a broad social network of medical information sharing. In the 191 medical recipes she mentioned over 150 different individuals as patients, givers or receivers of advice, or intermediate parties in the information exchange. Information moved in all directions in society: from washerwomen to heads of household, from tavernkeepers to Indians, from neighbor to neighbor, between men and women. Mrs Paschall was often careful to note the social standing or reputation of people from whom she received intelligence. Thus, she credited the report of a cancer cure described by "a Sober Sollid Country man" (Recipe #16), and was pleased to have her recipe for consumption commended by "a young woman that was well Dressd & Seemd like a person of Credit" (Recipe #24).

Mrs Paschall also got considerable guidance from printed sources: books, newspapers, magazines, and an almanac. Her reading included several standard contemporary scientific and medical texts, among them Boerhaave's *New Method of Chemistry* (London, 1727; 2nd edition, 1741), John Quincy's *Pharmacopoeia Officinalis et Extemporanea; or, a Complete English Dispensatory* (London, 1721; 12th edition, 1749), and Robert Boyle's *Philosophical Works* (she cited the 2nd edition, London, 1738). She also listed several recipes from John Hill's *History of the Materia Medica* (London, 1751) and one from Pliny's *Historiae Naturalis Libri xxxvii* (edition unknown).

More often than any of these works, though, she consulted "James^{es} Grate Dictionary," that is, the weighty three-volume set entitled *A Medicinal Dictionary* by Robert James (London, 1743–1745), which contains extensive theoretical discussions as well as numerous recipes. It was from James, for example, that Mrs Paschall extracted directions for making "Tarr water," which was briefly in vogue as a panacea in Europe and America in the mid-18th century,[41] and three recipes for the use of this concoction in smallpox, bloody flux, and fevers. To the directions she added, as she often did, notes from her own experience and observations:

> Take a Gallon of well or Spring water & 1 quart of Common tarr. Stir them well Togather for 2 or 3 minuets & then lett it Stand for 48 hours & Scum it Clean. then pour of [ie, off] the Clear & Bottle it off for use. take halfe a pint in a morning fasting for a Draught & halfe a pint an hour after Supper . . . Look in James^{es} Dictionary in Letters PEX for Pitch, where the Vertues are Described at Large as Recomended by the Bishop of Cloyn. it was tarr water that Cured our Neighbour Mattias Bush of a Long Lingering wasteing Flux when all other medicine faild. it Corrected the Sharp Humours in the blood & Revived him Like a Cordiall. you must take fresh tarr Every time & throw the tarr away after you have used it butt once. [Recipe # 107]

All of the books Mrs Paschall cited were British editions, which was not surprising given that only about 100 medical works were printed in the American colonies before 1765.[42] Although most of the books she consulted were assuredly available for purchase in Philadelphia around the middle of the century,[43] she most likely did not own them. Twice she indicated that she had John Hill's *History of the Materia Medica* "out of the Library," almost certainly the Library Company of Philadelphia, which had opened its doors in 1732 and quickly built up a substantial collection of medical and scientific works. She also may have borrowed books from personal collections. Her brother-in-law Benjamin Shoemaker, for example, owned at least five medical books, including Quincy's dictionary, which she is known to have consulted.[44] There are no references to cookbooks, which commonly contained medical recipes along with culinary ones, or to medical guides intended for laymen. These genres became more common later in the 18th century.


A
MEDICINAL
DICTIONARY:
INCLUDING
PHYSIC, | CHYMISTRY
SURGERY, | AND
ANATOMY, | BOTANY
In all their BRANCHES relative to MEDICINE.
TOGETHER WITH A
HISTORY *of* DRUGS;
AN ACCOUNT
PREPARATIONS, COMBINATIONS, and USES;
AND AN
INTRODUCTORY PREFACE,
Tracing the PROGRESS of PHYSIC, and explaining the THEORIES which have principally prevail'd in all Ages of the World.

With COPPER PLATES.

By *R. JAMES*, M. D.

VOL. II.

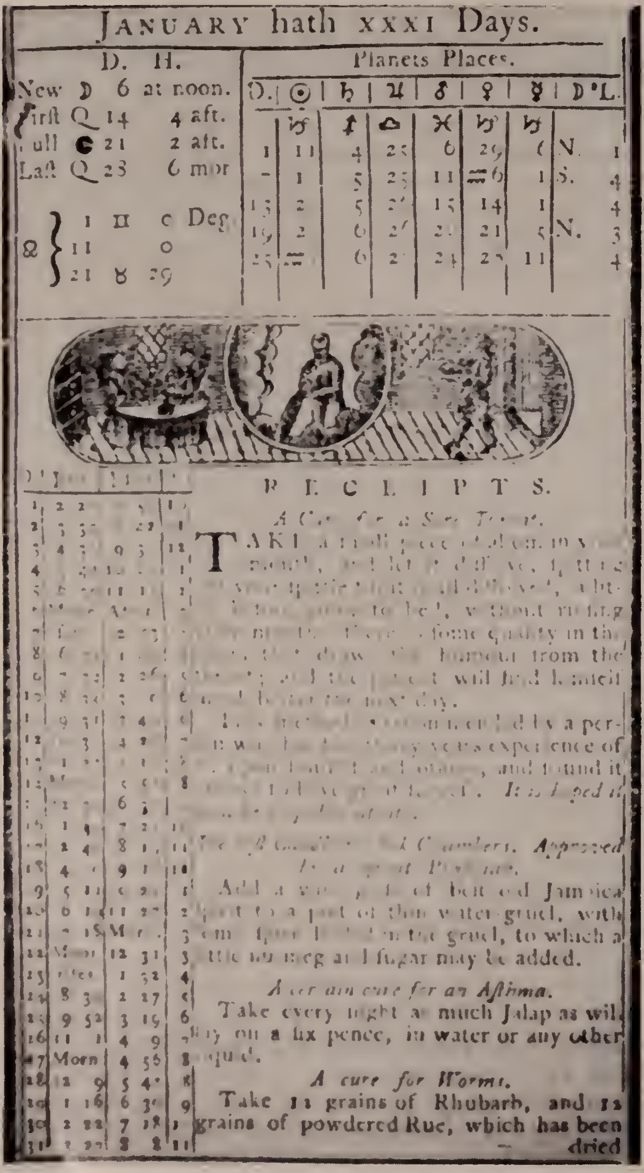
LONDON:
M.DCC.XLV.

FIGURE 3. "James^{es} Grate Dictionary," the source of several recipes copied by Mrs Paschall (from Trent Collection, Duke University Medical Center Library).

The four magazines from which she took five recipes may also have come from the library. Like the books, all the magazines were British: *Gentleman's Magazine*, *Universal Magazine*, *London Magazine*, and *London Chronicle*. They frequently contained medical essays and recipes copied from scientific texts or contributed by laymen. From them she recorded three items on the stone, one on gout, and one on the bite of a mad dog, which were among the most common topics dealt with in 18th century popular medical literature.

At least five recipes came from Benjamin Franklin's newspaper *The Pennsylvania Gazette* and his *Poor-Richard Almanack*. Both newspapers and almanacs commonly carried considerable medical information in the form of hearsay or reprints from standard texts or European periodicals, thus making mainstream medical advice (as well as medical gossip) fairly widely available in American society.

We have seen that Elizabeth Paschall, in common with most women of her time, played an active role in diagnosing and treating ailing members of her family and community. While it is not possible to determine exactly what her standing was as a healer, it does not appear from her remedy book or surviving financial records that she accepted money for her medical counsel or prepared remedies for a profit. Whether she had a particular reputation in the community as a skilled healer or was merely participating as an equal in the broad-based information exchange is also not apparent.



JANUARY hath XXXI Days.

D. H.

New ☽ 6 at noon.
First Q 14 4 aft.
Full ● 21 2 aft.
Last Q 23 6 mor

Planets Places.

RECEIPTS.

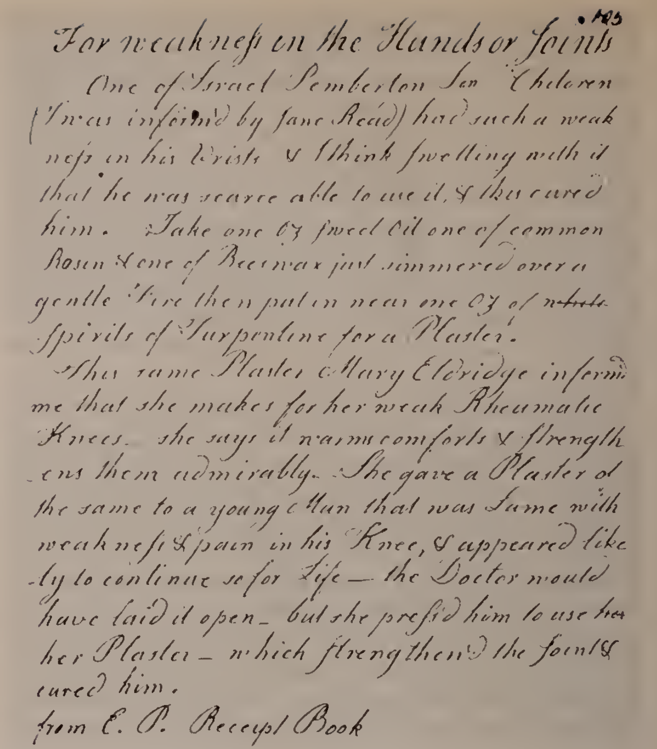
A certain cure for an Asthma.

Take every night as much Jalap as will lay on a six pence, in water or any other liquid.

A cure for Worms.

Take 12 grains of Rhubarb, and 12 grains of powdered Rue, which has been dried

FIGURE 4. Medical recipes in an 18th century Philadelphia almanac.



105

For weakneſs in the Hands or Joints

One of Israel Pemberton Sen Children (I was inform'd by Jane Read) had such a weakneſs in his Wrists & I think ſwelling with it that he was scarce able to use it, & this cured him. Take one Oz ſweet Oil one of common Rosin & one of Beeswax just simmered over a gentle Fire then put in near one Oz of ~~[illegible]~~ Spirits of Turpentine for a Plaster.

This same Plaster Mary Eldridge inform'd me that she makes for her weak Rheumatic Knees. She says it warms comforts & ſtrengthens them admirably. She gave a Plaster of the same to a young Man that was Lame with weakneſs & pain in his Knee, & appeared likely to continue so for Life — the Doctor would have laid it open — but she preſs'd him to use ~~her~~ her Plaster — which ſtrengthen'd the Joint & cured him.

from E. P. Receipt Book

FIGURE 5. Recipe marked "from E.P. Receipt Book," from the late 18th century manuscript based upon Mrs Paschall's compilation (courtesy of The College of Physicians of Philadelphia).

Mrs Paschall left a medical legacy to the next generation in the form of another remedy book which is substantially based upon her own.[45] Its unidentified compiler may well have been Mrs Paschall's daughter Beulah, her daughter-in-law Patience, or some other female relative. It provides a glimpse of the written transmission of information across generations.

The dates of the later compilation were approximately 1770–1807, judging from the dates of the many newspaper clippings that are interspersed with the handwritten entries. The clippings came from a wide variety of publications, including several British papers, and American newspapers from seven cities. The book contains 95 recipes. Eighteen of the 95 were explicitly attributed to "E.P.'s Receipt Book," and several others also match the earlier volume in content. The later book is neither as personal nor as extensive as Mrs Paschall's, but by and large the tone and content of the recipes are virtually identical, reflecting the persistence into the 19th century of domestic medical practice by women based upon the same medical belief system shared by physicians and laymen a generation earlier.

Acknowledgments. The author thanks Charles E. Rosenberg of the University of Pennsylvania for his encouragement and advice throughout the course of this work, and Whitfield J. Bell, Jr, Clarissa F. Dillon, J. Worth Estes, Beatrice Garvan, Nancy Tomes, and staff members at the following institutions in Philadelphia: Historical Collections of the Library of the College of Physicians of Philadelphia, Library of the American Philosophical Society, Library Company of Philadelphia, Historical Society of Pennsylvania, Friends Historical Collection at Swarthmore College.

REFERENCES

1. Paschall EC: *Receipt Book*. Philadelphia, ca. 1749–1765, 90 pp. Manuscript at College of Physicians of Philadelphia Library Historical Collections.
2. Tomes N: The Quaker connection: Visiting patterns among women in the Philadelphia Society of Friends, 1750–1800, in Zuckerman M (ed): *Friends and Neighbors: Group Life in America's First Plural Society*. Philadelphia, Temple University Press, 1982, pp 174–195.
3. Ackerknecht E: A plea for a "behaviorist" approach in writing the history of medicine. *J Hist Med* 1967; 22:211–214.
4. Bynum WF: Health, disease, and medical care, in Rousseau GS and Porter R (eds): *The Ferment of Knowledge: Studies in the Historiography of 18th-Century Science*. Cambridge, Cambridge University Press, 1980, pp 211–253.
5. Porter R (ed): *Patients and Practitioners: Lay Perceptions of Medicine in Pre-Industrial Society*. Cambridge, Cambridge University Press, 1985.
6. Sigerist H: The social history of medicine. *West J Surg Obstet, Gynecol* 1940; 48:715–722.
7. King L: *The Medical World of the Eighteenth Century*. Chicago, University of Chicago Press, 1958, p 31.
8. Smith E: *The Compleat Housewife: or, Accomplish'd Gentlewoman's Companion*. Williamsburg, Va, William Parks, 1742, pp v–vi.
9. Scholten C: "On the importance of the obstetrick art": Changing customs of childbirth in America, 1760–1825, in Leavitt JW (ed): *Women and Health in America: Historical Readings*. Madison, University of Wisconsin Press, 1984, pp 142–154.
10. Ballard MM: *Diary*. Maine, 1785–1812. Manuscript at Maine State Library, cited in Goler RI: *The Healing Arts in Early America*. New York, Fraunces Tavern Museum, 1985, p 41.
11. Norton MB: *Liberty's Daughters: The Revolutionary Experience of American Women, 1750–1800*. Boston, Little, Brown, pp 139–141.
12. Dillon CF: "*A Large, an Useful, and a Grateful Field*": *Eighteenth-Century Kitchen Gardens in Southeastern Pennsylvania, the Uses of Plants and their Place in Women's Work*, PHD thesis. Bryn Mawr College, Bryn Mawr, Pa, 1986, p 130.
13. Bell WJ: Martha Brand (1755–1814): An early American physician. *J Hist Med* 1978; 33:218–219.
14. Biddle HD (ed): *Extracts from the Journal of Elizabeth Drinker, from 1759 to 1807, A.D.* Philadelphia, JB Lippincott, 1889.
15. Drinker CK: *Not So Long Ago: A Chronicle of Medicine and Doctors in Colonial Philadelphia*. New York, Oxford University Press, 1937.
16. Hawley B: *Diaries*, 1759–1782. Manuscript at Chester County Historical Society, West Chester, Pa, as quoted in Dillon CF, op cit, p 130.
17. Jordan JW (ed): *Colonial Families of Philadelphia*. New York, Lewis, 1911, vol 1, pp 650–653.
18. Coates T: *Ledger*, 1705–1726. Manuscript no. Am 9079 at Historical Society of Pennsylvania, folio 11.
19. Coates HT (comp): *Thomas Coates, Who Removed from England to the Province of Pennsylvania, 1683*. Philadelphia (privately printed), 1897.
20. Glenn TA: *Lloyd Manuscripts . . . Welsh Records from the Collection of Howard Williams Lloyd*. Lancaster, Pa, New Era Printing Co, 1912.
21. Coates HT: Cedar Grove, in Glenn TA (ed): *Some Colonial Mansions*. Philadelphia, HT Coates, 1899, c.1897, vol, pp 111–112.
22. Paschall J: *Will*, no. 247, probated March 30, 1742. Archives of the City of Philadelphia.
23. Paschall EC: *Receipt Books*, 1741–1762 (2 vols), in William Henry Russell Collection of Morris Family Papers, 1684–1935, at Eleutherian Mills Historical Society, Wilmington, Del.
24. "Elizabeth Paschall, Shopkeeper, in Market-street . . ." in *Pennsylvania Gazette* no. 909, May 15, 1746, p 3.
25. Eberlein HD, Lippincott HM: *The Colonial Homes of Philadelphia and Its Neighbourhood*. Philadelphia, JB Lippincott, 1912, pp 318–324.
26. Paschall EC: *Will*, no. 210, probated October 18, 1768. Archives of the City of Philadelphia.
27. Rosenberg CE: The therapeutic revolution: Medicine, meaning, and social change in nineteenth-century America. *Perspect Biol Med* 1977; 20:485–506.
28. Gunn JC: *Gunn's Domestic Medicine: A Facsimile of the First Edition, with an Introduction by Charles E. Rosenberg*. Knoxville, University of Tennessee Press, 1986, p xi (reprint of 1830 edition).
29. Rush B: An inquiry into the comparative state of medicine, in Philadelphia, between the years 1760 and 1766, and the year 1805, in his *Medical Inquiries and Observations*, ed 2, vol 4. Philadelphia, J. Conrad, 1805, pp 370–373.
30. Wolman RS: *Some Aspects of Community Health in Colonial Philadelphia*, PHD thesis. University of Pennsylvania, Philadelphia, Pa, 1974, pp 170–174.
31. Grieve M: *A Modern Herbal*, 1931, reprint ed. New York, Hafner, 1959.
32. Sydenham D: *Practitioner and Patient: The Practice of Medicine in Eighteenth-Century South Carolina*, PHD thesis. Johns Hopkins University, Baltimore, Md, 1978, pp 217–218. In a survey of 4,703 South Carolina household inventories from 1739–1785, Sydenham found that 87.19% listed no medical equipment; only 6% definitely included drugs or medical equipment, 5.1% were unclear, and 2.01% listed other related items such as medical books.
33. Ackerknecht E: *Therapeutics from the Primitives to the 20th Century*. New York, Hafner Press, 1973, p 83.
34. See, for example, a remedy reprinted from medical recipes of "J. Cook, M.D." in *Poor Richard Almanack for 1769*, in *The Complete Poor Richard Almanacks Published by Benjamin Franklin*. Reproduced in facsimile with an introduction by Whitfield J. Bell, Jr, 2 vols. Barre, Mass, Imprint Society, 1970. This recipe recommends a poultice of bran wet with stale urine and "sal armoniac" (derived from cow dung), because the latter ingredients are "agreeable to the Body, and . . . penetrating."
35. Yoder D: Folk medicine, in Dorson RM (ed): *Folklore and Folklife*. Chicago, University of Chicago Press, 1972, p 203.
36. Hand WD: Magical Medicine: *The Folkloric Component of Medicine in the Folk Belief, Custom, and Ritual of the Peoples of Europe and America*. Berkeley, University of California Press, 1980.
37. Leventhal H: *In the Shadow of the Enlightenment: Occultism and Renaissance Science in Eighteenth Century America*. New York, New York University Press, 1976, pp 27–32.
38. Bond T, Bond P: *Co-Partnership Ledger*, Philadelphia, vol 2, 1754–1756. Manuscript at College of Physicians of Philadelphia Library Historical Collections, pp 314–315.
39. *Pennsylvania Gazette*, Philadelphia, no. 614, September 18, 1740.
40. Williams WH: *America's First Hospital: The Pennsylvania Hospital, 1751–1841*. Wayne, Pa, Haverford House, 1976, pp 5, 28, 30.
41. Nicolson M, Rousseau GS: Bishop Berkeley and tar-water, in Miller HK, et al (eds): *The Augustan Milieu: Essays Presented to Louis A. Landa*. Oxford, Clarendon Press, 1970, pp 102–137.
42. Austin RB: *Early American Medical Imprints: A Guide to Works Printed in the United States, 1668–1820*. Washington, DC, US Dept of Health, Education, and Welfare, Public Health Service, 1961.
43. Kaser DA: *A Book for a Sixpence: The Circulating Library in America*. Pittsburgh, Beta Phi Mu, 1980, p 10.
44. Wolf E: Medical books in colonial Philadelphia, in Blake JB (ed): *Centenary of Index Medicus 1879–1979*. Bethesda, Md, US Dept of Health and Human Services, Public Health Service, National Institutes of Health, National Library of Medicine, 1980, pp 75, 78–80, 83.
45. *Receipt Book*, Philadelphia?, ca. 1790–early 19th century, 171 [vero 182] pp. Manuscript at College of Physicians of Philadelphia Library Historical Collections.

The impact of smallpox on the native population of the 18th century South

PETER H. WOOD, PHD

Within the present generation public health experts have claimed a final victory over smallpox. The world's last naturally occurring case was recorded in Somalia in October 1977. Supposedly, the genie had been forced back into the bottle; from then on the variola virus would be confined forever to test tubes at epidemic disease laboratories around the world. Ironically, less than a year later the virus escaped from such a research laboratory in Birmingham, England, infecting a worker and her mother, but since then the illness has not reappeared. As a result, no current medical students, and increasingly few doctors, have ever seen a case of smallpox firsthand.[1]

Until its recent confinement, this disease—known in Egypt, the Near East, India, and China for several thousand years—played a substantial role in shaping human history. During the so-called "Age of European Expansion," as contact increased between previously isolated populations around the globe, its dreaded power became particularly great. In the New World, the novel virus infected "virgin" populations that had no prior immunity whatsoever. It spread quickly through the Caribbean and then entered the continental mainland, traveling with the European explorers and settlers who came after Columbus. Indeed, it often moved ahead of foreign colonization, being carried along existing trade routes by persons who sometimes showed no sign of sickness.[2(pp170-171)] For as a standard medical text explains, "Although any person infected with variola virus is potentially contagious, the most dangerous disseminators are persons with unrecognized disease, e.g., the partially immune patient who has relatively few lesions."[3]

During the 16th century, Spanish, French, and English expeditions touched the southeastern shores of North America, and while their numbers were small, the impact of their foreign diseases upon the coastal populations was tremendous. In the 17th century, the number of colonial settlements along the southern coastline increased, and consistent trading relations with neighboring tribes helped smallpox and other contagious foreign diseases move inland. By the 18th century, smallpox was a recurrent and devastating phenomenon, even among the larger and better protected Indian nations of the southeastern interior. By 1700 the decimation of the South by new pathogens had been going on for almost two centuries, and in many areas had already passed its most extreme phase. But the ongoing effects continued to be crucial for all concerned. Since the records of the late colonial period are relatively full and accessible, it is now possible to survey the impact of these illnesses, especially smallpox, on the region's overall Indian population during the 18th century, and to look particularly at the effect of that disease upon the populous and centrally located Cherokee nation.

EARLY SMALLPOX EPIDEMICS IN AMERICA

"We know that the most deadly of the early epidemics in America were those of the eruptive fevers—smallpox, measles, typhus," writes Alfred Crosby in *The Columbian Exchange*. "The first to arrive, and the deadliest, said contemporaries, was smallpox."[4(p42)] The disease reached the island of Hispaniola via Spanish ships in 1518, spread to Puerto Rico by the end of the year, and raged through the native population of Cuba six months later. When Narváez set out from Cuba for the Mexican mainland in order to curtail the designs of Cortez, his soldiers carried with them the smallpox virus that would ravage the Aztec capital of Tenochtitlán and contribute to its sudden downfall.[5(p259)] An Aztec account, complete with graphic illustrations, describes the fearful epidemic which "lasted for seventy days, striking everywhere in the city and killing a vast number of our people. Sores erupted on our faces, our breasts, our bellies; we were covered with agonizing sores from head to foot."

> . . .The illness was so dreadful that no one could walk or move. The sick were so utterly helpless that they could only lie on their beds like corpses, unable to move their limbs or even their heads. They could not lie face down or roll from one side to the other. If they did move their bodies they screamed with pain. A great many died from this plague, and many others died of hunger. They could not get up to search for food, and everyone else was too sick to care for them, so they starved to death in their beds. Some people came down with a milder form of the disease; they suffered less than the others and made a good recovery. But they could not escape entirely. Their looks were ravaged, for wherever a sore broke out, it gouged an ugly pockmark in the skin.[6]

This article is based on a presentation at the symposium entitled, "Interpreting Medical History and Understanding Its Importance in the Study of Early American Life," held April 18, 1986, at Fraunces Tavern Museum, New York, NY.

Address correspondence to Dr Wood, Associate Professor of History, Duke University, 205 East Duke Bldg, Durham, NC 27708.

Copyright © 1987, Sons of the Revolution in the State of New York (Fraunces Tavern Museum).

The disease moved more rapidly than the Spaniards, spreading out across much of South and North America along ancient routes of contact. Smallpox reached the Incas in 1524, so their empire was greatly weakened by the time Pizarro's army arrived eight years later. And this first great post-Columbian pandemic stretched across part of North America as well, apparently reaching Florida and the Gulf Coast before Narváez himself appeared with another flotilla in 1528. Smallpox could have arrived via coastal trading contacts from Mexico or with the natives who fled the scourge in Cuba by canoe and sought refuge with the Calusa Indians of southwest Florida. Indeed, Ponce de León's search for curative waters in Florida, which began in 1513, was aided by desperate Indians seeking relief and protection from new European diseases. "So earnestly did they engage in the pursuit," wrote Escalante later in the century, "that there remained not a river or a brook in all Florida, not even lakes and ponds, in which they did not bathe."[5(pp11-16,254-260,256)]

Whether or not smallpox returned to the region during the 16th century, via coastal slaving raids or the massive entrada of De Soto across the southern interior, the disease was present again in Florida by the mid-17th century, where it killed Spanish colonists, their African slaves, and their Indian neighbors around the little garrison at St Augustine. By the 1670s, the establishment of a rival English port up the coast at Charlestown increased the possibility of new epidemics from abroad. Smallpox contributed to the demise of La Salle's colony on the Texas coast in the 1680s and may have spread inland. In 1691, after several years of contact with French and Spanish intruders, an estimated 3,000 Caddo Indians in East Texas died of an unidentified disease that may have been smallpox.[7] The creation of a Spanish fort at Pensacola and a French outpost at Mobile in 1699 increased similar contacts along the Gulf Coast, with devastating effects. As the 18th century began, imperial rivalries, expanding immigration, increased warfare, and intensified trading activity were all conspiring to spread non-American diseases to inland populations.

"The Small-Pox has been fatal to them," wrote John Lawson, who explored Carolina shortly after 1700; "they do not often escape, when they are seized with that Distemper, which is a contrary Fever to what they ever knew. Most certain it had never visited America, before the Discovery thereof by the Christians." Lawson provided eyewitness testimony of the devastation among coastal and piedmont tribes, reporting that "Their running into the Water, in the Extremity of this Disease, strikes it in, and kills all that use it. Now they are become a little wiser; but formerly it destroyed whole Towns, without leaving one Indian alive in the Village." According to the Englishman, "The Small-Pox and Rum, have made such a Destruction amongst them that, on good Grounds, I do believe, there is not the sixth Savage living within two hundred Miles of all our Settlements, as there were fifty Years ago." Lawson clarified one means of spreading smallpox to the interior tribes when he related, regarding rum, that these same coastal and piedmont Indians "carry it back to the Westward Indians, who never knew what it was, till within very few Years. Now they have it brought them by the Tuskeruros, and other Neighbour Indians, but the Tuskeruros chiefly, who carry it in Rundlets several hundred Miles, amongst other Indians."[8]

In Europe, where smallpox had long been endemic, it was primarily a childhood sickness. While frequently causing death, it also created widespread immunity among survivors, who gladly exchanged the indignity of pockmarked faces for increased life expectancy. In contrast, isolated populations on the periphery of the expanding European economic system continued to be decimated by initial exposure to the smallpox virus. Death rates often soared to one third or more of the entire community. When smallpox suddenly appeared in Iceland in 1707, for example, 18,000 of the island's 50,000 people died in less than two years.[4(p44)] It is not surprising that when Sir William Petty, a pioneer demographer (as well as a physician, economist, colonizer, and noted member of England's Royal Society), wrote to William Penn and others in America in the 1680s seeking detailed information on the Indian population, he asked specifically about the numbers who had smallpox.[9]

In the Near East and Africa, as will be seen, the process of variolation—inoculating a healthy individual with pus from an infected person to induce a (generally) mild case of smallpox and subsequent immunity—was already well known. But Europeans knew virtually nothing of that technique before 1700, and Edward Jenner would not discover his safer vaccination procedure, using cowpox virus, until the 1790s. So at the start of the 18th century, Europeans and Indians were similarly baffled by the variola virus. Each group explained smallpox primarily as an act of supernatural providence, and each society tested its full range of traditional medicine against the disease and desperately sought new cures.

INDIAN REMEDIES

At first most Indian medicine men probably viewed the sickness as a new attack by the animals, for many Native American cultures believed that diseases had their origin in the animal kingdom's effort to retaliate against the aggression of increasingly numerous and well-armed humans. According to Cherokee lore, for example, all the animals, resentful of being hunted down or trodden under foot, came together in a huge council and began "to devise and name so many new diseases, one after another, that had not their invention at last failed them, no one of the human race would have been able to survive. The Grubworm grew constantly more pleased as the name of each disease was called off, until at last they reached the end of the list, when some one proposed to make menstruation sometimes fatal to women." Upon hearing this, the Grubworm "rose up in his place and cried: '*Wadâñ*! [Thanks!] I'm glad some more of them will die, for they are getting so thick that they tread on me.' The thought fairly made him shake with joy, so that he fell over backward and could not get on his feet again, but had to wriggle off on his back, as the Grubworm has done ever since."[2(pp250-252)]

In the Cherokee worldview, a series of polar oppositions had to be kept in harmonious balance: summer and winter, east and west, man and woman, agriculture and hunting.[10] Animals and plants were two important oppositional forces in this scheme, so there was an internal logic, as well as a practical record of success, in combating disease with herbal remedies.[11] Indeed, according to Cherokee legend, medicine came to man from the plants themselves, when they heard the animals naming ingenious new human sicknesses. "Each Tree, Shrub, and Herb, down even to the Grasses and Mosses, agreed to furnish a cure for some one of the diseases named, and each said: 'I shall appear to help Man when he calls upon me in his need.' Thus came medicine," the Cherokees conclude, "and the plants, every one of which has its use if we only knew it, furnish the remedy to counteract the evil wrought by the revengeful animals . . . When the doctor does not know what medicine to use for a sick man the spirit of the plant tells him."[2(p252)]

Native Americans in the South, as elsewhere, rapidly earned the respect of Europeans for their effective practice of herbal medicine. "The Indians," wrote James Adair, "instigated by nature, and quickened by experience, have discovered the peculiar properties of vegetables, as far as needful in their situation of life. For my own part, I would prefer an old Indian before any chirurgeon whatsoever, in curing green wounds by bullets, arrows, &c. both for the certainty, ease, and speediness of cure." Adair, who spent the middle decades of the 18th century among the southern Indians, observed that they "have a great knowledge of specific virtues in simples; applying herbs and plants, on the most dangerous occasions, and seldom, if ever, fail to effect a thorough cure, from the natural bush." Nevertheless, he conceded, they showed "little skill in curing the small pox."[12(p246)]

If so, it was not for want of effort. According to a British surgeon, some Canadian Indians used the pitcher plant (*Sarracenia purpurea*) as a remedy for smallpox "with good results," and southern Indians likewise experimented with possible cures. Whether advised by plant spirits or by neighboring tribes, they undoubtedly administered snakeroot (*Aristolochia serpentaria*) to smallpox victims. In addition, patients were given cassine tea, the renowned "black drink" brewed widely among southeastern Indians from the yaupon bush (*Ilex vomitoria*).[13] As late as the Civil War, yaupon tea was still being recommended as a cure for smallpox in the southern states.[14]

While these Native American cures were not effective against the new disease, still they were picked up and tried by anxious Englishmen in the late 17th century. Thomas Ashe wrote home from Carolina in 1682 regarding snakeroot: "In all Pestilential Distempers, as Plague, Small Pox, and Malignant Fevers, it's a Noble Specific."[15] John Peachie's letter to a friend in London on the "Admirable Virtues" of cassine in curing smallpox was published in London as an eight-page pamphlet, only months after England's Queen Mary died of the disease in December 1694. The Carolina physician reported success in prescribing a few drops of tincture of "cassiny" added to the patient's gruel, beer, or broth. He related stories of "a famous Sea-Chyrurgeon" who had used cassine to cure passengers of smallpox and of a London doctor who had administered drops of cassine imported from Carolina with good results.

VARIOLATION

Peachie was among the first in a long time of southern mountebanks making careless claims for Indian herbal cures and any other theories at hand. He recommended, for example, keeping a live sheep near the smallpox sufferer "to draw away the malignity," so the sheep would die instead of the patient.[16] With advice like this coming from America, apothecaries in Europe soon perceived that the New World was unlikely to prescribe anything for smallpox as useful as the "jesuit bark" that had been provided for malaria sufferers. As their competing empires expanded around the globe, Europeans watched the range and intensity of smallpox increase and began paying attention to treatments from the Near East and Africa.[17] Correspondence to the Royal Society in London from Turkey and Greece in 1714 and 1716 confirmed reports of successful variolation, and as all medical historians know, in 1721 Lady Mary Wortley Montagu, wife of England's ambassador to Turkey, then used the technique on her own child. The next year she convinced Caroline, Princess of Wales, to do the same, giving initial social respectability to the controversial process in England.[18] (England's first smallpox hospital opened in 1746, and by the following decade most English physicians had come to accept variolation.[19]) Since Caroline of Anspach was the wife of George II, and her child, Frederick, was the father of George III, it is interesting to speculate upon the political consequences for England and America of the successful inoculation of the Princess of Wales.

Less well known is the fact that at this same time in America, Africans were relating the mysteries of smallpox variolation to European colonists. In Boston Rev Cotton Mather discovered that a slave purchased for him by members of his church in 1706 understood the practice. In 1716 Mather wrote to a member of the Royal Society that:

> . . . many months before I mett with any Intimations of treating ye Small-Pox with ye Methods of Inoculation, any where in *Europe*, I had from a Servant of my own, an Account of its being practiced in *Africa*. Enquiring of my Negro-Man *Onesimus*, who is a pretty Intelligent Fellow, Whether he ever had ye Small-Pox; he answered *Yes*, and *No*; and then told me that he had undergone an Operation, which had given him something of ye Small-Pox, and would forever preserve him from it, adding, That it was often used among ye *Guaramantese* [Coromantees], and whoever had ye Courage to use it, was forever free from ye Fear of the Contagion. He described ye Operation to me, and showed me in his Arm ye Scar.[20]

In Ethiopia, a traditional treatment for smallpox had long involved pricking several pustules of a sufferer and then inoculating another person by placing some of the infected matter in a scratch made on the arm. If the incision was not too deep, this process generally induced only a mild case of smallpox. Apparently, the useful precaution of drawing variola only from the eruptions of other induced cases was not known, but the general practice still had a lower mortality rate than the disease itself and provided subsequent immunity.[21] So in the face of virulent epidemics this prophylactic measure, in which a priest or doctor used a knife or thorn to infect those who had never been exposed, apparently spread gradually (though the chronology is not yet clear) throughout Muslim Africa and beyond.[22] For example, we know that vaccination moved south to the Manos of present-day Liberia from the Mandingo people further north.[23]

We also know that by the early 18th century this practice was prevalent in the English Gold Coast region near the Bight of Benin, where Coromantee (Akan) captives, like Cotton Mather's Onesimus, were being sold into transatlantic slavery in record numbers. The Coromantees impressed Europeans with their particularly robust health and independent spirit.[24] The local slave ports such as Coromantin "supplied more than half the British exports of 1700–30," according to Philip Curtin, who points out that any English colony "that imported heavily in the early part of the eighteenth century would receive a correspondingly high proportion of Akan peoples from the southern Gold Coast, and of Fon and Gun from southern Dahomey."[25] It is certainly not coincidence that the powerful religious cults centered around smallpox, which still persist in Dahomey (Republic of Benin) and Western Nigeria as well as Cuba and Brazil, made their appearance at precisely this time.[26]

The West African use of inoculation against smallpox and the possible transmission of this knowledge to the New World has gone largely unrecognized during the recent decades of vigorous research into African-American connections. But Eugenia Herbert points out that Mather himself confirmed Onesimus' account of inoculation by speaking with other West African slaves in Boston. In an interesting rendition of 18th century black English, he reported that "a considerable Number of these Africans" seemed to agree on the same story: "That in their Country *grandy-many* dye of the *Small-Pox*; But now they learn This Way: People take Juice of Small-Pox; and Cutty-skin, and Putt in a Drop." The result, they told Mather, was "by'nd by a little Sicky, sicky: then very few little things like *Small-Pox:* and nobody dy of it; and no body have *Small-Pox* any more." With Boston facing a serious outbreak of the disease in 1721, another clergyman, Benjamin Colman, gathered similar stories from local Africans regarding "how to help against the Poison of the Small Pox." When criticized for this approach, Colman asserted that white colonists must "be willing to learn of the poorest Slave in Town."[22]

In contrast, the anonymous author of *A Letter from One in the Country*, aware of the non-European roots of inoculation, urged that the practice be banished to the "Turks and Pagans" whence it came. William Douglass, a leading opponent of inoculation, carried this hostility to nonwhite heathens a step further. In a satire in the *New England Courant* of August 7–14, 1721, Douglass suggested using inoculation as a weapon against the Indians; he offered a bounty of five pounds for each native killed by inoculation and ten pounds for any who survived and spread the disease.[27] This lethal strategy, which Douglass proposed as a cynical jest, would become an acceptable tactic for General Jeffery Amherst and Colonel Henry Bouquet in the British war against Indian allies of the French a generation later.[28]

In colonial Charlestown (modern-day Charleston) and other southern ports, the number of African-born slaves was much larger than in Boston, and this may have contributed to local knowledge of inoculation among blacks and whites. Far from being the unwitting victims of medical experimentation, perhaps some African newcomers accepted or even promoted the practice of variolation during 18th century epidemics. When arriving slave ships precipitated a major smallpox outbreak in Charlestown in 1738, for example, hundreds of Europeans and Africans were inoculated by several white doctors. While more than 2,000 persons (almost half the town's population) caught the disease and over 300 died, the new technique limited the disaster.[29] Conceivably, while venturesome physicians were arousing fear among the town's free citizens by inducing new cases of smallpox, African-born healers with longer experience may have been doing the same thing. This would help explain some of the deep-seated antipathy to the "heathen" practice among the white minority, even in the face of evidence that the risky process worked. After the 1738 outbreak, the South Carolina Assembly passed a bill prohibiting inoculation and imposing a heavy fine on anyone guilty of "communicating" the infection.[30]

Despite such resistance, knowledge of variolation spread through many parts of the Atlantic world during the 18th century and constituted, according to some medical historians, an unprecedented contribution to the control of human mortality. In Africa's Gold Coast region, which sent thousands of blacks to English North America, kings, generals, priests, and slave dealers all would have had a stake in the powerful potential of the method. Early in the century, Cotton Mather heard that captains visiting Guinea sometimes arranged to have captives inoculated before their deportation, "that the poor Creatures may sell for a better price."[22] Inoculation of slave cargoes can be confirmed for later dates, and there is no question that by the middle of the century many blacks selling slaves on the West African coast and most whites purchasing them in America were keenly aware that induced immunity gave those in bondage a higher value and a longer working life.[31]

Consider the caption of a Charlestown slave trading advertisement that appeared in the South Carolina *Gazette* when smallpox was present in the port in the 1760s. The advertisement, from the firm of Austin, Laurens & Appleby, assured readers that the "250 fine healthy NEGROES, just arrived from the Windward & Rice Coast," had not been infected with smallpox since arrival and added, significantly: "N.B. Full one Half of the above Negroes have had the SMALL-POX in their own Country."[32] A subsequent notice in the same newspaper (suggesting a possible convergence of English and African inoculation practices) advertised "230 choice Negroes . . . just arrived from Gambia . . . in perfect health, and have been inoculated for the smallpox on the coast."[33] Whatever their extent, the impact of such measures is hard to assess. While possibly protecting sizable numbers of enslaved Africans from smallpox, this practice also had the effect of keeping the disease in circulation, so it probably did little to prevent appearance of the contagion aboard slave ships. Indeed, such a procedure, when conducted in an unregulated way for immediate gain, may actually have increased the transmission of smallpox via the Middle Passage.

As the smallpox contagion rebounded throughout Europe's Atlantic empires in the 18th century, knowledge of the first effectual preventive measures began to spread as well—whether due to the publication of authoritative medical pamphlets or to the testimony of inoculated West Africans and Europeans. But information about this practice never reached, or perhaps was rejected by, the American Indians of the southern region. To gauge the impact of smallpox on the interior, it is worth examining the experience of the Cherokee nation in southern Appalachia during the first century after the permanent arrival of Europeans and Africans in Carolina. In 1674, four years after the English founded a settlement on the Ashley River, Henry Woodward visited a Westo Indian town on the nearby Savannah River and brought back a report that "Cowatoe and Chorakae Indians" inhabited the headwaters of the Savannah.[34] During the following decade low-country tribes may have been induced to make slaving raids on the Cherokee for captives to sell to the English, but direct contact with Europeans probably did not materialize until the 1690s. "Largely owing to Southern Appalachian physiography," states geographer Gary Goodwin, "the Cherokee Indians were the last of the great southeastern tribes to enter the mainstream of the southern Indian trade."[35]

SMALLPOX AMONG THE CHEROKEES

In 1693, 20 Cherokee leaders visited Governor Thomas Smith in Charlestown to seek aid against the encroachments of the Coosa and Catawba tribes from the piedmont, and the elders lodged protests that Cherokee people were being kidnapped by slave hunters. By this time traders from Charlestown and from the Occaneechi Post on the Indian trail leading southwest from Virginia had already begun to initiate barter with the Cherokees for furs.[35(pp86-87)] Such tenuous contacts, as well as the increasing presence of Indian middlemen, were undoubtedly responsible for the major smallpox epidemic that swept through the Creek and Cherokee villages "four or five hundred miles inland" during the winter and spring of 1697.[36] As the Cherokees became more dependent upon the Carolina trade in the early 18th century, their contacts with the coast increased, and the onslaughts of smallpox recurred at predictable intervals. By 1730 the Cherokee population had been reduced to about 15,000, literally cut in half in two generations, and the disease would soon return to the southern mountains.[37]

"About the year 1738," writes James Adair, "the Cheeroke received a most depopulating shock, by the small pox . . .: it was conveyed into Charlestown by the Guinea-men, and soon after among them by the infected goods." Adair goes on to recount at length the responses of the Cherokee medicine men (perhaps already somewhat influenced by their Christian rivals near the coast):

> The old magi and religious physicians who were consulted on so alarming a crisis, reported the sickness had been sent among them, on account of the adulterous intercourses of their young married people, who the past year, had in a most notorious manner, violated their ancient laws of marriage in every thicket, and broke down and polluted many of the honest neighbours bean-plots, by their heinous crimes, which would cost a great deal of trouble to purify again. To those flagitious crimes they ascribed the present disease, as a necessary effect of the divine anger; and indeed the religious men chanced to suffer the most in their small fields, as being contiguous to the town-house, where they usually met at night to dance, when their corn was out of the stalks; upon this pique, they shewed their priest-craft. However, it was thought needful on this occasion, to endeavour to put a stop to the progress of such a dangerous disease: and as it was believed to be brought on them by their unlawful copulation in the night dews, it was thought most practicable to try to effect the cure, under the same cool element. Immediately, they ordered the reputed sinners to lie out of doors, day and night, with their breast frequently open to the night dews, to cool the fever: they were likewise afraid, that the diseased would otherwise pollute the house, and by that means, procure all their deaths. Instead of applying warm remedies, they at last in every visit poured cold water on their naked breasts, sung their religious mystical song, *Yo Yo*, &c. with a doleful tune, and shaked a callabash with the pebble-stones over the sick, using a great many frantic gestures, by way of incantation....
>
> When they found their theological regimen had not the desired effect, but that the infection gained upon them, they held a second consultation, and deemed it the best method to sweat their patients, and plunge them into the river,—which was accordingly done. Their rivers being very cold in summer, by reason of the numberless springs, which pour from the hills and mountains—and the pores of their bodies being open to receive the cold, it rushing in through the whole frame, they immediately expired: upon which, all the magi and prophetic tribe broke their old consecrated physic-pots, and threw away all the other pretended holy things they had for physical use, imagining they had lost their divine power by being polluted; and shared the common fate of their country.[12(pp244-245)]

According to Adair, many Indians who were not killed by the disease itself or by harsh treatments chose to take their own lives rather than suffer the shame which would accompany disfigurement: "some shot themselves, others cut their throats, some stabbed themselves with knives, and others with sharp-pointed canes; many threw themselves with sullen madness into the fire, and there slowly expired, as if they had been utterly divested of the native power of feeling pain." With only slight exaggeration, Adair concluded that the epidemic "reduced them almost one half, in about a year's time."[12(pp244-245)] At the very least, it seems likely that several thousand persons, from among a total population of fewer than 15,000, died in 1739. More may have been lost the following year, when Cherokee warriors, recruited by Georgia's Governor Oglethorpe to join the attack on Spanish Florida, supposedly brought back smallpox from the English camps around St Augustine.[38] By 1755, when North Carolina's Governor Dobbs submitted a careful census of "The Warriors of the Cherokee Nation" conforming roughly to traditional divisions (Overhill Towns, Middle Towns, Valley Towns, and Lower Towns), he put the total number of fighting men at 2,590.[39] Thus by the mid-18th century, the Cherokee nation's total population was scarcely 10,000 persons.

This number was soon diminished once again by another invasion of smallpox, which began spreading out among the southeastern tribes in the fall of 1759, following Cherokee attacks on Carolina settlements in late summer which signaled the start of a new Indian war. On December 15, the South Carolina *Gazette* stated:

> It is pretty certain that the Smallpox has lately raged with great Violence among the Catawba Indians, and that it has carried off near one-half of that nation, by *throwing themselves into the River*, as soon as they found themselves ill—This Distemper

> has since appeared amongst the Inhabitants of the *Charraws* and *Waterees*, where Many Families are down; so that unless special care is taken, it must soon spread thro' the whole country.

In early January (a week before the epidemic reached Charlestown via soldiers and captives returning from the frontier), Governor William Lyttleton received a message from Richard Colymer at the Indian trading post of Fort Prince George saying that many Indians at the nearby Cherokee town of Keowee had died of the disease and that, "The living are all fled to the woods to avoid it. It is now got up to Yarasee and Yamasee." Colymer continued, "and I can't help being so inhuman as to wish it spread through the whole nation." Throughout the spring the sickness raged in the mountains and also along the coast where, despite prohibitions against inoculation, wealthy merchants and planters clamored to protect their families and slaves using the new procedures. Once they obtained permission to inoculate their family members and slaves, others protested to the Assembly that the widespread inoculation of Negroes would prolong the epidemic, so that "the disorder will continue through the summer months."[40]

By mid-1760, after more than 600 deaths, the sickness was subsiding in Charlestown, but it continued to spread in the mountains. An informant wrote from Augusta on August 13: "We learn from the Cherokee country, that the People of the Lower Towns have carried smallpox into the Middle Settlement and Valley, where that disease rages with great Violence, and that the Upper Towns are in such dread of the Infection, that they will not allow a single Person from the above named Places to come amongst them."[41] According to Adair, the Cherokees' southern neighbors, the Creeks, had learned from traders to prevent smallpox "from spreading among their towns, by cutting off all communication with those who are infected, till the danger is over."[12(p276)] But by October newspapers noted briefly that the "Cherokees had brought the Small-Pox into the Upper Creek Nation."[42]

In 1768 a New England minister passed along information on the Cherokees from a Reverend Richardson of South Carolina, "that when he was amongst them as a missionary, ten years ago, they had about 1500 fighting men; but since then they have been greatly diminished by the wars with other nations, with the English, and by the small-pox, which is a most fatal disease among them."[43] In the spring of 1775 the Quaker naturalist William Bartram passed through several of the recently devastated Cherokee villages. No 18th century observer of the South was more aware of the destruction that war and disease had brought to the once-populous mountain nation. In his impressive *Travels*, published in 1791, Bartram recalled visiting the previously thriving town of Keowee opposite Fort Prince George (in present-day Oconee County, SC), where a congress of Lower Cherokee chiefs was about to be held:

> . . . this fertile vale, within the remembrance of some old traders with whom I conversed, was one continued settlement, the swelling sides of adjoining hills were then covered with habitations, and the rich level grounds beneath lying on the river, were cultivated and planted, which now exhibit a very different spectacle, humiliating indeed to the present generation, the posterity and feeble remains of the once potent and renowned Cherokees: the vestiges of the ancient Indian dwellings are yet visible on the feet of the hills bordering and fronting on the vale, such as posts or pillars of their habitations, &c.[44]

Continuing its 20-year pattern, smallpox touched the Cherokees again during the bitter years of the American Revolution. Shortly before Bartram visited Keowee in 1775, a Cherokee warrior named Dragging Canoe had broken publicly with tribal elders and walked out of a conference with Judge Richard Henderson and his business associates at Sycamore Shoals. Protesting Henderson's "purchase" of valuable Cherokee land, Dragging Canoe reputedly offered the memorable prediction, "You will find its settlement dark and bloody." He refused to treat further with the foreign invaders of his homeland and withdrew westward to the strategic heights of Chickamauga on the Tennessee River where his so-called Chickamauga Cherokees offered fierce resistance to white interlopers for nearly two decades.[45]

In the spring of 1780, Dragging Canoe's forces ambushed John Donelson's flotilla of 30 boats and 200 people on the Cumberland River as it passed the new Chickamauga towns on the way west to Nashborough, the settlement promoted by Colonel Henderson. The Indians picked off the final boat, killing or capturing all 28 passengers. Only later did they discover why this boat had isolated itself behind the rest: Some of the occupants were sick with smallpox. The disease quickly spread through the remote Indian settlement and, according to one authority, "caused the death of several hundred Indians."[46] In a single century, the Cherokee population had been decimated. A nation which had included roughly 30,000 people in 1690 numbered 8,000—scarcely one quarter as many—a century later. Thomas Jefferson's summary of the experience of the coastal Indians applies on a larger scale to the interior nations as well. In his *Notes on the State of Virginia*, Jefferson observed that, "Spiritous liquors, the small-pox, war and an abridgement of territory to a people who lived principally on the spontaneous productions of nature, had commmited terrible havoc among them . . . ."[47]

THE DEMOGRAPHIC CONSEQUENCES

The "terrible havoc" to which Bartram and Jefferson testified in their different ways 200 years ago can now be confirmed more explicitly in demographic terms. In my own current research on the southern population in the century before the revolution, broken down by decade, subregion, and race, I am finding some startling preliminary statistics that help put the preceding discussion into context. I have divided up the South into ten logical smaller areas and compiled data on Indians, Europeans, and Africans living in each area over time, using everything from census records and traders' accounts to governmental reports and strategic military estimates. Naturally, the statistics vary in their accessibility and accuracy. Still, it has been possible to pull together a rough but remarkably complete overall picture.[37]

Though Virginia is often taken as being "typical" of the colonial South, its expansive 18th century population of Europeans and Africans—some 465,000 by 1775—makes it the demographic exception in an otherwise sparsely settled domain. So the Old Dominion can be set aside in the following estimations. When one does so, and then looks at the entire South below Virginia—from Carolina to East Texas—during the 1680s, there appear to have been slightly more than 200,000 inhabitants, and fully 95 of every 100 persons were Native Americans. The major interior tribes—Creek and Cherokee, Choctaw and Chickasaw, Natchez and Shawnee—were so significant at that time, and their numerical demise was so sharp, that the overall population of the South beyond Virginia fell by almost one third to scarcely 140,000 in 1730, despite a steady influx of arrivals from Europe and Africa. Contrary to popular imagery, the South in the early 18th century was a region of demographic decline. The Indian population, examined alone, had dropped catastrophically, from 197,000 to fewer than 68,000 in less than 50 years.

In the next 50 years, the rate of Indian decline would be less great—the net loss of another 12,000 or 15,000 persons. But the growth of the white and black populations on the South's perimeter, through steady immigration and rapid natural increase, would be enormous—nearly doubling every decade for more than a generation. By the eve of the American Revolution the total population of the non-Virginia South, stretching from the Sea Islands to the border of the Great Plains in eastern Texas, had shot upward to more than 500,000 persons, but now among every 100 inhabitants scarcely ten were Native Americans, nearly 40 were Afro-American, and more than 50 were of European origin. The ethnic and cultural makeup of the South had been transformed forever, and no single factor had been more significant than the smallpox virus.

REFERENCES

1. Hopkins DR: *Princes and Peasants: Smallpox in History.* Chicago, University of Chicago Press, 1983, p 310.
2. See Mooney J: *Myths of the Cherokee.* Washington, Bureau of American Ethnology, 1900.
3. Davis BD, Dulbecco R, Eisen HN, et al: *Microbiology*, ed 2. New York, Harper & Row Publishers Inc, 1973, p 1270.
4. Crosby AW Jr: *The Columbian Exchange: Biological and Cultural Consequences of 1492.* Westport, Conn, Greenwood Press, 1972.
5. Dobyns HF: *Their Number Become Thinned: Native Population Dynamics in Eastern North America.* Knoxville, University of Tennessee Press, 1983.
6. Leon-Portilla M (ed): *The Broken Spears: The Aztec Account of the Conquest of Mexico.* Boston, Beacon Press Inc, 1962, pp 92–93.
7. Ewers JC: Influence of epidemics on the Indian populations and cultures of Texas. *Plains Anth* 1973; 18:104–115.
8. Lawson J: *A New Voyage to Carolina*, 1709, reprint ed. Chapel Hill, University of North Carolina Press, 1967, pp 231–232.
9. Cassedy JH: *Demography in Early America: Beginnings of the Statistical Mind, 1600–1800.* Cambridge, Harvard University Press, 1969, p 56.
10. Hudson C: Cherokee concept of natural balance. *Indian Hist* 1970; 3:51–54.
11. See Martin C: *Keepers of the Game: Indian-Animal Relationships and the Fur Trade*, Berkeley, University of California Press, 1978; and Krech S III (ed): *Indians, Animals, and the Fur Trade: A Critique of "Keepers of the Game,"* Athens, University of Georgia Press, 1981.
12. Adair J: *Adair's History of the American Indians*, 1775, reprint ed. Ann Arbor, University of Michigan Press, 1966.
13. Vogel VJ: *American Indian Medicine.* Norman, University of Oklahoma Press, 1970, p 89.
14. Porcher FP: *Resources of the Southern Fields and Forests,... Being a Medical Botany of the Confederate States....* Charleston, 1863, pp 393–394. Also see Wood PH: People's medicine in the early South. *Southern Exposure* 1978; 6:50–53.
15. Ashe T: Carolina, or a description of the present state of that country (London, 1682), in Salley AS Jr (ed): *Narratives of Early Carolina, 1650–1708.* New York, Charles Scribner's Sons, 1911, p 145.
16. Peachie J: *Some Observations Made upon the Herb Cassiny; Imported from Carolina: Shewing its Admirable Virtues in Curing the Small Pox....* 1695, cited by WC Sturtevant in Hudson CM (ed): *Black Drink, A Native American Tea.* Athens, University of Georgia Press, 1979, pp 151–152.
17. See McNeill WH: *Plagues and Peoples.* Garden City, NY, Doubleday & Co Inc, 1976, pp 249–258.
18. Turberville AS: *English Men and Manners in the Eighteenth Century*, ed 2. Oxford, England, Clarendon Press, 1929, pp 110–113.
19. See Miller G: *Adoption of Inoculation for Smallpox in England and France*, Philadelphia, University of Pennsylvania Press, 1957; and her article, Smallpox inoculation in England and America: A reappraisal. *William and Mary Quarterly*, 3rd series, 1956; 13:476–492.
20. "America's share in the successful conquest of smallpox began at precisely this minute," writes O. E. Winslow in *A Destroying Angel: The Conquest of Smallpox in Colonial Boston.* Boston, Houghton-Mifflin Co, 1974, p 32. On p 33 she quotes (in this slightly abridged form) the letter of C. Mather to J. Woodward, Boston, December 16, 1716. (The original "Onesimus" was a runaway slave mentioned by Saint Paul.)
21. Pankhurst R: History and traditional treatment of smallpox in Ethiopia. *Med Hist* 1965; 9:343–355.
22. Herbert EW: Smallpox inoculation in Africa. *J Afr Hist* 1974; 16:539–559.
23. Harley GW: *Native African Medicine, With Special Reference to its Practice in the Mano Tribe of Liberia*, 1941, reprint ed. London, Frank Cass, 1970, p 45.
24. Pope-Hennessy J: *Sins of the Fathers: A Study of the Atlantic Slave Traders, 1441–1807.* New York, Alfred A. Knopf Inc, 1967, pp 58–59.
25. Curtin PD: *Atlantic Slave Trade: A Census.* Madison, University of Wisconsin Press, Madison 1969, pp 154–155.
26. Verger P: *Dieux d'Afrique: Culte des Orishas et Voduns.* Paris, Memoires de L'Institut Francais d'Afrique Noire, 1954, p 240. (I am grateful to David Brown of Yale, an expert on African smallpox deities, for this reference.) On the Yoruba cult of Obaluaiye, the earth deity able to conjure or cure smallpox and honored with imagery of spears, thorns, and cactus plants, see Thompson RF: *Flash of the Spirit: African and Afro-American Art and Philosophy.* New York, Random House Inc, 1983, pp 61–68.
27. *A Letter from One in the Country, to his Friend in the City: In Relation to their Distresses occasioned by the doubtful and prevailing Practice on the Innoculation of the Small-Pox.* Boston, 1721. A physician from France, who had knowledge of several disastrous efforts by the French army to use inoculation as early as 1696, influenced Boston's smallpox debate. A translation of Dr Lawrence Dalhonde's testimony (by W. Douglass and J. Marion) appears as an appendix to the second, corrected edition of Boylston Z: *An Historical Account of the Small-pox Innoculation in New England.* Boston, Gerrish and Hancock, 1730, pp 51–52. Sarah Blank Dine kindly provided these references.
28. Duffy J: Smallpox and the Indians in the American colonies. *Bull Hist Med* 1951; 25:340.
29. Duffy J: *Epidemics in Colonial America.* Baton Rouge, Louisiana State University Press, 1953, pp 32–35, 50–52.
30. An Act for the Better Preventing of Spreading of the Infection of the Small Pox in Charles Town, in Cooper T, McCord D (eds): *Statutes at Large in South Carolina*, vol 3. Columbia, SC, AS Johnson, 1836, p 513.
31. Stewart L: Edge of utility: Slaves and smallpox in the early eighteenth century. *Med Hist* 1985; 29:54–70.
32. Low WA, Clift VA (eds): *Encyclopedia of Black America.* New York, McGraw Hill Book Co, 1981, p 15.
33. South Carolina *Gazette*, December 1, 1772.
34. Swanton JR: *Indians of the Southeastern United States.* Washington, Bureau of American Ethnology, 1946, p 111.
35. Goodwin GC: *Cherokees in Transition: A Study of Changing Culture and Environment Prior to 1775.* Chicago, University of Chicago Department of Geography, 1977, p 87.
36. Crane VW: *Southern Frontier, 1670–1732*, 1928, reprint ed. New York, WW Norton & Co Inc, 1981, p 142.
37. See my essay on southeastern Indian population in Wood PH, Waselkopf G, Hatley MT (eds): *Powhatan's Mantle.* Lincoln, University of Nebraska Press (forthcoming).
38. Corkran DH: *Cherokee Frontier: Conflict and Survival. 1740–1762.* Norman, University of Oklahoma Press, 1962, p 17.
39. Letter of January 4, 1755, in Saunders WL (ed): *Colonial Records of North Carolina.* Raleigh, State of North Carolina, 1887, vol 5, pp 320–321.
40. R. Colymer to Gov Lyttleton, January 7, 1760 (photocopy, Waring Library, Charleston): *Journal of the South Carolina Commons House of Assembly*, April 18, 1760 (South Carolina Department of Archives and History, Columbia). I am indebted to Suzanne Krebsbach for these references.
41. Pennsylvania *Gazette*, September 4, 1760.
42. Pennsylvania *Gazette*, October 30, 1760.
43. Letter from Rev Elam Potter to Rev Dr Stiles, in *Mass Hist Soc Coll*, 1st series, vol 10, p 120. (Richardson seems to have underestimated the 1758 population somewhat.)
44. Bartram W: *Travels through North & South Carolina, Georgia, East & West Florida, the Cherokee Country....* Philadelphia, James & Johnson, 1791, p 332.
45. Pate JP: The Chickamauga: A forgotten segment of Indian resistance on the southern frontier, PhD thesis. Mississippi State University, 1969, pp 53ff.
46. Brown JP: *Old Frontiers: The Story of the Cherokee Indians from Earliest Times to the Removal to the West, 1838.* Kingsport, Tenn, Southern, 1938, p 182, n 9. Cf John Donelson's journal, in Williams SC (ed): *Early Travels in the Tennessee Country, 1540–1800.* Johnson City, Tenn, Watauga, 1928, p 236.
47. Jefferson T: *Notes on the State of Virginia*, 1785, reprint ed. New York, Harper & Row Publishers Inc, 1964, p 91.

Patterns of drug usage in colonial America

J. WORTH ESTES, MD

One of the more perceptive students of American medicine noted that historians have tended to avoid trying to analyze drug usage by earlier generations, partly because it has seemed to be so complex, and partly because many historical remedies could not have been truly effective by the criteria conventionally applied to modern drugs.[1] Probably the most important step in interpreting medicines of the past is recognizing that each of them can be seen as fitting into any of several different usage patterns. Thus, the clearest explanation of a given remedy is one that unambiguously inteprets it within only one pattern at a time, relying only on contemporary definitions of the drug's mechanism of action. Three major patterns, or levels, of drug use provide the most practical contexts in which colonial American medical practices can be explained. The slightly annotated bibliography will provide further guidance in selecting a usage pattern that will be particularly appropriate to each interpretive need.

ADJUSTING HUMORS AND TONES

The first patterns, or levels, to be considered are the most basic of all. They are implicit in the underlying concepts of the pathophysiology of disease that were prevalent in the 17th and 18th centuries, and which persisted for another century or more, to some extent at least, although they were being modified continually.

Until bacteria were discovered (by the late 19th century) to be responsible for a substantial majority of the illnesses of Americans, physicians had to rely on notions of disease and its treatment that were totally unlike the concepts of specific diseases, each with its own specific cause and specific treatment, that today we take for granted. Colonial doctors hypothesized that some diseases were inherited, that others were associated with trauma or meteorological phenomena, and that most were contagious, although they did argue about just how some contagions were transmitted. In general, the doctors focused their therapeutic attentions on each patient's symptoms, regardless of what might have induced the appearance of those symptoms in the first place.

The earliest colonists interpreted symptoms as evidence of imbalance among the body's four humors, the four fluids they identified as blood, phlegm, yellow bile, and black bile. This pattern, a relic of ancient Greek thinking, provided so many satisfactory explanations that it was never tested, nor was it abandoned completely until long after the discovery of bacteria had provided more practical explanations of disease patterns. One factor that made the humoral theory so readily acceptable was the multiple correlation of the four humors with the four seasons of the year, with the four ancient elements—fire, water, air, and earth—and with the extremes of temperament implicit in the adjectives sanguine, phlegmatic, bilious (or choleric), and, for black bile, melancholic.

Imbalances in the humors were detected at the bedside as excesses, or as deficits, in four corresponding characteristics: heat and cold, moisture and dryness (Fig 1). For instance, an excess of heat usually implied a fever. And the absence of increased body temperature, even when the patient had other signs ordinarily associated with fevers, led to the diagnosis we still call a "cold." Today, when we say someone is "in a good humor" or "in a foul humor," we are only continuing an ancient medical tradition.

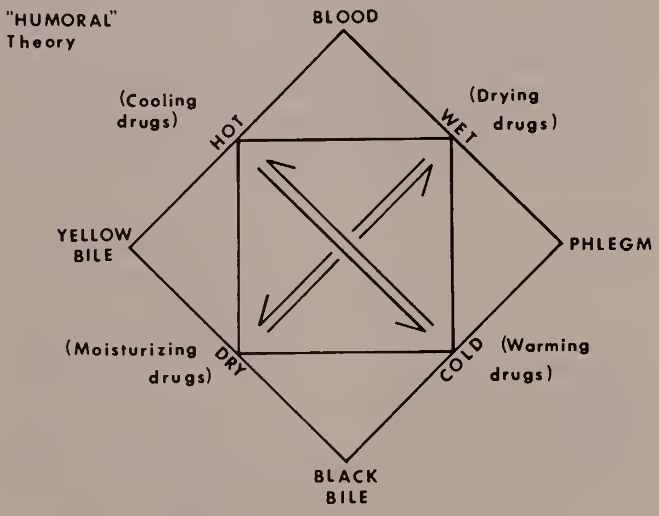


FIGURE 1. Schematic diagram illustrating the correspondences among the four humors and their clinical manifestations, as well as the application of drugs to the rectification of humoral imbalances (abstracted from several medieval diagrams).

This article is based on a presentation at the symposium entitled, "Interpreting Medical History and Understanding Its Importance in the Study of Early American Life," held April 18, 1986, at Fraunces Tavern Museum, New York, NY.

Address correspondence to Dr Estes, Professor of Pharmacology (History of Pharmacology), Boston University School of Medicine, 80 E Concord St, Boston, MA 02118.

Copyright © 1987, Sons of the Revolution in the State of New York (Fraunces Tavern Museum).

During all the centuries when symptoms were explainable chiefly in terms of unbalanced humors, it was easy to conclude that appropriate medical therapy would consist of treatments that were directed toward restoring the normal four-square balances among the humors. This meant, for instance, that fevers should be treated with what were considered to be "cold" remedies, in order to neutralize the patient's excessive heat, just as abnormal accumulations of fluid were to be treated with "drying" remedies, drugs which are still called diuretics because they enhance the removal of excess fluid from the body via the urine.

By the mid-18th century, a second way of interpreting symptoms had spread throughout the English-speaking world, chiefly through the writings of Friedrich Hoffmann (1660–1742) in Germany. Emphasizing the importance of anatomy and physics for interpreting medical phenomena, he concluded that the chief principle of life was motion. Because the heart and large blood vessels could actually be seen or felt to contract in living organisms, Hoffmann associated health with optimum strength and tissue irritability, the property that today we call excitability, in all the body's fibers, just as the humoral theory postulated that health was associated with balances among the body's fluids.[2]

Colonial physicians, therefore, focused on the tone—the innate strength and elasticity—of the solid portions of the blood vessels and nerves (Fig 2), as well as on the humors. Even nerves were considered to be tubes, as arteries clearly were, and both were thought to propel their respective contents through the body with forces that were proportional to the strength of the fibers in the walls of those tubes. We still exploit these concepts when we describe a person as "tense" or as "relaxed."

Thus, the body was healthy when the blood or the hypothetical nerve fluids could circulate freely, or when feces, urine, or sweat could be expelled freely, and so on. Besides, it was well known that tying off a blood vessel—that is, constricting it artificially—was like constipation—that is, prolonged constriction of the intestines—in that both kinds of narrowing would produce localized stagnation of the contents of both kinds of tubes, eventually leading to putrefaction and death in each.

Altered humors could be detected by obvious clues, like skin temperature, or by abnormal production of body fluids, such as coughing up phlegm, increased or decreased urine output, or by facial flushing; a red face indicated to the doctor that his patient had too much blood rushing around in his body. The pulse was regarded chiefly as an index of the activity of the fibers of the blood vessels and as the most convenient measure of body temperature (Fig 3)—it was seldom used as an index of heart action alone. For instance, if the pulse was slow or weak, the vascular fibers were inferred to be weakened, or lax. If, on the other hand, the pulse was fast, the physician could conclude that the vascular fibers were hyperactive, and that fever was present, because heat always accompanied hyperactivity. By the late 18th century the oxygenating function of the lungs had been demonstrated, and the analogy between oxygenation and combustion had been drawn. As a result, the respiratory rate was used as another clue to the presence of fever (Fig 4).

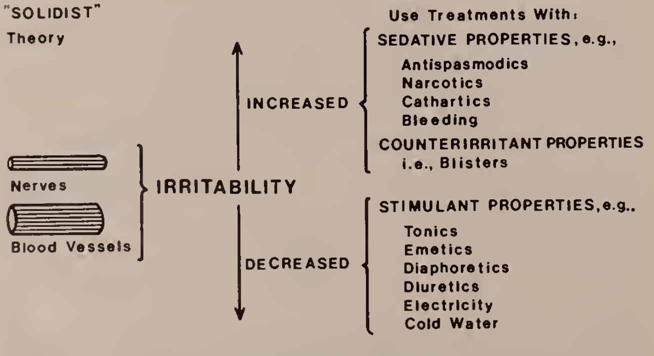


FIGURE 2. Outline of the principles underlying the application of treatments to imbalances in the irritability, or excitability, of solid tissues in the late 18th century.

Once the doctor had determined whether his patient's symptoms were associated with hypoactivity or with hy-

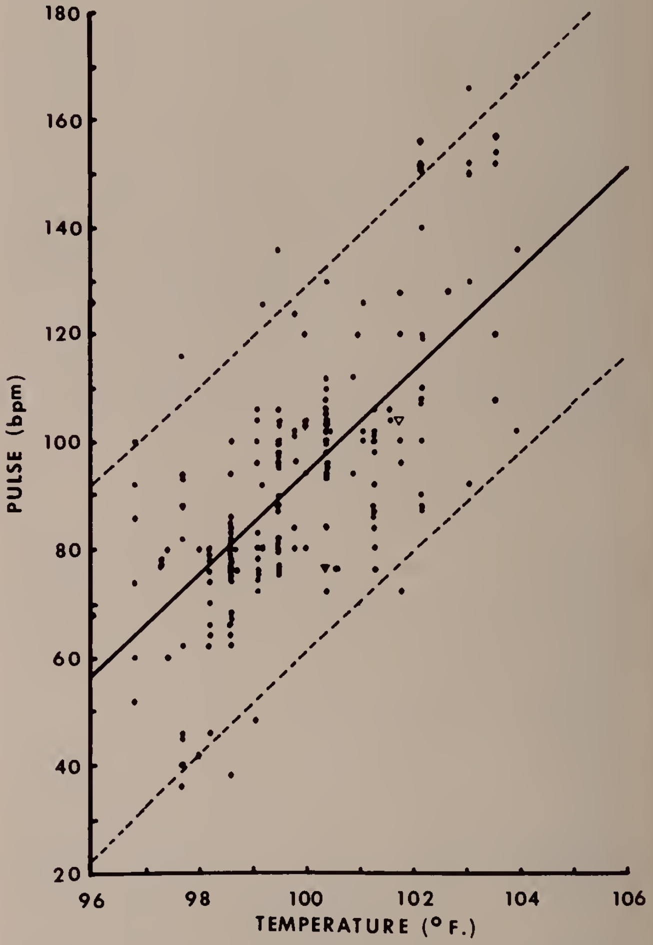


FIGURE 3. Computed linear regression line of best fit (Y = 9.34X − 840), with 95% confidence limits, relating pulse rate to body temperature measured thermometrically by Dr Alfred Donné in the early 1830s. He observed the normal temperature range to be 96.8–98.4°F. The slope of the line is consistent with the common 18th century assumption that the pulse rate rose 10 bpm for each degree rise in temperature. The open triangle represents the pulse of a fever patient before he was bled, and the closed triangle represents his pulse afterward; the 26-bpm reduction in his pulse, which corresponded with a 1.4°F reduction in temperature, illustrates the therapeutic benefit expected from phlebotomy. Data from: Donné A: Recherches sur l'état du pouls, de la respiration et de la température du corps dans les maladies, et sur les rapports qui existent entre ces divers phénomènes. *Archiv Génér Méd* 1835; 9(2d ser):129–157.

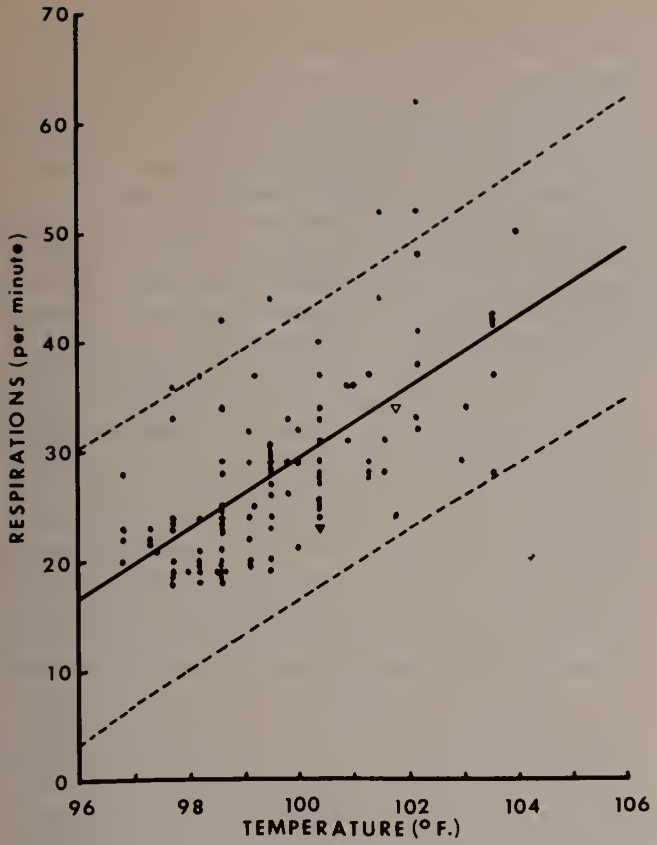


FIGURE 4. Computed linear regression line of best fit (Y = 3.15X − 285), with 95% confidence limits, relating respiratory rate to thermometrically measured body temperature. The triangles represent the patient described in the legend for Fig 3, and lead to the same conclusion. Other calculations show that the respiratory rate was a statistically more precise index of temperature than pulse rate, although there is no evidence that 18th century physicians were aware of the difference. For data source, see Fig 3.

peractivity of the blood vessels or nerves, he could then decide how best to treat the illness, with drugs he regarded as stimulants for patients with weak systems, or with drugs he called sedatives for overactive systems (see Fig 2). For instance, strong stimulants, including cold water and sometimes even applications of static electricity,[3] were prescribed for patients who were paralyzed, because their nerves were too weak. Conversely, sedatives like opium were given to patients with convulsions. This therapeutic approach also meant that both fever and convulsions, as examples of conditions associated with hyperexcitability—although originating in organs as different as blood vessels and nerves—might sometimes be treated with the same sedative drug. The humoral and solidist theories were not mutually exclusive or even competitive; that is, most illnesses could be interpreted in terms of both simultaneously.

How, then, could an 18th century physician know when he had adjusted the balances among the patient's humors or solid tissue tones successfully? This question brings us to a second kind of pattern in colonial medical practices, one that emerges when we examine the frequencies with which doctors prescribed drugs of different therapeutic classes.

The clinical notes of Dr Peter St Medard (1756–1822) of Boston, on a 16-month naval cruise that began in 1802, provide valuable clues to everyday drug use in the late 18th century.[4–6] Because the modern medical record was invented only in the 20th century, until recently we have had to rely chiefly on the books and letters of a relatively few early physicians for information about their routine practices—and even then it is not always reasonable to assume that the majority of physicians routinely did what they said they did, much less that they regularly followed the recommendations of others. Detailed analyses of the records of ordinary, nonacademic physicians like Dr St Medard can often provide more reliable clues to prevailing medical practices than can books written by the professors. Textbooks, herbals, and pharmacopeias are only modestly helpful for accurately interpreting the uses of historical remedies.

St Medard and his contemporaries evaluated their attempts to adjust internal balances not only in terms of recovery from illness, but, more immediately, by assessing the effects of drugs that produced clear-cut changes in the body's natural products and excretions, such as increased or decreased amounts of urine, feces, sweat, blood, tissue fluids, and so on[1] (Table I). Dr St Medard relied on cathartics, or strong laxatives, to flush out unbalanced hu-

TABLE I. Treatments Prescribed Most Often by Dr Peter St Medard on a 16-Month Naval Cruise in 1802–1803*

- **Cathartics** (19.3% of all treatments)
  - Rhei (medicinal rhubarb)
  - Calomel (mercurous chloride)
  - Ricini (castor oil)
  - Jalap
  - Elixir Proprietatis (tincture of aloes and myrrh)
  - Nitre (nitrous acid salts and mixtures)
  - Glauber's salt (sodium sulfate)
  - Balsam of Capivi (extract of *Copaifera officinalis*, from South America)
  - Glycerrhiza (licorice)
  - Senna
  - Cremor tartar (sodium potassium tartrate)
  - Aloes
  - Enemas (usually soapy)
- **Tonics** (26.7% of all treatments)
  - Peruvian bark, or Cortex Peruviana
  - Huxham's tincture (mixture of Peruvian bark, Virginia snakeroot, and flavorings)
- **Emetics** (5.6% of all treatments)
  - Tartar emetic (antimony potassium tartrate)
  - Ipecac
- **Diaphoretics** (4.8% of all treatments)
  - Antimony salts in wine
- **Absorbents** (5.5% of all treatments)
  - Probably magnesium carbonate and/or kaolin
- **Narcotics** (2.3% of all treatments)
  - Opium pills
  - Laudanum, Paregoric, and Thebaic tincture (nearly identical alcohol solutions of opium)
- **Epispastics or Blisters** (0.9% of all treatments, prescribed for 11.0% of all patients)
  - Tincture of cantharides (powdered *Lytta vesicatoria* beetle)
- **Bleeding** (1.4% of all treatments, prescribed for 17.0% of all patients)

* Individual drug preparations are listed in decreasing order of use within each class. Abstracted from work cited in reference 5.

mors by way of the intestines. At the same time, he thought such drugs would relax the abnormal tensions that had constricted his patients' intestinal fibers and inhibited normal evacuations. However, constipation alone was seldom an indication for treatment with cathartics in the 18th century. Indeed, they might even be prescribed for a patient with diarrhea, if his physician determined that the underlying problem was associated with hyperactive intestinal fiber tones that could be reduced, or sedated, with drugs that also produced catharsis.

St Medard could choose from among a variety of laxative drugs. He based each choice on the patient's weight, body build, skin color, and so on, even on the patient's mood and temperament, as well as on the actual symptoms. This was very personalized and highly tailored therapy, which made it all the more complex. All the cathartics St Medard used are effective evacuants; all of them can, and do, increase stool production. However, catharsis all by itself really does not contribute very much to restoring any patient's health.

The naval surgeon had many other methods for adjusting humors and tones. He prescribed Peruvian bark, which today we know contains quinine, as specific treatment for the "intermittent fevers," which we now call the malarias. However, since the bark was so effective a cure for the intermittent fevers, 18th century doctors reasoned it should be effective in many other fevers as well. The bitter taste and astringent property of the bark also contributed to its reputation as an all-purpose tonic that strengthened the body—that is, gave it new tone—when it had been weakened by fever. Consequently, St Medard and his contemporaries usually administered it only after the first, most severe, stage of a continued (or nonintermittent) fever had passed, when the patient was weakest.

St Medard used emetics, drugs that caused vomiting, to strengthen what he took to be weak stomach muscle fibers. He thought that a medicine that increased the tone of those fibers would transmit new strength to other organs that had been weakened by disease, via the nerves that connected the stomach with the rest of the body. The act of vomiting was seen as evidence that such a drug had acted on the stomach as it was intended to do, and that it would then augment the strength of other organs—the primary therapeutic goal of emetic drugs.

St Medard gave diaphoretics to make his patients sweat out their diseases and, at the same time, to remove excess fluid. He reasoned that these medicines, especially those made with antimony, produced their effects by strengthening the blood vessels that carried fluid to the sweating apparatus. A similar rationale led him to use drugs he called absorbents, because they were thought to absorb noxious disease-producing materials out of the blood and into the intestines, so that those materials could be flushed out with the stools.

St Medard reserved two forms of treatment for his most seriously ill patients, usually those who had the worst fevers or injuries. One was the drug class called epispastics, or blisters. For this purpose he applied an alcohol solution of pulverized cantharides beetles. When placed on the skin, cantharides does raise a large, painful blister. Although it was first used in ancient humoral therapy to facilitate the removal of noxious materials within the body into the blister fluid, cantharides continued in use even after solidist theories came to the fore. When that time came, physicians could reason that the artificially induced inflammation produced by the blistering process was able to neutralize the naturally occurring inflammation that had caused the feverish patient's symptoms in the first place, by the process called "counterirritation."

The other especially drastic treatment used by colonial doctors was bleeding, on the humoral grounds that it removed any chemically or otherwise unbalanced blood that might be producing the patient's symptoms, and on the solidist grounds that it could relax tension on the hyperactive fibers of the fevered cardiovascular system. That is, pulling the plug to release some of the blood would reduce the friction between the blood and the walls of the arteries. Dr St Medard bled about 17% of his patients—and he was entirely typical of his time—although bleedings comprised only about 1.4% of all the treatments he gave. He removed an average of 12 ounces at each phlebotomy, twice as much from his sickest patients. He bled only patients with full, strong pulses, which indicated to him not that they had blood to spare, but that their circulatory systems were overexcited and that they needed to be calmed down, or sedated, by draining off their excess contents.

It is clear, then, that 18th century treatments were not designed specifically to counteract individual, well-defined disease processes, as modern drugs are designed to do. Instead, colonial physicians used drugs to adjust, to fine-tune, each patient's own special internal equilibria, his physiological balances, regardless of what might have disrupted them in the first place. The occurrence of catharsis, or vomiting, or sweating, or diuresis, or blistering after the administration of a corresponding drug indicated to the doctor and his patient that the remedy had indeed altered the humors and tones in the way that had been intended.[1] Both were well aware that many drugs were disagreeable to take, but even the most unpleasant effects provided good evidence that the drug had done something to the patient's body, that the doctor's fee represented money well spent.

PATTERNS OF PRACTICE AND PRESCRIBING

Comparisons of drug usage by four civilian practitioners reveal further patterns. The ledgers of Dr Josiah Bartlett (1729–1795) of Kingston, New Hampshire, provided a standard for making comparisons with those of three contemporary physicians in late 18th century New England. Bartlett's practice was used as a "baseline" chiefly because so many of his ledgers, covering 37 years of practice, have survived. Both he and Dr Benjamin Rowe, Jr (1750–1818), lived in small towns in southern New Hampshire, while Drs William Aspinwall (1743–1823) and David Townsend (1753–1829) lived and worked 50 or 60 miles away in the New England metropolis.[7] Unlike Dr St Medard, these four physicians included no clinical observations in their surviving records, which are primarily ledgers—records of financial transactions. However, because it was customary then to charge for each treatment rather than for the office visit or house call, the ledgers list all drugs prescribed for each patient.

The practices of the two rural New Hampshire doctors differed somewhat from those of their Boston colleagues.

For instance, only Drs Bartlett and Rowe extracted painful teeth; Aspinwall and Townsend did not, presumably because dental specialists had already appeared in Boston. On the other hand, Bartlett and Rowe delivered no babies at all, while Aspinwall and Townsend (and other urban practitioners) charged their largest nonsurgical fees for their obstetrical services. It seems likely that in rural New Hampshire babies were routinely delivered by midwives or by other women in the family, not by doctors. Although all four of these men performed some minor surgery, including opening abscesses and reducing simple fractures and dislocations, none of them performed the so-called "capital operations" of the day, such as removing cataracts or bladder stones, or amputations.

The four physicians prescribed a total of 225 different drugs all together, compounded from 100 different raw materials that they thought were the pharmacologically active ingredients of each drug. Some contained two active ingredients, and a very few contained three, but in general colonial American drug preparations were not very complicated. Table II lists the drug classes prescribed most often by Drs Bartlett, Rowe, Aspinwall, and Townsend. Some patients, especially those in Boston, were given no medicines at all, while the two rural doctors prescribed up to twice as many drugs for each patient as did their city colleagues.

Statistical analyses showed that the four physicians used the available drug classes with similar relative frequencies. That is, each drug class shared a similar priority in all four practices. For instance, like Peter St Medard, these doctors prescribed cathartics and tonics more often than any other drugs. However, each doctor had a favorite medicine within each category. For instance, Bartlett preferred elixir of camphor among the narcotics, while the other three men relied chiefly on opiates for the same purpose. And Aspinwall was even more remarkably dependent on an ancient formula called Spirits of Mindererus—originally an extract of aloes—when he wanted to prescribe a cathartic. But even these two aberrations do not substantially affect the relative rankings of drug class usage by those two physicians. It's just that Aspinwall, for example, thought that, because his favorite cathartic was also believed to have tonic properties, it would also benefit those of his patients who required such a stimulant.

Eighteenth century concepts of how drugs affected the body cannot be equated with those understood today. For instance, these four men did not always use even narcotics for the same reasons we do. They prescribed such drugs—usually opiates—most often for patients with diarrhea, and less frequently to relieve pain or to promote sleep. Today opium derivatives, especially morphine and codeine, are prescribed chiefly for pain, while their constipating effect is regarded as one of their most predictable, and annoying, side effects. All four of these physicians prescribed narcotic opiates, mostly in the form of laudanum (which is the same as paregoric), with about equal frequency.

Most colonial doctors relied on Peruvian bark or Virginia snakeroot when they wanted an all-purpose tonic. Aspinwall, on the other hand, not only preferred cascarilla for that purpose, he prescribed tonics only half as often as his colleagues did, probably because he thought his favorite cathartic, Spirits of Mindererus, would also provide all the strengthening his patients needed. Both rural doctors used antispasmodic drugs—those thought to calm down overexcited nervous systems, or "sedatives"—more often than the Boston physicians, while Aspinwall prescribed emetics far less frequently than the other three doctors.

Finally, Table II shows that the country doctors were much more likely than their Boston colleagues to bleed patients. Bleeding may have been less popular in the big city, or held in less esteem by doctors there, but it is also possible that bleeding was performed in the city by people who specialized in the technique, perhaps by apothecaries, after the treatment had been prescribed (but not billed for) by physicians.

The ten specific drug ingredients most often prescribed by Josiah Bartlett are listed in Table III along with the frequencies with which each was prescribed by his colleagues. The two New Hampshire doctors were the most alike in their reliance on these remedies. Detailed statistical studies showed that the four men differed more in their

TABLE II. Drug Classes Most Frequently Prescribed in Late 18th Century New England by Drs Bartlett, Rowe, Aspinwall, and Townsend[*]

Drug Class	Percent of All Prescriptions of Drs			
	Bartlett	Rowe	Aspinwall	Townsend
Cathartics	20.0%	27.5%	52.3%	21.8%
Tonics and astringents	25.8	25.1	13.1	23.9
Antispasmodics[†]	5.8	7.1	1.6	1.3
Narcotics	17.8	4.8	8.8	5.2
Emetics	3.5	5.7	0.9	8.4
Diaphoretics	2.1	2.9	2.2	7.6
Diuretics	1.5	2.3	1.3	2.8
Epispastics, blisters	4.9	5.9	3.0	2.0
Bleeding	5.8%	5.2%	0.8%	0.0%
Average number of drugs given to each patient	1.66	1.38	0.77	0.96

[*] Abstracted from work cited in reference 7.

[†] In effect, anti-tonics, but also called sedatives.

TABLE III. Ten Drug Ingredients, with Presumed Major Therapeutic Properties, Prescribed Most Often by Dr Bartlett, and Compared with the Frequencies with Which Each Was Prescribed by Drs Rowe, Aspinwall, and Townsend[*]

Active Ingredient (Drug Class)	Percent of Drugs Prescribed by			
	Bartlett	Rowe	Aspinwall	Townsend
Camphor (narcotic)	14.3%	3.8%	4.2%	1.6%
Peruvian Bark (tonic)	7.0	10.7	2.6	13.8
Cantharides (blister)	4.9	5.9	3.0	2.0
Rhubarb (cathartic)	4.1	3.9	2.5	1.0
Opium (narcotic)	4.0	3.4	6.7	3.7
Aloes (cathartic)[†]	3.8	5.3	25.5	1.4
Castor oil (cathartic)	3.5	2.7	0.5	0.8
Mercury (chiefly as cathartic)	3.4	4.3	1.3	2.8
Cajeput oil (antispasmodic)[‡]	3.0	1.1	0	0
Virginia snakeroot (tonic)	2.6	1.9	1.8	1.5

[*] Abstracted from work cited in reference 7.

[†] Essentially, the same as Aspinwall's favorite drug, Spirits of Mindererus.

[‡] Extracted from *Melaleuca leucadendron.*

usage of the drugs that each of them prescribed the most often than in their uses of those medicines they prescribed least frequently. That is, drugs which seldom found use in any one man's practice were used about as infrequently by all four. The men differed most in their favorite remedies, relying on past experience to help them select just the right drug for each patient's special needs.

TAILORING THERAPY TO THE INDIVIDUAL PATIENT

The third major pattern of 18th century drug usage emerges when we can determine exactly why a physician chose the treatments he prescribed for any given patient. The rationale that led to each selection can often be difficult to detect in retrospect, because the detailed medical record is such a comparatively recent invention. Even the few anecdotal case reports that have survived from colonial times can seldom be evaluated in modern terms. For instance, although infectious diseases, many of which produced severe fevers, were common causes of death in the colonies, physicians then could not measure temperature directly, with thermometers, because the short clinical thermometer did not enter everyday medical usage until after about 1860. Instead, colonial doctors depended chiefly on elevated pulse and respiratory rates as the best available indices of body temperature (Figs 3, 4).

No appropriately detailed and systematically recorded clinical observations from any large colonial practices have yet come to light, but some from the Royal Infirmary in Edinburgh have revealed many important clues to therapeutic thinking in the 18th century.[8] Although these data are not of American origin, they can be taken to be representative of medical reasoning throughout the English-speaking world of the time. Indeed, several physicians from Royal Infirmary, especially William Cullen (1710–1790), wrote many of the books that were most often read, consulted, and quoted by contemporary physicians in British North America.

We can see how and why Dr Andrew Duncan, Sr (1744–1828), made daily adjustments to the balances of his patients at the Infirmary in the manuscript case records which his students compiled as learning aids. The students recorded the symptoms, skin color, pulse and respiratory rates, number and quality of bowel movements, and other signs for each of Duncan's patients virtually every day they were in the hospital. In many instances, the students also recorded the reasons that dictated each therapeutic choice. The rationales for most other remedies could be found in the drug compendia and medical texts written by Duncan and his colleagues at the Infirmary.

Figure 5, for example, shows the daily changes in the pulse rate of Alexander Forbes, aged 22, when he was hospitalized in 1795 with a diagnosis of rheumatismus—which meant that he had severe pains in his head and chest, not that he had joint pains. Duncan's primary therapeutic objective in this case was to reduce the inflammatory, or feverish, hyperexcitability of the blood vessels of the head and chest. First he prescribed an opiate, to relieve the pain, and an emetic, to strengthen the stomach, to divert the responsible contagious factors away from the diseased organs and remove them from the body altogether. He prescribed cathartics for four days because Forbes was constipated, so that the contagious factors could escape in his stools. On day six Duncan applied a blister to the patient's chest—indicated by the star near the upper left of the figure—to remove what the doctor called the "morbid action" from the lungs by counterirritation.

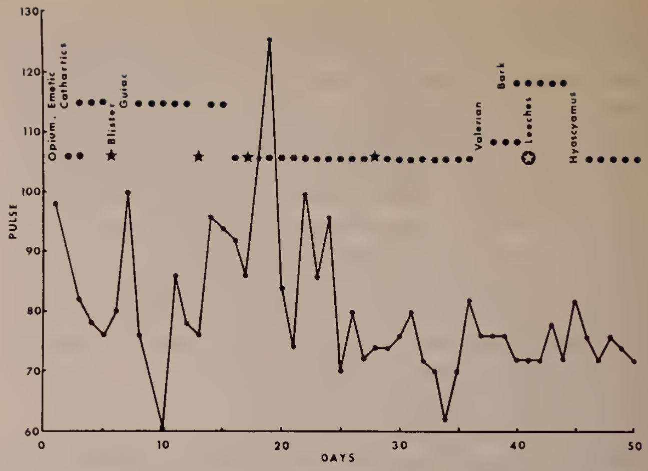


FIGURE 5. Clinical course of Alexander Forbes, admitted to the teaching ward of the Royal Infirmary of Edinburgh because of rheumatismus in the winter of 1795, as reflected by his pulse rate and treatments. Closed circles indicate days on which treatments indicated to the left in each row were administered to the patient; closed stars indicate blister applications; and the open star indicates the application of leeches. Reproduced with permission from work cited in reference 8.

Duncan gave him tincture of guaiac the next day because, as a tonic, it would strengthen the blood vessels and facilitate the removal of impure fluids into the urine and sweat. However, the pulse had not stabilized by day 13, so another blister was applied between the shoulder blades, and a third was started on the head four days later. Nevertheless, the pains continued, so opium was resumed, and a fourth blister was raised on day 28. By now the pulse was no longer fluctuating as wildly as it had been when the patient was first admitted.

On day 37, Duncan substituted valerian for opium, in his continuing attempt to calm the hyperexcited nervous system he thought was causing the headaches. Two days later he added Peruvian bark, because it was an astringent tonic. It was also considered to be an antiseptic, conveying the idea that it removed sepsis—another word for acute inflammation—from the body, presumably because patients with fevers usually recovered after taking the bark.

By day 40, although his pulse was relatively stable, Mr Forbes was not free from what Duncan interpreted as a "continued fever," as manifested by steady pain and signs of blood vessel constriction. So, on day 42, Duncan ordered six leeches to be applied to the patient's temples overnight, to remove enough blood to relax the inflamed vessels in the head. And three days later the doctor tried hyoscyamus as a pain remedy and to relax the nerves—he thought it was a sedative, somewhat like opium.

The graph does not show the last 15 days of Forbes' hospitalization, but during that time he was given more cathartics and opiates, and two more doses of eight leeches each applied to his temples. He was discharged on day 65 because his pulse had remained near normal for some time, indicating that his fibers had been relaxed by the treatments, just as his physician had set out to accomplish. However, although Dr Duncan could infer that he had treated the fever successfully, Mr Forbes still had his pains when he left the hospital.

Daniel Robertson, aged 34, was admitted with pneumonia, characterized by chest pain, cough, and copious expectoration of phlegm (Fig 6). He was treated first with a blister in the middle of his chest, and then with crystals of tartar (sodium potassium tartrate, also called cremor tartar or Rochelle salt), a mild cooling cathartic which Duncan thought would help remove fluid from Robertson's septic lungs; he was not constipated. Opiates were begun on day 14 to help him sleep, and another blister was raised on his chest to counteract the inflammation deep inside it.

Two days later, Duncan began prescribing only charcoal suspended in water, in an explicitly experimental attempt to absorb the septic material out of his patient's body. He then added opiates on days 33 to 39, but this time it was to reduce the number of stools, not for pain. Robertson's pulse finally stabilized after two months, although it was still higher than normal, and he was discharged. Dr Duncan concluded that the experimental charcoal probably had hastened this patient's recovery.

Leslie Campbell was a 22-year-old mother admitted with an intermittent (quartan) fever; the small stars along the bottom left of Figure 7 indicate that she had the characteristic severe chills and shivering fits every third day, at about 4:30 in the afternoon. Duncan treated her first with cinchona, the Latin name for Peruvian bark, but when it failed to cure her he tried another experiment, with soymida bark, a new tonic drug recently imported from India. But Mrs Campbell continued to have her intermittent fits every three days.

Duncan then prescribed emetics, to strengthen her body via its connections with the stomach. He tried garlic for a few days, and corrosive sublimate (mercuric chloride)—labelled as "Hg" at the top of the graph. He thought that both should enhance the tone of the whole body. But neither of them helped Mrs Campbell, despite Duncan's opinion that "unquestionably they are capable of combatting the disease."

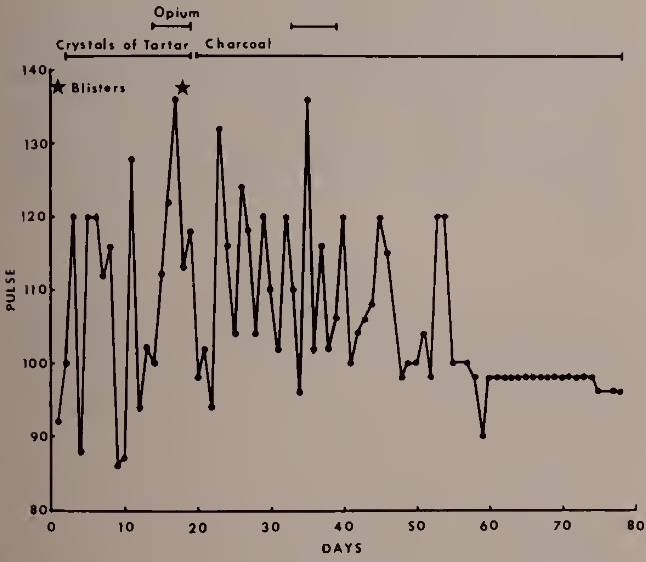


FIGURE 6. Clinical course, as reflected by pulse rate and treatments, of Daniel Robertson after his admission to the Royal Infirmary of Edinburgh with a diagnosis of phthisis catarrhalis. The duration of each treatment is indicated by the horizontal lines at the top, and applications of blisters by closed stars. Reproduced with permission from work cited in reference 8.

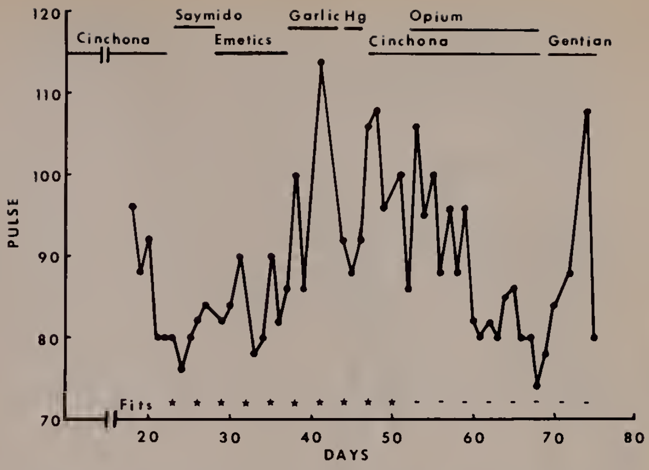


FIGURE 7. Clinical course, as reflected by pulse rate, occurrence of "fits," and the treatments prescribed by Dr Andrew Duncan, Sr, of Mrs Leslie Campbell, admitted to the Royal Infirmary of Edinburgh for quartan fever. The duration of each treatment is indicated by the horizontal lines at the top. The small stars at the bottom indicate days on which she had the characteristic attacks of this form of intermittent fever, or malaria, and the dashes to the right of the stars indicate days on which she did not suffer the attacks that were expected on each. Reproduced with permission from work cited in reference 8.

Finally, on day 47, he tried cinchona again, but this time he prescribed eight times the dose he had given earlier with no success. After day 50 she had no more attacks of her fever, as seen at the bottom of the graph. Duncan also gave her opiates for pain and to sedate her blood vessels; the pulse graph shows that is what actually happened, insofar as Duncan could tell. At the end he gave her gentian to stimulate her stomach once more, so he was not surprised just afterward when her pulse rose temporarily on day 74, just before he discharged her.

James Lander, a 50-year-old gardener, was admitted with shortness of breath caused by the accumulation of fluid within his chest cavity, the syndrome then called dropsy (from the Greek word *hydrops*, for fluid). Because the pulse was almost always rapid, as in this case, physicians thought dropsy was a fever, one that made vascular fibers so weak that fluids could leak out of them into other tissues. Not for many years would it be recognized that in dropsy the heart beats faster in order to compensate for the weakened force of cardiac contraction that is characteristic of this syndrome, now called congestive heart failure.

Duncan first raised a blister on his patient's chest (Fig 8), to promote an artificial evacuation of the dropsical chest fluid by counterirritation. The next day he gave jalap, a very potent cathartic, to provide a "natural" route for removing the fluid that was making Mr Lander short of breath. At the same time, Duncan began giving him digitalis because, as William Withering (1741–1799) had demonstrated in Birmingham, England, in 1785, it was the only dependably effective drug then available for the treatment of dropsy. Duncan, Withering, and their con-

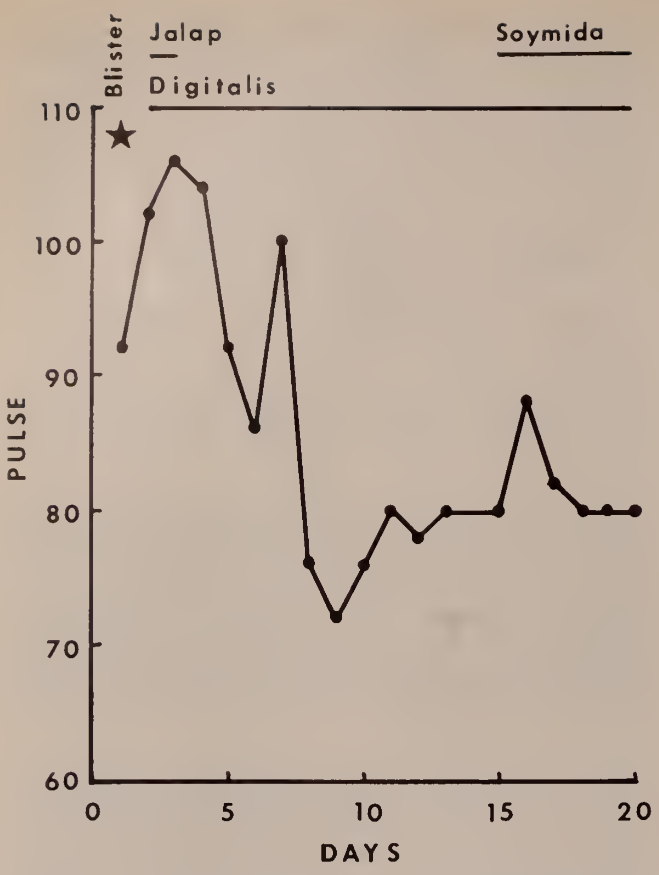


FIGURE 8. Clinical course, as reflected by pulse rate and by the treatments prescribed by Dr Duncan, of James Lander, admitted to the Royal Infirmary of Edinburgh because of hydrothorax and dropsy. The horizontal lines indicate the duration of each treatment, and the star indicates a blistering. Reproduced with permission from work cited in reference 8.

temporaries thought digitalis was a diuretic, because it provoked the removal of excess fluid as urine; they could not have known that the direct effect of the drug is on the heart muscle, to strengthen it when it has become weakened, and that the diuresis is secondary to the resulting increased blood flow to the kidneys.

In this case, diuresis did occur, Lander's symptoms were relieved, and his pulse fell toward normal. Duncan added soymida bark for the next few days because he thought the new tonic might further strengthen the weak blood vessels from which fluid had leaked into the chest, but it gave no additional relief. The doctor concluded that this cure was definitely attributable to the drugs he had prescribed. He also surmised that some of his other patients probably would have recovered without any treatment, because of the body's tendency to heal itself, he said.

CONCLUSIONS

As noted earlier, drug use in colonial America can be studied and interpreted within the contexts of several different patterns. Indeed, in order for a historical drug to be interpreted in a meaningful way, it should be presented within only one carefully defined context at a time. For instance, merely labelling a given drug as a cathartic, or as a diuretic, tells one little about exactly why the prescribing physician chose it for his patient; such a label merely indicates an obvious effect, not a mechanism of therapeutic action.

Although the three patterns described above overlap to some extent, each can be exploited historically all by itself. The usefulness of any one pattern as an interpretive framework will depend on whether one is attempting to place a given drug into its overall physiological context of humors and solids, or into its usage pattern by a specific group of physicians, including various "specialists," or into a sequence of treatments provided by one physician for a given patient, taking the particular symptoms into account. Each situation will require choosing and exploiting a different kind of pattern in order to explain the drug's use as clearly as possible.

One question of interpretation remains: What good did the drugs prescribed by colonial physicians do for their patients? Today it is fairly easy to ridicule historical remedies, especially when we know that only a few of them—notably opium, cinchona, and digitalis—could have affected the course of certain illnesses in ways that we can recognize two centuries later. And even in those three instances, 18th century explanations of their efficacy as medicines differed markedly from modern concepts of the sites and modes of their pharmacologic action.

Still, despite the fact that most of the more than 200 drugs prescribed in the 18th century were not truly effective by today's criteria, patients continued to seek professional medical care when they felt they needed it, because they had every reason to believe that it helped them. Many colonial American physicians agreed with Andrew Duncan at Edinburgh that the body can sometimes heal itself, but they went on prescribing drugs that they, and their patients, thought were therapeutic. After all, not only did the drugs move their patients' bowels, make them throw up, lower their pulse rates, or do whatever else the physician wanted to happen, in the end a very large majority—up to about 95%—of adult patients who sought professional medical help managed to recover, no matter how they were treated (at least in the absence of major epidemics of devastating diseases like smallpox). Thus, the medical standards of the 18th century permitted most treatments to be assessed as truly effective, although today we might be more inclined to attribute much of their apparent efficacy to the *vis medicatrix naturae*, which is how Josiah Bartlett liked to refer to the healing powers of nature.

REFERENCES

1. Rosenberg CE: The therapeutic revolution: Medicine, meaning, and social change in nineteenth-century America. *Perspect Biol & Med* 1977; 20:485-506 (essential to a thorough understanding of the apparent efficacy of historical medicines, especially in America).

2. *Encyclopaedia Brittanica* (3 vols). Edinburgh, A. Bell and C. Macfarquhar, 1771, vol 3, pp 60-61. The description of fever on these pages is an unusually lucid explanation of contemporary concepts of its pathophysiology:

> Hoffman defines a fever to be, "A spasmodic affection of the whole nervous and vascular system, annoying all the functions of the body, arising from any cause which has power to irritate the nervous parts to a more intense contraction; and when it operates, it drives the vital fluids from the outward parts to the heart and great vessels; and afterwards, when the systole of the heart and arteries are increased, they are drove [sic] back with rapidity and heat, through the constricted vessels, to the outward parts again, till the spasms being relaxed, the secretions are performed, and the fever vanishes."
>
> The formal or fundamental cause of a fever consists in the spasmodic affection of the whole nervous and fibrous genus. This plainly appears from the usual phenomena of a fever, *viz.* a pain in the back, more particularly about the loins; a coldness, especially of the extreme parts; a shivering, shaking, trembling; a livid

colour of the nails; a subsidence of the vessels of the hands and feet; a shrunk, dry skin; a yawning; a stretching; a pale, livid countenance; a trembling and palpitating motion of the heart; an anxiety of the praecordia, difficult breathing, inquietude, restlessness; a sensation of an ebullition of the blood about the heart; a contracted, weak, small pulse; a nausea, and an inclination to vomit; a suppression of perspiration; costiveness [constipation], with thin watery urine.

Hence it naturally follows, that whatever has a power to irritate the nervous and vascular system to spasms, is most likely to generate a fever . . .

The cause of the febrile motion is an universal spasm; and that motion never ceases till the spasm is resolved. The signs of its being resolved are, a free perspiration, and a breathing sweat; the pulse, which before was hard, impetuous, and quick, becomes soft, moderate, and slow; the urine lets fall a sediment, and the strength gradually returns. When these appear all together, they declare the solution of the disease, and are called the crisis.

3. Geddes LA: *A Short History of the Electrical Stimulation of Excitable Tissue*. Supplement to *The Physiologist* 1984; 27:S1-S7.

4. St Medard P: *Physical and Chirurgical Transactions*, September 1, 1802–December 9, 1803, in USS *Constitution* Museum, Boston.

5. Estes JW: Naval medicine in the age of sail: The voyage of the *New York*, 1802-1803. *Bull Hist Med* 1982; 56:238-253 (includes details of Dr Peter St Medard's shipboard therapies).

6. Estes JW: A naval surgeon in the Barbary Wars: Dr. Peter St. Medard on *New York* 1802-3, in Dept of History, US Naval Academy (ed): *New Aspects of Naval History*. Baltimore, Nautical and Aviation Publishing Co, 1985, pp 81-92 (here the emphasis is on St Medard's diagnoses).

7. Estes JW: Therapeutic practice in colonial New England, in Cash P, Christianson EH, Estes JW (eds): *Medicine in Colonial Massachusetts, 1620-1820*. Boston, Colonial Society of Massachusetts, 1980, pp 289-383. (The entire volume provides many helpful overviews; for more on drugs, see Gifford GE Jr: Botanic remedies in colonial Massachusetts, pp 263-288.)

8. Estes JW: Drug usage at the infirmary: The example of Dr. Andrew Duncan, Sr., 1795, Apppendix D, in Risse GB: *Hospital Life in Enlightenment Scotland: Care and Teaching at the Royal Infirmary of Edinburgh*. New York, Cambridge University Press, 1986, pp 351-384. (Some of the most influential early American physicians studied at Edinburgh, and books written by the faculty there were in the libraries of many other American doctors. The entire book, therefore, goes far toward explaining medical treatments on this side of the Atlantic as well.)

Annotated Bibliography

Davis A, Appel T: *Bloodletting Instruments in the National Museum of History and Technology*. Washington, DC, Smithsonian Institution Press, 1979 (a good overview of the entire subject.)

The Edinburgh New Dispensatory, ed 4. Edinburgh, William Creech, 1794. (Other editions, so long as they were not published too long after the time period under study, are equally helpful for "decoding" most British drugs of the same period. The contemporary *Dispensatories* published in London are more helpful for drugs used in southern England; specific methods for compounding some drugs differed in the two capital cities.)

Estes JW: John Jones' *Mysteries of Opium Reveal'd* (1701): Key to historical opiates. *J Hist Med & All Sci* 1979; 34:200-209 (a comparison of 15 opium preparations used in the 17th and 18th centuries).

Estes JW: *Hall Jackson and the Purple Foxglove: Medical Practice and Research in Revolutionary America, 1760-1820*. Hanover, NH, University Press of New England, 1979. (See chapters 4-7 for a detailed study of the introduction of digitalis, a truly effective drug, to medical use in both America and Europe.)

Estes JW, Kuhnke L: French observations of disease and drug use in late eighteenth-century Cairo. *J Hist Med & All Sci* 1984; 39:121-152 (demonstrates similarities among drug uses in America and elsewhere, and global trade routes for some drugs).

Estes JW: George Washington and the doctors: Treating America's first superhero. *Med Heritage* 1985; 1:44-57. (The well-documented example of the lifetime of this one patient illustrates the clinical usage of several 18th century drugs.)

Forbes TR (ed): *The Admirable Secrets of Physick and Surgery*, by Thomas Palmer. New Haven, Conn, Yale University Press, 1985. (Palmer's 1696 "vademecum" exemplifies the confluence of ancient humoral therapies with medieval concepts that were disappearing from common usage by then.)

Jones GW (ed): *The Angel of Bethesda: An Essay upon the Common Maladies of Mankind*, by Cotton Mather. Barre, Mass, American Antiquarian Society and Barre Publishers, 1972. (Although unindexed, Mather's 1724 manuscript, here published for the first time, is a goldmine of colonial remedies.)

King LS: *The Medical World of the Eighteenth Century*. 1958, reprint ed. Huntington, NY, Krieger Publishing Co, 1971 (a good review of the theories underlying most treatments).

Leighton A: *Early American Gardens, "For Meate or Medicine,"* Boston, Houghton Mifflin Company, 1970 (includes a wealth of information about plant drugs in the 17th century).

Leighton A: *American Gardens in the Eighteenth Century, "For Use or Delight."* Boston, Houghton Mifflin Company, 1976 (valuable on medicinal uses of plants of the time).

Thacher J: *The American New Dispensatory*. Boston, Thomas B. Wait & Co, 1810. (The editions of 1813, 1817, and 1821 are equally useful for "decoding" drug uses in late 18th century America.)

A subglottic foreign body in an adult patient

RAMIRO REQUENA, MD; RAMESHBHAI PATEL, MD; INDERJIT SINGH, MD

Laryngeal foreign bodies are rare in the adult. They are, however, fairly common in children and comprise about 2–4% of all foreign bodies in the airways.[1,2] Laryngeal foreign bodies are accompanied by a high incidence of complications. Advances in bronchoscopic and anesthetic techniques have facilitated the removal of laryngeal foreign bodies and have lowered the associated mortality rate to about 1%.[3] Tracheostomy and laryngofissure may be required to remove foreign bodies impacted in the subglottic region.

CASE REPORT

A 47-year-old man was admitted to the hospital because of severe respiratory distress, stridor, and cough. His voice was hoarse and barely audible. He reported having accidentally aspirated a bone while eating mutton soup 18 months before. During this period, he saw several physicians, but a foreign body could not be detected. Instead, he was treated for allergic bronchitis.

Physical examination showed a middle-aged man who was moderately dyspneic. A radiograph of the neck did not reveal any abnormal radiopaque density. Direct fiberoptic laryngoscopy disclosed hyperemic and edematous epiglottis and vocal cords. A foreign body was seen through the opening of the vocal cords in the subglottic region. The position of the foreign body was in the midline, and resembled the carina.

An attempt at extraction with a biopsy forceps failed, because the foreign body was imbedded in inflammatory tissue. A tracheostomy was performed under local anesthesia. General anesthesia was administered through the now controlled airway, and the foreign body (a piece of bone) was removed by means of a laryngofissure. After surgery, the patient received antibiotics, steroids, and respiratory support therapy. He was discharged from the hospital on the sixth day after surgery.

DISCUSSION

Aspiration of foreign bodies into the airways is common in children one to three years of age, but is rare in children older than six.[3,4] Airway obstruction due to foreign bodies is extremely rare in adult patients. Cohen et al,[1] in a review of 143 cases of foreign bodies in the airways, found that only 10% of these cases occurred in children over six years of age, and only two cases occurred in children over the age of 12. In another series of 225 cases of tracheobronchial foreign bodies, Harboyan and Nassif[5] found only five cases (2.2%) in patients more than 12 years old. Similar experiences were reported by Aytac et al[3] and Schloss et al.[6] Aytac et al reported only two cases in adults in a series of 600 cases of foreign bodies in the airways.

The larynx is an uncommon site for foreign bodies to lodge. Harboyan and Nassif[5] reported only one case of a laryngeal foreign body in their series of 225 cases. Rothman and Boeckman[2] reported an incidence rate of laryngeal foreign bodies of 2%, while Cohen et al[1] reported an incidence rate of 4%.

According to Brama and Fearson,[4] laryngeal foreign bodies may either be sharp and thin, such as chicken bones or a portion of eggshell, or blunt and large, such as a piece of meat. The latter usually requires immediate treatment, but the former may remain embedded for prolonged periods before causing edema, infection, and symptoms of upper airway obstruction. Although subglottic foreign bodies have been reported in children, we are unaware of any reported case in adults.

More than half of patients with foreign bodies in the upper airway seek medical care within the first 24 hours of the event. However, considerable delay in seeking treatment may occur depending on the location of the foreign body. Zajaczkowska[7] reported on a patient who had had a foreign body lodged in the tracheobronchial tree for 15 years.

The most common clinical signs of laryngotracheal foreign bodies are acute respiratory distress with cyanosis, cough, and wheezing. If the foreign body remains in the tracheobronchial tree for a prolonged period, bronchiectasis, chronic bronchitis, asthma, bronchial stenosis, and lung abscess may complicate the clinical picture.

Radiopaque foreign bodies can be detected in plain chest or neck films, while radiolucent foreign bodies can produce secondary pathologic changes in the chest film, such as infiltrates and atelectasis. Loss of patency in bronchograms and unilateral pulmonary hypertension can also occur.[3]

Laryngoscopic or bronchoscopic removal of the foreign body is the treatment of choice.[1–4] As laryngeal foreign bodies are associated with more complications than foreign bodies in other locations (even after their removal), these patients should be monitored for airway problems. Tracheostomy or intubation should be performed expeditiously, if necessary.[3,4] Routine use of steroid therapy after the removal of tracheobronchial foreign bodies helps to decrease the need for tracheostomy.[3] A preliminary tracheostomy may be required in emergency situations, or when severe glottic edema precludes bronchoscopic extraction.[8] Longstanding subglottic foreign bodies causing severe edema and fibrosis may require tracheostomy and laryngofissure to ensure safe removal.

REFERENCES

1. Cohen SR, Herbert WI, Lewis G Jr, et al: Foreign bodies in the airway. Five year retrospective study with special reference to management. *Ann*

From the Department of Surgery, Jewish Hospital of Brooklyn, Interfaith Medical Center, Brooklyn, NY.

Address correspondence to Dr Requena, Associate Director, Department of Surgery, Interfaith Medical Center, 555 Prospect Pl, Brooklyn, NY 11238.

Otol Rhinol Laryngol 1980; 89:437-442.
2. Rothman BF, Boeckman CR: Foreign bodies in the larynx and tracheobronchial tree in children: A review of 225 cases. *Ann Otol Rhinol Laryngol* 1980; 89:434-436.
3. Aytac A, Yurdakul Y, Ikizler C, et al: Inhalation of foreign bodies in children: Report of 500 cases. *J Thorac Cardiovasc Surg* 1977; 74:145-151.
4. Brama I, Fearson B: Laryngeal foreign bodies in children. *Int J Pediatr Otorhinolaryngol* 1982; 4:259-265.
5. Harboyan G, Nassif R: Tracheobronchial foreign bodies—A review of 14 years' experience. *J Laryng* 1970; 84:403-412.
6. Schloss MD, Pham-Dang H, Rosales JK: Foreign bodies in the tracheobronchial tree—A retrospective study of 217 cases. *J Otolaryngol* 1983; 12:212-216.
7. Zajaczkowska J, Gluskowski J, Byszewska D, et al: Ciala obce zalegajace w oskrzelach u doroslych. *Gruzlica* 1973; 41:373-383.
8. Chatterji S, Chatterji P: The management of foreign bodies in air passages. *Anaesthesia* 1972; 27:390-395.

Cystic artery pseudoaneurysm

JOHN W. RHEE, MD; DAVID C. BONNHEIM, MD; JAMES UPSON, MD

Pseudoaneurysm of the cystic artery is a rare complication of acute cholecystitis. In the past, this diagnosis has been made primarily at the time of surgery. We describe a patient whose clinical history gave us a high index of suspicion for visceral artery aneurysm. Using preoperative arteriography, we were able to establish with relative certainty a diagnosis of cystic artery pseudoaneurysm, thereby simplifying the surgical procedure.

CASE REPORT

A 73-year-old man with a history of cholelithiasis sought treatment at another hospital for severe pain in the right upper quadrant and fever of two days' duration. A drop in the hemoglobin level from 9 to 7 mg/dL and leucocytosis were noted. A mass was palpable in the right upper quadrant. No melena or hematochezia was noted.

Due to the patient's multiple medical problems, the initial cholecystostomy was performed under local anesthesia at an outlying hospital. The abdominal cavity was found to have free dark blood, a hematoma of the right mesocolon, and a distended, acutely inflamed gallbladder approximately 5 cm in diameter. A cholecystostomy was attempted, and multiple gallstones, blood clots, and bright red blood were evacuated. No bleeding point could be identified before the site was hastily closed. The patient was transferred to this hospital.

On the patient's arrival, an angiogram was done. This demonstrated an aneurysmal, cystic artery with gross extravasation of contrast material into the gallbladder (Fig 1). At surgery, a subhepatic hematoma and an aneurysmal cystic artery eroding into the distended gallbladder were identified. Proximal control of the common hepatic artery was achieved, thus allowing complete occlusion of the blood supply to the cystic artery. After dissection of the fundus of the gallbladder off of the liver bed—with the base of the gallbladder still attached—the vascular clamps were released, allowing for the visualization of a ruptured aneurysmal cystic artery. The remaining portion of the gallbladder was removed, and the cystic artery was oversewn with an absorbable suture. No other abnormalities were demonstrated during the remaining surgical exploration.

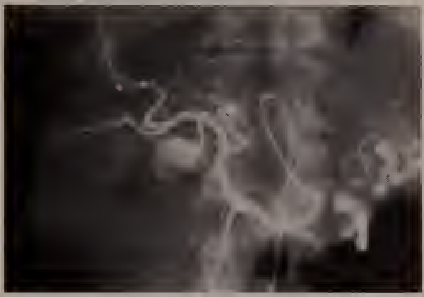


FIGURE 1. Visceral angiogram demonstrating a cystic artery aneurysm.

The patient did well after surgery and had an uneventful recovery. He was discharged from the hospital on the seventh day after surgery. The culture of the gallbladder grew enterococci. The pathologist reported that the gallbladder wall was edematous and acutely inflamed.

DISCUSSION

Cystic artery aneurysm is a rare entity, and its cause remains obscure. It appears that this patient had a pseudoaneurysm, since it apparently developed from the erosion of an inflamed gallbladder into the wall of the cystic artery. The patient's heart was in normal sinus rhythm, and there was no evidence of a septic embolus to any other area. Thus we doubt that the false aneurysm was secondary to a septic embolus.

This patient presented with signs and symptoms of acute cholecystitis; hemobilia may have been manifested by the drop in hemoglobin level. Angiography was helpful in clarifying the diagnosis and simplifying the surgical procedure. Bleeding was minimized by clamping the hepatic artery before proceeding with the cholecystectomy.

Although acute cholecystitis is common, the formation of a pseudoaneurysm and subsequent rupture into the gallbladder is infrequent, because the inflammatory process usually leads to thrombosis of the cystic artery. Hakami et al[1] described a patient with massive hemorrhage from a cystic artery aneurysm, presumably resulting from damage to the cystic artery caused by a large calculus in the gallbladder. Berland et al[2] described a patient with hemorrhage into the gallbladder diagnosed by preoperative computed tomography. The source of the hemorrhage was probably portal hypertension from large veins in the wall of the gallbladder. Devan[3] described a patient with a cystic artery aneurysm, the patient having presented with clinical signs suggestive of pancreatitis. Without the benefit of an arteriogram before surgery, dissection of both the right and common hepatic arteries was required before a diagnosis of a cystic artery aneurysm could be ascertained.

REFERENCES

1. Hakami M, Beheshti G, Amirkhan A: Hemobilia caused by rupture of cystic artery aneurysm. *Am J Proctol* 1976; 27:56-57.
2. Berland LL, Doust BD, Foley WD: Acute hemorrhage into the gallbladder diagnosed by computed tomography and ultrasonography. *J Comput Tomogr* 1980; 4:260-262.
3. Devin R, Mambrini A, Winninger A, et al: Un cas d'anévrysme de l'artère cystique rompu dans la vésicule biliaire. *Chirurgie* 1971; 97:332-335.

From the Department of Surgery, The Buffalo General Hospital, and the State University of New York at Buffalo, Buffalo, NY.

Address correspondence to Dr Upson, 85 High St, Buffalo, NY 14203.

Mesonephric hamartoma of the seminal vesicle: A rare cause of a retrovesical mass

HELEN KINAS, MD, MATTHEW J. KUHN, MD

Mesonephric hamartomas are rare tumors.[1] They represent primary benign epithelial masses composed of ductal and stromal proliferation, which arise in mesonephric duct derivatives.[2] Mesonephric duct hamartomas are known to develop in the vas deferens; a review of the literature did not disclose any prior reports of a mesonephric hamartoma arising in the seminal vesicle. We report a case of this unusual entity, emphasizing its benign radiologic appearance, which helps to distinguish it from the more common tumors of the prostate and seminal vesicles that it may superficially resemble.

CASE REPORT

A 63-year-old man was found to have a pelvic mass on physical examination. He reported no symptoms except for an occasional sensation of increased pressure before voiding. His medical history was significant only for a myocardial infarction.

An intravenous pyelogram (IVP) (Fig 1) showed extrinsic compression of the urinary bladder by a pelvic mass. A barium enema (Fig 2) revealed the pelvic mass displacing the rectosigmoid to the left and upwards. A computed tomographic (CT) scan of the pelvis (Fig 3) demonstrated a large, sharply demarcated, soft tissue density located on the posterolateral aspect of the urinary bladder. The urinary bladder and rectum were displaced toward the left side by the mass but there were no signs of invasion. The mass extended from the bladder to the sacrum without obliteration of the fat planes, thus displaying characteristics of a benign lesion. There were areas of inhomogeneity within the mass. Pelvic arteriography (Fig 4) revealed a hypovascular soft tissue mass in the mid-pelvic region, with minimal focal neovascularity.

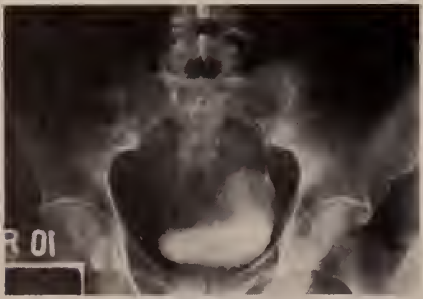


FIGURE 1. Ten-minute film of the pelvis from an IVP demonstrates a mass externally compressing the right side of the urinary bladder.

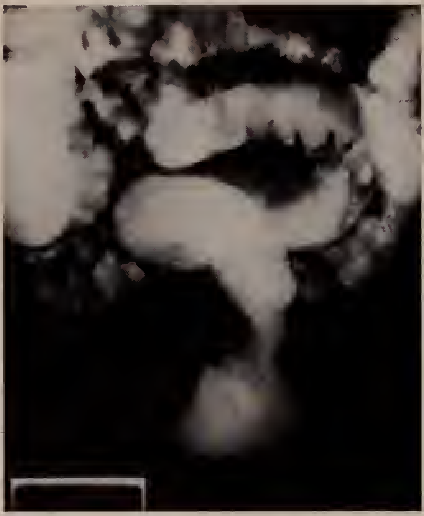


FIGURE 2. A barium enema shows elevation and displacement of the rectum and sigmoid by a predominately right-sided pelvic mass.

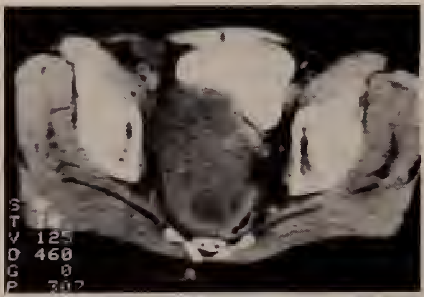


FIGURE 3. Contrast enhanced CT scan of the mass demonstrates that the surrounding fat planes are intact. The urinary bladder and rectum are displaced but not invaded. Areas of inhomogeneity are seen within the mass.

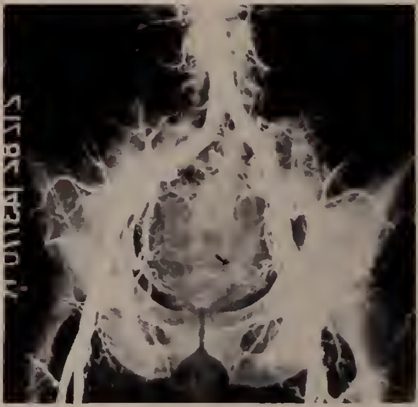


FIGURE 4. Pelvic arteriography shows that the mass is largely hypovascular with pelvic arteries draped around it. A small, focal area of neovascularity is demonstrated (arrow).

At exploratory laparotomy, a large mass arising from the right seminal vesicle was found and excised. Exploration of the peritoneal cavity disclosed no abnormalities. The pathologic specimen (Fig 5) was found to be a hamartoma of the seminal vesicle with stromal proliferation. The patient had a normal postoperative course and was dis-

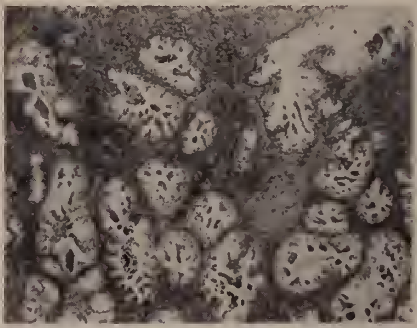


FIGURE 5. Photomicrograph of surgical specimen of hamartoma of the right seminal vesicle demonstrating glandular, smooth muscle, and vascular proliferation with distortion of normal architecture (hematoxylin-eosin stain; original magnification × 50).

From the Department of Radiology, Beth Israel Medical Center, Mount Sinai School of Medicine, New York, NY.

Address correspondence to Dr Kinas, Department of Radiology, Beth Israel Medical Center, First Ave at 16th St, New York, NY 10003.

charged from the hospital; he suffered no residual problems.

DISCUSSION

Retrovesical masses in men are usually related to prostate carcinoma or hyperplasia, colon carcinoma, or lymphoma. Primary tumors or inflammatory masses arising from the seminal vesicles are rare, but may also present as retrovesical masses on radiological studies. We are not aware of any prior reports of hamartoma of the seminal vesicle. This case is presented as an additional consideration in the radiologic diagnosis of pelvic masses.

The seminal vesicles originate as diverticula from the caudal portion of each mesonephric duct. Other mesonephric duct derivatives in men include the vas deferens, ureters, renal collecting systems, appendix of the epididymis, ductus epididymis, and ejaculatory ducts. Small cystic mesonephric duct remnants can occasionally be identified on the posterolateral aspect of the urinary bladder along the course of the vas deferens.[3]

A hamartoma is a tumor composed of mature, differentiated normal cells and tissues that have formed an abnormal focal overgrowth in an organ composed of identical cellular elements.[4] Hamartomas are known to occur in many organ systems throughout the body; they are extremely rare in mesonephric duct derivatives.

A hamartoma of the seminal vesicle must be distinguished histologically from other benign and malignant lesions of the seminal vesicle and adjacent structures. All of these tumors are rare. Benign lesions include seminal vesicle cysts, fibromas, myomas, cystic adenomas, and amyloid infiltration. Primary adenocarcinoma is the most common malignant tumor of the seminal vesicle, although it is a rare tumor and has often been misdiagnosed when prostate carcinoma extends into the seminal vesicle.[5] Only four confirmed cases of sarcomas of the seminal vesicle have been reported.[6]

A mesonephric hamartoma of the seminal vesicle must also be distinguished from other pelvic neoplasms, especially benign prostatic hyperplasia and prostate carcinoma, as well as from pelvic masses representing lymphoma and retroperitoneal sarcomas.

With no reports in the literature of a mesonephric hamartoma of the seminal vesicle, a histologic diagnosis of this disorder from radiologic studies alone would be virtually impossible. Despite its large size, however, this lesion demonstrates many characteristics typical of a benign mass and should not be mistaken for a malignancy. The mass in this case caused only external compression of the urinary bladder as seen on the IVP, and, based on findings of the barium enema, merely displaced the rectosigmoid. Computed tomography was most useful in demonstrating the benign nature of this lesion, in that the tumor was clearly seen to be smooth with well circumscribed, distinct margins, and the surrounding fat planes were preserved. The seminal vesicles are located anterior to Denonvilliers' fascia, separating it from the prostate and ampullae of the rectum.[3] These tissue planes are not disrupted by benign lesions.

Mesonephric hamartomas represent primary benign epithelial masses composed of ductal and stromal proliferation that arise in mesonephric duct derivatives. We report what we believe to be the first case of this unusual entity in the seminal vesicle. The benign radiologic appearance of this lesion distinguishes it from the more common malignancies of the prostate and seminal vesicles, which it may resemble.

Acknowledgments. The authors thank Alyssa Kuhn and Osmay Yalis for their assistance in preparation of the manuscript.

REFERENCES

1. Tamayo JL, Ruffolo EH: Spermatic cord tumor: Mesonephric hamartoma of the vas deferens. *Arch Surg* 1967; 94:430–431.
2. Graham JB, O'Connor VJ: Spermatic cord tumors: Review of literature and case of unusual vas deferens tumor in infertility problem. *J Urol* 1954; 72:946–949.
3. Walsh PE (ed): *Textbook of Urology*, ed 5. Philadelphia, WB Saunders Co, 1986.
4. Robbins SL, Cotran RS, Kumar V: *Pathologic Basis of Disease*, ed 3. Philadelphia, WB Saunders Co, 1984.
5. Benson RC, Clark WR, Farrow GM: Carcinoma of the seminal vesicle. *J Urol* 1984; 132:484–485.
6. Buck AC, Shaw RE: Primary tumours of the retro-vesical region with special reference to mesenchymal tumours of the seminal vesicles. *Br J Urol* 1972; 44:47–50.

Choledochal cyst in an adult

NANAKRAM AGARWAL, MD, BENEDICT M. REYNOLDS, MD

Choledochal cyst is rare in the adult population. In the past it had been infrequently diagnosed prior to surgical exploration for obstructive jaundice with cholangitis. With the advent of sonography, computed tomographic (CT) scanning, and endoscopic and percutaneous transhepatic cholangiography, the preoperative diagnosis is usually apparent. The decision as to the appropriate surgical therapy in the adult is the subject of this presentation.

CASE REPORT

A 27-year-old woman had been admitted to the hospital in 1967 at the age of 11 years for epigastric pain, vomiting, and fever of about four days' duration. Except for a similar episode of abdominal pain at five years of age, she had been in good health.

Physical examination on admission revealed a temperature of 100°F, markedly enlarged, congested tonsils, and slight tenderness in the epigastrium. An initial white blood cell count was 12,000/mm^3; the differential was within normal limits. Liver chemistry studies, urinalysis, and a chest radiograph disclosed no abnormalities. An

---

From the Department of Surgery, Our Lady of Mercy Medical Center, New York Medical College, Bronx, NY.

Address correspondence to Dr Agarwal, Department of Surgery, Our Lady of Mercy Medical Center, 600 East 233rd St, Bronx, NY 10466.

oral cholecystogram demonstrated fair concentration of the contrast material in the gallbladder, but there was an unusual contour which raised the possibility of a double gallbladder. An intravenous cholangiogram revealed a normal gallbladder and marked dilation of the hepatic radicles and the proximal portion of the common duct as well as the cystic duct, with some narrowing of the distal portion of the common bile duct. There was no definite evidence of calculus, and no delay in excretion of the contrast material.

The patient was treated with intravenous oxytetracycline. Her condition improved, and as throat cultures were positive for *Streptococcus viridans*, she was discharged with a diagnosis of acute tonsillitis and mesenteric adenitis.

In July 1974, at 17 years of age, the patient was admitted to a hospital in Puerto Rico and underwent cholecystectomy, exploration of the common bile duct, and choledocholithotomy. A T-tube cholangiogram showed dilation of the common bile duct with a 2.5-cm stenosis just proximal to the ampulla. The patient was discharged from the hospital with a T-tube in place. One month later, she was readmitted, and a T-tube cholangiogram revealed a stone measuring 0.5 cm in the distal part of the common bile duct. She again underwent exploratory laparotomy, and this time a choledochocystduodenostomy was performed. The patient did well after surgery.

She remained asymptomatic until June 1977, when she was admitted to the hospital with pain in the right upper quadrant. A physical examination, white blood cell count, blood chemistry studies, and urinalysis were negative. Her pain subsided, and she was discharged the next day. Subsequently, sonography showed stones in the cyst. In September 1977, she was again admitted to the hospital with colicky pain in the right upper quadrant, vomiting, fever, and jaundice. No abdominal masses were palpated on physical examination. Laboratory data revealed a white blood cell count of 19,000/mm^3 with a marked shift to the left, an increased serum alkaline phosphatase level, and a total bilirubin of 4.8 mg/dL (direct bilirubin, 3.1 mg/dL). Blood cultures were positive for *Escherichia coli* and group D enterococcus.

The patient was treated with intravenous fluids and antibiotics (cephalothin and doxycycline), which produced some improvement in her condition. At surgery, a large choledochal cyst containing about 1.5 pounds of serous inspissated bile and numerous stones was found. The choledochocystduodenostomy was stenosed. Extraction of the stones and the inspissated bile, as well as dilation of the cystduodenostomy, were done manually. A long arm T-tube, with the distal end going into the duodenum through the anastomosis, was left in place. A biopsy from the cyst wall showed acute inflammatory tissue and aggregates of dysplastic epithelial cells.

In 1978 and 1979, the patient had repeated attacks of cholangitis, and in 1981 she was admitted to Our Lady of Mercy Medical Center with pain in the right upper quadrant, fever, and chills. Physical examination did not reveal icterus or an abdominal mass. Liver chemistry studies and blood counts were within normal limits. At surgery, a large choledochal cyst measuring 8 × 5 × 6 cm, starting at the upper end of the common hepatic duct and ending at the retropancreatic portion of the duct, was found. The cyst contained a large amount of inspissated bile and multiple small stones. Excision of the cyst and an end-to-end Roux-en-Y hepaticojejunostomy was done. Histopathologic examination of the cyst wall revealed marked infiltration with chronic inflammatory cells and hyperplastic mucosa showing papillary changes. The patient's postoperative recovery was uneventful, and she has remained asymptomatic since that time.

DISCUSSION

Choledochal cyst, first described by Vatero[1] in 1723, is seen more frequently in people from the Orient. In 1975, Flanigan[2] reported that there were 955 cysts recorded in the literature. In 1980, Yamaguchi[3] reviewed 1,433 cases in the Japanese literature, and in 1981, Powell et al[4] updated Flanigan's series by adding 382 more patients—making a total of 1,337 patients with choledochal cysts in the English language literature.

Associated cystolithiasis was described in only 19 cases (2%) in Flanigan's[2] review. In the Japanese literature, Yamaguchi[3] reported 111 (8%) cases with stones, mostly cystolithiasis, in 1,433 patients. Of significance is the fact that in 37 cases, cystolithiasis was seen following surgical treatment with internal drainage. Cystolithiasis has been considered a contributing factor in the development of carcinoma of the cyst wall.[5] Evidence suggests that stasis and repeated episodes of cholangitis, with or without stone formation, may induce malignant changes in the cyst. Carcinomas arising in choledochal cysts are most often seen in younger individuals, are difficult to diagnose prior to surgery, and, as a result, are associated with a poor prognosis.[6]

The present case highlights many of the controversies pertaining to management of choledochal cysts. Conservative management and/or external drainage alone are both associated with a high mortality—97% and 65%, respectively[7]—from biliary cirrhosis, suppurative cholangitis, liver abscess, spontaneous rupture of the cyst, pancreatitis, gastrointestinal hemorrhage, portal hypertension, portal vein thrombosis, and cancer arising from the cyst.[2] These approaches to managing the disorder are, therefore, no longer practiced.

Internal drainage of choledochal cysts by choledochocystjejunostomy or choledochocystduodenostomy has been the treatment of choice in the United States. In a survey based on replies from 44 pediatric surgeons in the United States, Kim[8] stated that an internal drainage operation was done in 83% of patients. While choledochocystduodenostomy was preferred in earlier years, Roux-en-Y choledochocystjejunostomy is now more common.

Several authors[3,9-12] have recommended excision of the cyst as the treatment of choice. The proponents of internal drainage have shunned cyst excision based on technical hazards and the high mortality rate associated with this procedure. However, new methods of preoperative diagnosis, refinement of surgical techniques, and improved postoperative care have decreased the mortality rate from 51% in 1943[13] to 23% in 1956,[7] 12% in 1959,[14] and 1.6% in 1980.[4] In his collective review in 1975, Flanigan[2] reported a 7% mortality rate with excision of cyst and 17% with Roux-en-Y choledochocystjejunostomy. Similarly, Powell et al,[4] in their analysis of 382 patients with choledochal cysts treated during the five-year period 1975–1980, reported a mortality rate of 1.6% in 61 patients who underwent primary excision, as compared to 12% in 91 patients who had internal drainage.

Internal drainage, as compared to excision, results in no obvious decrease in the mortality rate associated with surgery, but leads to frequent postoperative ascending cholangitis with anastomotic stricture. This is clearly demonstrated in the patient described here. Flanigan[2] reported a 50% morbidity rate (defined as the incidence of one or more of the findings of recurrent pain, jaundice, cholangitis, or stricture) following internal drainage operations versus 8% after excision. Similarly, 47 of 158 patients (29.7%) who underwent internal drainage required a second operation, as compared to no patients out of 83 who underwent excision of cyst.[5] Powell et al[4] have reported a complication rate of 10% and a multiple operation rate of 22% following internal drainage in 91 patients, versus a 1.6% incidence of complication and a second operation following excision. In contrast to the large number of cases of cystolithiasis reported following internal drainage, there is only one such case subsequent to cyst excision.[5] Of the internal drainage procedures, complication and multiple operation rates were least common with Roux-en-Y choledochocystjejunostomy.[2-4] Conservation of the fibrous wall of the cyst with incomplete mucosal lining and areas of ulceration may be responsible for the strictures, stasis, stone formation, and recurrent infections after internal drainage.

The experience with this patient and those reported in the literature suggests that cyst excision is now the favored primary method of treatment. Roux-en-Y choledochocystjejunostomy, if done when cyst excision is considered unsafe, requires a 40 cm defunctionalized limb with a large anastomosis (greater than 5 cm) at the most dependent part of the cyst. Biopsy of the cyst wall should always be done to rule out the presence of carcinoma in the choledochal cyst. The gallbladder should be removed at the time of initial operation, whether this be for excision or drainage. In patients who require a second operation after a previous internal drainage procedure for complications, cyst excision is the definitive treatment. This can be performed safely without increased mortality or complications. Such an approach will decrease the long-term morbidity due to recurrent cholangitis and may also decrease the disastrous late sequelae of bile duct carcinoma.

REFERENCES

1. Vatero A: Dissertation in Auguralis Medica Proes. Diss qua scirrhis viscerum disseret c.s. exlerus. 1723; 70:19 (University Library, Edinburg).
2. Flanigan PD: Biliary cysts. *Ann Surg* 1975; 182:635-643.
3. Yamaguchi M: Congenital choledochal cyst. Analysis of 1,433 patients in the Japanese literature. *Am J Surg* 1980; 140:653-657.
4. Powell CS, Sawyers JL, Reynolds VH: Management of adult choledochal cysts. *Ann Surg* 1981; 193:666-676.
5. Flanigan PD: Biliary carcinoma associated with biliary cysts. *Cancer* 1977; 40:880-883.
6. Todani T, Tabuchi K, Watanabe Y, et al: Carcinoma arising in the wall of congenital bile duct cysts. *Cancer* 1979; 44:1134-1141.
7. Tsardakas EN, Robnett AH: Congenital cystic dilatation of common bile duct. Report of 3 cases, analysis of 57 cases, and review of literature. *Arch Surg* 1956; 72:311-327.
8. Kim SH: Choledochal cyst: Survey by the surgical section of the American Academy of Pediatrics. *J Pediatr Surg* 1981; 16:402-407.
9. Lilly JR: Total excision of choledochal cyst. *Surg Gynecol Obstet* 1978; 146:254-256.
10. Kasai M, Asakura Y, Taira Y: Surgical treatment of choledochal cyst. *Ann Surg* 1970; 172:844-851.
11. Saito S, Ishida M: Congenital choledochal cyst (cystic dilatation of the common bile duct). *Prog Pediatr Surg* 1974; 6:63-90.
12. Todani T, Watanabe Y, Mizuguchi T: Hepaticoduodenostomy at the hepatic hilum after excision of choledochal cyst. *Am J Surg* 1981; 142:584-587.
13. Shallow TA, Eger SA, Wagner FB: Congenital cystic dilatation of the common bile duct. *Ann Surg* 1943; 117:355-386.
14. Alonso-Lej F, Rever WB Jr, Pessagno DJ: Congenital choledochal cyst, with a report of 2, and an analysis of 94 cases. *Intern Abst Surg* 1959; 108:1-30.

FROM THE LIBRARY

FOREIGN BODY IN A BRONCHIAL TUBE—RECOVERY

C. McN., aged 23, born in Ireland, presents a family history free from tendencies to pulmonary disease, and a previous personal history blemished only by an occasional attack of tonsillitis.

On June 6, 1898, while holding a common bone collar-button in his mouth he indulged in a hearty laugh. The deep inspiration drew the button from his mouth into his larynx. He was immediately seized with a choking sensation—a feeling of suffocation—which, with spasmodic cough, persisted for more than five minutes. Emetics were at once administered, and though successful in emptying the stomach, brought forth no button. For the ensuing three days the feeling of an obstruction to the ingress of air increased in severity and he entered the hospital.

The clinical history of his stay there has not been obtainable, but the patient reports that he remained twenty-nine days, twenty-one of which he spent in bed; that he was suffering from an attack of pneumonia, and at the same time an acute specific urethritis; that his temperature was as high as 106° F., and that no attempts at laryngeal examination nor operation were made. At the time of his discharge he suffered from cough, profuse fetid, muco-purulent expectoration and dyspnea. His after-treatment was continued under varying physicians until January, 1900, when he came under my observation. . .

On examination with the X-ray, the consolidated portion of the left lung was revealed, but neither at this nor at subsequent examinations with the fluoroscope and radiographs was there seen any trace of a foreign body. Although examined many times, no tubercle bacilli were ever found in the sputum.

Under treatment there was immediate and continuous improvement. Fever disappeared, night sweats and vomiting ceased, digestive functions returned to normal. The cough grew less, but has never disappeared; the sputum lost its purulent character and fetid odor, and diminished in amount. The weight steadily increased, on May 21, 1901, sixteen months after his first visit, being 126 pounds, and on October 4, 1901, 146 pounds, which he has since held.

On August 8, 1902, during a mild spell of coughing, the patient expectorated the button, which, save a slight staining, was unchanged. Since its expulsion the physical signs have undergone but slight modification, the entire upper and part of the lower lobes of the left lung presenting a chronic fibrosis, which has undergone some contraction. For a small area posteriorly—the sixth to eighth ribs—the breathing sounds have become louder in intensity. The left half of the chest is, in circumference, one and one-half inches in antero-posterior diameter, one inch smaller than the right. The heart is drawn to the left, the impulse at the apex being in the fifth interspace, just outside the nipple. There is still considerable cough and expectoration, but the patient has retained a good weight—146 pounds, for 5 feet 7 inches—is earning his livelihood and feels well.

THOMAS A. MCGOLDRICK, MD
(*NY State J Med* 1903; 3:389)

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication. All letters are personally acknowledged by the Editor.*

Cigarette advertising

TO THE EDITOR: Your September 1986 issue of the *Journal,* "Cigarette Smoking, Tobacco, and Health," was both excellent and timely. Kozák,[1] Wörgötter et al,[2] Hastings et al,[3] and Taylor[4] conclusively showed that European physicians and legislators have been able to bring about a diminution in cigarette smoking. In the United States, with the exception of television, we have not been able to ban smoking commercials. Many of the advertisements are directed at teenage smokers.

Members of the American Medical Association and the Medical Society of the State of New York, working in conjunction with other organizations, can achieve the same results as have been achieved in Europe. This will require a continuation of efforts to bring about legislative action at both the national and state levels. Meanwhile, a voluntary refusal on the part of the media to accept smoking commercials would go a long way. Hopefully we will have a smokeless society in America by the year 2000, and an "America, free of the commonest cancer," by the year 2020. We would all much rather see cancer being prevented than treated.

MUHAMMAD ABU HENA, MD
St Peter's Hospital
632 New Scotland Ave
Albany, NY 12208

---

1. Kozák JT: Smoking and health in Czechoslovakia. *NY State J Med* 1986; 86:468-471.
2. Wörgötter GF, Kunze M: Cigarette prices and cigarette consumption in Austria, 1955-1985. *NY State J Med* 1986; 86:478-479.
3. Hastings GB, Leathar DS: Antismoking publicity in Scotland: A decade of progress. *NY State J Med* 1986; 86:480-484.
4. Taylor P: The British Medical Association's campaign against the tobacco industry. *NY State J Med* 1986; 86:490-492.

Medical liability premiums in Great Britain

TO THE EDITOR: It may be of some solace to physicians on this side of the Atlantic that our colleagues in Great Britain, a model of socialized medicine, are now suffering from similar trends in medical liability insurance. On August 19, 1986, *The Times* reported that 80,000 physicians and dentists belonging to the Medical Defence Union were levied increases in medical defense subscriptions of up to 70%.[1] In a document released to its members, the union stated that the highest sums awarded in medical negligence cases had risen from £132,970 in 1977 to £679,264 in 1986. Rates for physicians are to go up in January 1987 to £576 from the £336 formerly paid annually.[1] Mr Bernard Hargrove, a leading barrister, observed that lawyers seem to think that there is a bottomless bucket of insurance money available. He noted that in 1970 a brain-damaged child would receive between £20,000 and £40,000 in compensation, but that awards of between £300,000 and £650,000 are now common.[1]

The end result is that physicians in Great Britain are facing large increases in their annual premiums because of the rapid rise in the number of negligence claims and the larger compensation awards being made to patients. *The Times* reported that "doctors and dentists were 'bewildered' by the rising tide of damages awarded against their colleagues."[1] Clearly the legal profession on both sides of the Atlantic is operating under the false assumption that malpractice awards are limitless.

J. RICHARD TYNER, MD
4 Coulter Rd
Clifton Springs, NY 14432

---

1. Prentice T: High rise in GPs' defence premiums. *The Times,* August 19, 1986, p 3.

Primary retroperitoneal hydatid cyst

TO THE EDITOR: Primary retroperitoneal cysts, located in the retroperitoneal fatty tissue, are not associated with any adult anatomic structure. These cysts are usually congenital in nature, and they arise from urogenital or mesocolic remnants, cell inclusions (dermoids), or lymphatic elements.[1] Hydatid disease represents the only acquired form of cyst, and the most uncommon cause of cyst in the retroperitoneum. To our knowledge, only 14 such cases have been recorded in the English literature. We describe here another case of retroperitoneal hydatid cyst.

Case Report. A 16-year-old girl complained of a painless abdominal mass that had been slowly growing for one year. There were no bowel, urinary, constitutional, or menstrual symptoms. She lived in a mountainous, sheep raising area of North India, where contact with sheep, dogs, and other farm animals was common.

On physical examination, the only finding was a nontender, well defined, and fixed mass measuring about 12 × 8 cm and occupying the left lumbar and umbilical regions. The mass was firm, smooth, and nonballotable. A systemic examination was noncontributory. The hemoglobin level was 11.3 g/dL, and the white blood cell count was 6,400/mm^3 with 3% eosinophils. Plain radiographs of the chest and abdomen were normal. Intravenous urography revealed normal structure and functioning of the kidneys and medial displacement of the left ureter (Fig 1). An ultrasound examination identified a cystic mass lying below but free from the left kidney. The liver and the opposite kidney appeared normal.

With a preoperative diagnosis of retroperitoneal cyst, a laparotomy was done through a left paramedian incision, following colonic preparation. At laparotomy, the mass was confirmed to be a tense retroperitoneal cyst, pushing the left ureter and descending colon forward and burrowing below, in between the two leaves of the sigmoid mesocolon. The inferior mesenteric artery and its branches were stretched over the cyst. After mobilizing the descending colon and safeguarding the left ureter, the cyst was freed on all sides except the posteromedial region, where the cyst wall adhered to the left iliac vessels. After isolation of the cyst with packs, needle aspiration was performed, which revealed clear fluid. This finding raised the suspicion of hydatid cyst. The cyst contents were replaced with hydrogen peroxide after the evacuation of about 1,500 mL of fluid. The cyst was then opened and sucked dry. It was lined by a laminated membrane which was completely removed.

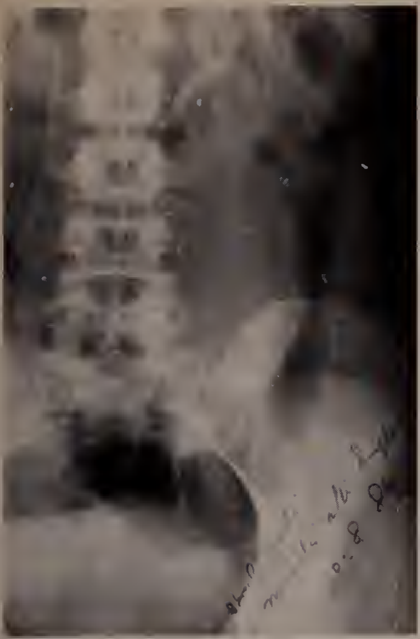


FIGURE 1. Intravenous urogram showing medial displacement of the left ureter, and a gas column in the descending and sigmoid colon.

As much as possible of the pericystic tissue was also removed. The area was then washed with saline solution, and the structure of the region was reconstituted after keeping a drain in the retroperitoneal space.

The patient's postoperative course was uneventful. Microscopic examination showed an outer, laminated hyaline membrane and an inner granular layer with brood capsules, confirming the diagnosis of *Echinococcus granulosus*. The patient was given mebendazole, 40 mg/kg/day for six weeks. She was symptomless 16 months after surgery.

Discussion. Nearly 15–20% of the hexacanth embryos of *Echinococcus granulosus* succeed in passing the hepatic and pulmonary barrier to reach the systemic circulation[2] and lodge in the spleen, kidney, myocardium, bones, and other organs. The chances of implantation directly depend on the vascularity of the organ. The retroperitoneum is an uncommon site of involvement, and in a report of 418 cases from an endemic area,[3] not a single case involved a cyst found in the retroperitoneum. Besides the systemic circulation, other possible modes of spread of hydatid embryos to the retroperitoneum include retrograde lymphatic flow from the gastrointestinal tract or via the portal system.

Handfield-Jones,[1] referring to a specimen in the Royal College of Surgeons museum in London, was the first to mention that hydatid cysts can occur in the retroperitoneal space. The first clinical report of such a cyst was published in 1958.[4] Mukerjee et al[5] reported one case and found only eight other instances in the literature. Five more cases have appeared since that review.[6-8] All the reported cases have occurred in adults, and a preoperative diagnosis was not made unless a sinusal discharge was present. The young age of the patient reported here is unusual.

Two of the nine patients reported prior to 1973 died of anaphylaxis during surgery.[5] Accidental puncture of an unsuspected abdominal hydatid cyst sometimes occurs. Ultrasound examination may help ensure a correct preoperative diagnosis and should always precede needle puncture of an obscure abdominal mass. The anesthetist and the surgeon must be prepared for the possibility of hydatid disease when dealing with any retroperitoneal cyst. As much pericystic tissue as possible should be excised to reduce the chances of recurrence due to the presence of free daughter cysts in the fibrous wall.

AJAY K. KRIPLANI, MS
M-67 Anasagar Link Rd
Near Mahboob Ki Kothi
Ajmer 305 001
India

1. Handfield-Jones RM: Retroperitoneal cysts; Their pathology, diagnosis and treatment. *Br J Surg* 1924; 12:119–134.
2. Rakower J, Milwidsky H: Primary mediastinal echinococcosis. *Am J Med* 1960; 29:73–83.
3. Talib H: Some surgical aspects of hydatid disease in Iraq. *Br J Surg* 1968; 55:576–585.
4. Lockhart J, Sapriza JC: *Excerpta Med* (*Surgery*) 1958:4513.
5. Mukerjee S, Nigam M, Saraf JC: Primary retroperitoneal hydatid cyst. *Br J Surg* 1973; 60:916–918.
6. Chatterjee H, Colaco RJ, Shanmugam M, et al: Hydatid cyst in rare sites: Kidney, retroperitoneum and thigh. *Ind J Med Sci* 1973; 27:20–23.
7. Emami HMD, Asadian A: Unusual presentation of hydatid disease. *Am J Surg* 1976; 132:403–406.
8. Sekar N, Madhavan KK, Yadav RSV, et al: Primary retroperitoneal hydatid cyst (a report of 3 cases and review of literature). *J Postgrad Med* 1982; 28:112–114B.

The Healing Arts in Early America

By Robert I. Goler. 56 pp, illustrated. New York, Fraunces Tavern Museum, 1985. $5.00 (softcover)

Seventeenth and 18th century medicine in America generally conjures up images of bloodletting, women dying in childbirth, typhoid fever, malaria, and moral interpretations of the causes of illness and death. As the author cogently observes, "... the treatment of disease in early America changed very little from the time of European settlement in the early seventeenth century until the establishment of the American republic at the end of the eighteenth century." The absence of a hierarchy of healers such as existed in Europe and of formal educational systems for the training of physicians created a social condition in which healing was undertaken by a wide variety of people. These ranged from Puritan clergymen who prescribed prayer and repentance for everything from typhoid to dystocia, to housewives who used herbal remedies and what was considered common sense to treat most illnesses.

Medicine did not remain static during these early years. Colonists incorporated Indian remedies into their armamentarium, specialization evolved among healers, remedy books were published, and with urbanization new medical institutions were established, among them hospitals and medical schools. Medicine as a profession was slow to evolve as such, crawling up out of a world of nostrums, superstition, untrained healers, charlatans, and an unhygenic environment. Toward the end of the 18th century serious attempts were made at the licensing and regulation of the profession and public health efforts were aimed at dealing with the problems of hygiene.

Documenting this long prescientific era in the American colonies and the new republic is no easy task. *The Healing Arts in Early America* attempts to do this through an analysis of artifacts, documents, and visual materials representative of this period. Rendering this task difficult is the fact that medical care was intensely integrated into everyday life, artifactual reflections of which are now rare. What does survive in terms of artifacts, documents, and visual materials such as sketches, drawings, and paintings was often associated with renowned medical practitioners who represented a departure of excellence from the not-so-excellent norm. Mr Goler is correct when he says that "... much of the understanding of early American disease and treatment must be drawn from printed and manuscript sources, from European materials or from the scattered, and perhaps not representative surviving artifacts."

The Healing Arts in Early America was published in part to serve as a catalog for an exhibition of the same name held at Fraunces Tavern Museum in New York City from December 6, 1985, through June 20, 1986. The author is the Curator of Collections at the museum and assembled a remarkable collection of artifacts, prints, paintings, published and written documents, and other items from over 60 institutions and individuals. A gift from the Metropolitan Life Foundation made publication of this book possible.

A foreword by Christine M. Miles, Director of Fraunces Tavern Museum, puts the publication and the exhibit into perspective. An introduction covers the period under consideration in great detail, the author providing insights into the larger social canvas against which healing was practiced. The introduction also sets the stage in terms of time, place, and persons and candidly reminds the reader of the limitations of surviving artifacts in providing a full comprehension of medicine during this era. As Goler notes, "Relatively few visual records of healing in eighteenth century America have survived. Paintings of doctors are rare, in part, because doctors did not have the same social or financial status in the eighteenth century as they command today and were less likely to commission portraits." Those paintings of physicians that do survive are frequently formal portraits that give little or no sense of the medical profession.

Goler traces the development of 18th century medicine in America, accurately noting that health related utensils such as bedpans, feeding vessels, and medicine chests were usually maintained in people's homes. Prints and portraits almost always depicted medical treatments in domestic settings. Toward the end of the century, medicine began to emerge as a profession, but it had a long road to tread before it distanced itself from its home-remedy origins.

The book is divided into 13 chapters followed by a very useful bibliography. These chapters consist of a brief introductory essay which precedes a detailed description of objects exhibited under the chapter heading. Social, historical, and medical details are provided for most of the objects, which enable the reader to understand not only use but larger issues as well. In the first chapter, "Medicine in 17th Century America," the author notes the dominant place of dysentery, scurvy, typhoid, influenza, malaria, syphilis, and accidents among the early colonists populating the wilderness. The colonists practiced what they knew, which early on represented the medical beliefs and practices they had carried across the ocean from Europe. Belief in the four humors was widespread, and medicine—known then as "chirurgery" or "physick"—was learned through apprenticeship. In New England, ministers often provided medical advice because of the belief in the linkage of disease and misfortune to the devil and sinful deeds. However, most healing was done at home, and in time the colonists added local flora to the medicinal plants they had brought from Europe. Among the former were sassafras, snakeroot, and pepperwort.

The second chapter, "Native Americans," although brief, explores the medical practices of the Indians, which were intertwined with religious beliefs. The colonists, impressed by the apparent health of the Indians, adopted native remedies while discarding the spiritual systems with which they were associated. Tobacco was a plant to which numerous medicinal properties were attributed. In Europe, as the author notes, a vast literature appeared describing these. The author also describes the sad fate of the eastern coast Native American populations that were decimated by imported diseases such as tuberculosis, measles, smallpox, and venereal diseases.

In the third chapter, "Medicine in the 18th Century," the author traces the evolution of medicine, including the appearance of male midwives and dentists, the development of herbal gardens, and the continued belief in the humoral theory of disease and treatment. He underscores the existence then of patients' complaints of poor treatment and mismanagement. Medical societies developed, serving as a forum for regular meetings, and in Philadelphia and New York the first medical schools appeared. Quarantine hospitals were built, as were hospitals for the poor and the insane.

In the fourth chapter, "Training and Practice," Goler provides a succinct account of the status of medical training which for most of the 17th century was virtually nonexistent. Alexander Hamilton noted that in the early part of the century, most doctors were "empyrick's, having no knowledge or learning but what they have acquired by bare experience." William Smith in his *History of the Province of New York* (1757) was more to the point: "Few Physicians amongst us are eminent for their skill. Quacks abound like locusts in Egypt . . . Any man at his pleasure sets up for Physician, Apothecary and Chirurgen." Amputations and trephining were performed by practitioners with little skill. Instruments for all manner of procedures abounded, including needles, sutures, clips, forceps, tourniquets, probes, scalpels, catheters, tooth extractors, and bloodletting devices, and these were often incompetently used. As Goler notes, most of these instruments and devices were imported from Europe, but occasionally local cutlers fashioned truly American designs. A few Americans went to London, Edinburgh, Paris, or Leyden for clinical training. Goler accurately observes that they were reluctant to adopt the practice of combining the skills of physician, surgeon, and apothecary when they returned and complained about having to compete with apprentice-trained practitioners. However, they set down the beginnings of a standard for medical education that would take more than a century to flower.

The fifth chapter, "Drugs and Apothecaries," explains the meshing of the separate disciplines of medicine and pharmacy. Apothecaries learned their trade through apprenticeship and often served as physicians as well. There were many who were unhappy about people being both apothecaries and physicians at once, including Thomas Twining, who in his diary of 1795 said: "In a country where a gentleman's coachman is obliged to be his own smith, it is not surprising that a surgeon should be an apothecary." Goler draws particular attention to the concern over fees and the quality of medications, issues that were first addressed by legislation passed in 1736 by the Virginia legislature. However, the concerns over the existence of these practitioners derived not so much from the fees they charged as from the empirical observation that they seemed to be jacks of all trades and masters of none. Except for a rare few, these individuals were not held in high esteem, partially because their success rates were low. Yet even well trained physicians were often unable to do much given the rudimentary level of medical knowledge and the paucity of effective therapeutic interventions. Clearly, though, a physician was perceived as being more competent than the apothecary-surgeon-physician, particularly if he had studied at one of the leading centers in Europe.

The practice of bloodletting was widespread in America up through the 19th century. Goler devotes his sixth chapter to a discussion of the practice, recalling the monopoly of it once held by English barber-surgeons. The red-and-white-striped barber poles are a residuary symbol of the barber's past association with bloodletting. The art required skill and mastery of the use of an array of instruments and devices. Blood was drawn from veins and the skin using scarificators, lancets, and fleams. The blood was collected in cups, by leeches, or in a piece of cloth wrapped around the site. Shaving bowls were often used to collect blood, which was frequently measured. Goler relates that the maintenance of bloodletting instruments was of serious concern to physicians, as evidenced by the numerous advertisements in newspapers by cutlers and smiths offering to sharpen them. Leeching was closely integrated with bloodletting, leeches being guided to the site by being placed on the edge of a wineglass which was then inverted over the area. Dry cupping and wet cupping in which scarification was first performed were also associated with the practice of bloodletting. Dry cupping was still performed in the United States into the 20th century and is still widely practiced, as is wet cupping, in many areas of the developing world.

Epidemic diseases such as smallpox, diphtheria, yellow fever, and many more were common in colonial America. It was during the Boston smallpox epidemic of 1721 that Cotton Mather introduced the practice of variolation, having learned of it from his African slave Onesimus. The practice spread rapidly throughout the colonies, but remained controversial. It was still being practiced in areas of sub-Sahara Africa in the 1960s, before smallpox was eradicated. A variety of techniques were used and, as Goler notes in his seventh chapter, "Epidemics," colonial Americans coated a thread with pus from a pustule, attached it to a sewing needle, and then ran it under the skin.

Late in the 18th century, following the discoveries of Edward Jenner (1749–1823), vaccination was introduced. In America, Benjamin Waterhouse championed vaccination over variolation, or inoculation, as it was frequently called. Smallpox was among the most dreaded epidemic diseases not only because it carried a high mortality but also because it sometimes left people with unsightly scars. What is often forgotten is that most people who recovered from smallpox were left with few or no permanent scars.

Home remedies abounded in colonial America and the author appropriately discusses this subject in considerable detail. The American colonists were an enterprising lot and even designed wheelchairs. Those people who reached adulthood could generally expect to live to their sixties, when old age was thought to begin. However, medicine was able to offer little help for the degenerative diseases of aging.

Childbearing was an event fraught with many dangers in colonial America. Midwifery was the almost exclusive domain of women, with male physicians being called in for complications. In his chapter on this subject, the author provides a clear discussion of obstetrical instruments and of the diaries of Martha Moore Ballard (1735–1812), a midwife who detailed her management of 996 births, and Dr Samuel Adams (1744–1819), who had acquired skills in rupturing the membranes and turning breech presentations.

Although dentistry was quite primitive, it was nonetheless practiced. There were no dental schools in the United States until the 19th century and those who practiced the skill were frequently recent immigrants from Europe. Some were highly skilled, such as Paul Baker, an American who trained in Europe and who trained several Americans, including Paul Revere. In his tenth chapter, "Dentistry," Goler cites Robert Wooffendale, an English dentist who arrived in America in 1766 and advertised his skill as "all operations on the teeth, gums and sockets; likewise in artificial teeth, so as to escape discernment." Pulling teeth was usually the only treatment known, which in turn created a demand for dentures. However, there were dentists such as John Greenwood (1760–1819), who introduced the foot drill and who also tried to transplant live teeth. He is best known for the set of dentures he made for George Washington. George Washington, as Goler notes, was troubled by his teeth in later years. A lower denture made of ivory to which eight human teeth are pinned, now in the collection of the New York Academy of Medicine, was made for Washington just after his inauguration. Goler relates that during the next ten years, however, Washington's dentures were repaired or replaced at least eight more times.

The late 18th century also witnessed the introduction of tooth picks, tongue scrapers, and dentifrices. Clearly, early Americans were concerned about their teeth and gums but could do little to prevent caries or periodontal disease.

In his 11th chapter, Goler covers medicine and the American Revolution. There were then 3,500 physicians in the colonies, of whom only 200 possessed a medical degree. The estimated 10,000 casualties among patriot soldiers per year of the war were largely due to "camp diseases" such as malaria, dysentery, typhoid, and other communicable diseases. Local decentralized hospitals were set up, following the British practice, but many soldiers preferred to return home rather than stay in them. Surgical skills were in demand and the reputations of certain surgeons such as Dr William Burnet (1730–1791) of New Jersey and Dr John Jones (1729–1791) of New York spread. Jones wrote a widely circulated book for military surgeons called *Plain Concise Practical Remarks on the Treatment of Wounds and Fractures*, which was published in Philadelphia in 1776.

Goler devotes one of his last chapters to electrical treatments, which became widespread in America late in the 18th century. This development paralleled the general interest in electricity stimulated by the studies of Benjamin Franklin and of Luigi Galvani of Italy, who found that muscle tissue responded to electricity.

The last chapter of this book is appropriately called "Institutions, Societies and Waterworks." As the country grew and society became increasingly complex and urbanized, Americans became aware of the need to improve hygiene, to care for the indigent and mentally ill, and to raise the standards of training for physicians. The contamination of wells by wastes from tanneries, glue factories, and general sewage spurred municipalities to improve water and sanitary systems. Health care concerns had now broadened beyond those of the individual patient to those of the public at large.

The Healing Arts in Early America eminently succeeds in clearly describing the development of medical care from the time of British settlement in North America until the early years of the republic. The author has drawn upon an impressive wealth of historical evidence in compiling this book and has presented it in a clear and easy to read style. These were not easy times for Americans, faced as they were with massive epidemic disease problems and meager scientific knowledge and skills. However, they openly debated the directions of medical care, initiated licensing and regulation of professionals, established hospitals and medical schools, and addressed public health concerns. In so doing they emerged from the colonial era with the beginnings of a strong medical care tradition.

PASCAL JAMES IMPERATO, MD
Editor

Professor and Chairman
Department of Preventive Medicine
and Community Health
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

CLINICAL COMPUTED TOMOGRAPHY. ILLUSTRATED PROCEDURAL GUIDE

By Lee C. Chiu, MD, James D. Lipcamon, RTR, Victoria S. Yiu-Chiu, MD. 261 pp, illustrated. Rockville, Md, Aspen Publications, Inc, 1986. $37.50 (hardcover)

The importance of computed tomography (CT) in diagnostic imaging continues to increase because of two major factors. First, the progressive development of CT techniques and technology has resulted in newer applications to such body regions as the spine, the soft tissues of the neck, and the extremities. Second, CT scanners are becoming more universally available to smaller hospitals because of lower costs per unit and the sharing of mobile CT units.

This book is organized into chapters discussing the following: principles of instrumentation for CT; the technologist's role; contrast media reactions; applications to specific body region with relevant serial cross-sectional anatomy sections; CT pathology of the head and body; and CT interventional techniques.

The material is presented in a straightforward style which presupposes no specialized knowledge of the subject. Thus a very broad field is covered in an all-inclusive but sketchy manner. The simplified standard scanning protocols as applied to each body region are the most useful information. Also quite useful are the high quality CT sectional images with accompanying clearly labeled line drawings. These serve as a good quality cross-sectional atlas of anatomy.

The section on CT pathology consists of a potpourri of CT pathology with many examples. These scattered examples, however, do not in any way constitute a comprehensive text.

Conspicuously absent from the book are descriptions of many important and fundamental subjects, such as the system of CT numbers (the so-called "Hounsfield Units") and their significance, CT artifacts, and the analytic process of CT interpretation, including its pitfalls and limitations. Another serious deficiency of this book is the rather superficial manner in which it treats such important subjects as contrast media reactions, which are accorded only two pages of text. And although there is much text devoted to the subject of contrast enhancement injection technique, the reader is given no guidance as to how one uses this information in the practical scanning situation. However, an adequate list of references at the end of the book somewhat compensates for these deficiencies.

Occasional statements are made that are at variance with current opinion and practice. For example, the statement is made that water soluble contrast agents for gastrointestinal opacification as preparation for abdominal CT exams are "an invaluable alternative to barium sulfate solutions [*sic*, suspensions], especially in cases such as gastrointestinal perforation where barium sulfate cannot be used." This violates the basic surgical precept that nothing should be given per os or per rectum in such cases.

Although this book purports to be an introductory text for CT, it is too sketchy and does not include enough information about the fundamentals to serve adequately for this purpose. It would not have taken too many more pages of text to cover the vital points of CT theory, technique, and interpretation and to provide an adequate overview of the subject. Nonetheless, those initiating a CT service will find the straightforward scanning protocols and the cross-sectional anatomic atlas quite useful. This material is not readily available from any other single source.

LAWRENCE J. ROSENBLUM, MD
Chenango Memorial Hospital
Norwich, NY 13815

THE COMPUTER IN RADIOLOGY

Edited by Tim B. Hunter, MD. 348 pp, illustrated. Rockville, Md, Aspen Systems Corp, 1986. $39.95 (hardcover)

This book is intended as a reference work for practicing radiologists and as a textbook for radiology residents. It attempts to deal with a vast area of computer applications in radiology in a little over 300 pages of text, a most difficult task even in expert hands. The authors have been drawn mainly from the radiology staff of the University of Arizona. Their experience with the implementation of a totally computerized electronic radiology department has served them well in the production of this book.

The book is divided into five main parts. Parts one and two cover the history of computers and explain and describe the computer hardware and software currently available. Part three covers the clinical applications of computers in ultrasound, nuclear medicine, magnetic resonance imaging, etc. Part four covers the concept of data bases and a totally digital department, with the final part covering the personal computer and its applications. There is also a 20-page glossary of computer terms and a list of the major computer vendors.

As with any multiauthored book, the quality and direction of each chapter varies considerably. For a radiologist with only a layman's knowledge of computers, the first two parts provide an excellent background to computers, their languages, and the various peripheral devices that can be attached to them. These two parts can almost be considered as an expanded dictionary of computer terminology that will aid the radiologist in understanding the relevance of many of the technical aspects of computer hardware and software.

Part three of this book is perhaps the weakest. It commences with a chapter on the digital image which provides brief introductions to signal detection theory, histogram equalization, etc, at a level that is not on a par with the succeeding chapters. These chapters deal with computerized tomography, magnetic resonance imaging, digital subtraction angiography, nuclear medicine, and ultrasound. I found a very large variation in the detail and direction of each chapter. For example, the chapter on digital subtraction angiography describes the equipment requirements but fails to describe the processing of images, ie, matched filtration, noise reduction, dual energy subtraction, etc. On the other hand, the chapter on nuclear medicine assumes the reader has little knowledge of the subject, and attempts in 30 pages to describe all of the clinical applications rather than concentrating on the more important computer applications such as SPECT, which is covered in one page.

Part four of the book deals with the concept of a computerized radiology department and with data bases. This section crystallizes a great deal of interesting information in one area on both the theoretical and practical aspects of the implementation of a digital department. This part also contains succinct advice for anyone contemplating the implementation of a departmental data base system.

The book is generally well written with good quality diagrams throughout. The editor intended that each chapter be a free-standing entity, hence there is considerable overlap between different chapters, particularly in the description of computer hardware. I was disappointed with the section on clinical applications, which I thought attempted to cover too much of the basics of the different subspecialties. These basics are readily available in many radiology textbooks. I would have preferred to see a more detailed description of the applications of computers in these areas. While I would be hesitant to recommend this work as a textbook for radiology residents, I think it is an excellent reference book, offering many gems of information on computer facts that are not readily available to the radiologist in either the literature or in standard computer textbooks.

MICHAEL K. O'CONNOR, PhD
Mayo Clinic
Rochester, MN 55905

ANGIOGRAPHIC DIAGNOSIS OF SUPRATENTORIAL CEREBRAL TUMOURS (COMPARED WITH CT SCAN)

By A. Pansini and P. Conti. 280 pp, illustrated. St Louis, Mo, Ishiyaku Euro-America, Inc, 1985. $80.00 (hardcover)

The authors of this book are neurosurgeons from the neurosurgery department of the University of Florence, Italy. A. Pansini is the director and P. Conti, the senior registrar. The authors' intent is to "evaluate the angioarchitectonic physiognomy of different encephalic tumours, and is dedicated to students of neurology and neurosurgery. It contains the fundamental concepts of a brain circulation and the polymorphous aspects of blood circulation during tumoral hemispheric processes."

The CT comparisons are limited almost entirely to the last chapter of the book, which is 22 pages long. The first chapter deals with arterial and venous morphology. The angiographic images are neither subtracted nor done with magnification techniques, and perhaps because of technical limitations lead to erroneous statements such as that the ophthalmic artery "does not appear owing to its thin calibre (1–1.5 mm)." In fact, the ophthalmic artery is seen uniformly and consistently with modern imaging chains. Perhaps for similar reasons the small caliber vasculature of the brain is omitted or not evaluated in detail.

The book's second chapter deals with the angiographic diagnosis of supratentorial tumors by regions. The authors' point of departure for localization is different from the usual orientation of the neuroradiologic literature but is valid in its own right. Chapters 3, 4, and 5 discuss the angiographic features of meningiomas, gliomas, and metastasis. Chapter 6 portrays angiographic aspects of recurrent neoplasms.

It is refreshing, in this time of fascination with neurologic imaging done with high technology in NMR, CT, and ultrasound, to see a reborn interest in cerebral angiography. However, past treatments of cerebral angiography can be found with greater completeness, detail, and technical finesse.

MICHAEL S. TENNER, MD
New York Medical College
Valhalla, NY 10595

PRACTICAL OBSTETRICAL ULTRASOUND

By John W. Seed, MD, and Robert C. Cefalo, MD. 198 pp, illustrated. Rockville, Md, Aspen Publications, 1986. $33.50 (hardcover)

The aim and scope of this book is to provide an introduction to obstetrical ultrasound, and to examine the latest methodologies and clinical applications. The book is written for the novice sonographer as well as for those individuals who would enhance their clinical management of patients by utilizing ultrasound assistance. The authors have included quality state-of-the-art real-time images and, when necessary, accompanying line drawings to explain the images.

The book presents in a logical sequence discussions of normal fetal sonographic anatomy, gestational age assignment, and evaluation of fetal growth and fetal weight. An excellent discussion of the use of ultrasound in assessing fetal growth quality and developing a biophysical profile of the fetus is included. In addition, the authors provide a chapter on fetal malformations in which superb state-of-the-art images are used. A final chapter discusses the intricacies of office-based sonography to enhance the practice of obstetrics and gynecology.

The text is well organized with excellent didactics, and there is a superb bibliography at the end of each chapter. The authors accomplish their goal of familiarizing the novice with the practical aspects of obstetrical ultrasound. However, after reading this book and viewing the excellent illustrations, the neophyte in sonography may not appreciate the more deceptive aspects of obstetrical ultrasound. There is the danger that clinical errors might result from well intentioned but poorly or incompletely performed ultrasound examinations. This book in no way serves as a "teaching machine" for obstetrical ultrasound. It should really serve as a quick reference for common obstetrical problems and not as a primer for learning sonography. It is mandatory that hands-on supervised fellowships in obstetrical sonography be undergone before one attempts to become a "practical obstetrical sonographer."

Overall, this is a well balanced book that will be most useful as a library reference and as a ready source of clear illustrations.

NOEL ROBERT ZUSMER, MD
Mt Sinai Medical Center
of Greater Miami
Miami Beach, FL 33140

APPLETON'S REVIEW FOR FLEX

By Ira Jeffrey Berg, MD. 408 pp, illustrated. East Norwalk, Conn, Appleton-Century-Crofts, 1985. $29.95 (paperback)

This 408-page, clearly printed book is a compendium of 750 sample FLEX questions arranged by subject and subcategorized into ten patient management problems and three small practice tests (which may be submitted to the publisher for computer grading for an additional $7.50 charge). Each set of questions is followed by correct answers and well-written explanations.

The book was written by a 21-member editorial board, and an additional 23 contributors having variable qualifications ranging from clinical instructor to academic professor. The total number of questions in this book approximates the actual number of just one FLEX examination. This would be acceptable if the questions were of a general nature that could instill broad conceptual knowledge and judgment in preparation for the actual examination. Unfortunately, this is not the case.

Most of the multiple choice questions appear to test only simple factual recall with little originality and minimal assessment of decision-making capabilities, perhaps more a commentary on FLEX itself than on this review book specifically. Many of the multiple choice questions appear ambiguous. My impression is that most of these questions were simply confabulated by the contributors and never actually tested on students for reliability.

There are many *K-type* questions ("choose A if 1, 2, and 3 are correct; B if 1 and 3 are correct; etc), that are known to be faulty discriminators between knowledgeable and poor students and are also didactically deficient questions. These dreaded questions are unfortunately a fact of life on standardized examinations and it is hard to condemn the authors for using them, but I believe this book suffers from "hyper-*K*'lemia."

Most of the figures are clear line drawings. Unfortunately, one critical x-ray reproduces so poorly that its major pathologic aspect is undiscernible, and it contains a misleading and most likely unintentional "red herring" that makes its associated question valueless.

There is a plethora of erroneous information, usually related to defective logic, among the patient management problems (PMPs). In the first PMP, for example, while it is admitted that oral herpes may be associated with an encephalitic process, asking the patient about a history of oral herpes is considered an incorrect choice. Portions of the routine physical examination (examination of the thyroid, carotid, and liver size) are discounted as incorrect simply because they are not considered "particularly important" in the diagnosis of the disease at hand. While sending cerebrospinal fluid for culture is scored as optional, it is regarded as frankly incorrect to ask the laboratory for this result later on in the problem! Overall, the quality of the PMPs suggests considerable inexperience among the contributors in writing this difficult type of problem.

The PMPs contain rather sketchy instructions for self-grading: "The total score is the sum of the correct choices and the incorrect options *not* chosen." If the authors provided the total number of choices in each PMP marked as correct (call this C) and the number of choices marked as incorrect (call this I), the user would then add all of his or her correct choices and subtract all of his or her incorrect choices. This raw score is then looked up on a table to convert it to a percentage ranging from 100% for a raw score of C to 0% for a raw score of −I.

Overall, this is yet another list of mediocre questions and answers with much ambiguity and little originality. In defense of the authors, the mediocrity, ambiguity, and lack of originality probably prepare the reader nicely for an examination that is similarly deficient.

RICHARD M. STILLMAN, MD
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

PRACTICAL STRATEGIES IN OUTPATIENT MEDICINE

By Brendan M. Reilly, MD. 832 pp, illustrated. Philadelphia, WB Saunders Co, 1984. $44.50 (hardcover)

It is clear from reading this book that it was written by both a practitioner and a teacher of outpatient internal medicine. It is a clearly written and lavishly illustrated text. Dr Reilly takes the reader through the evolution of carefully thought-out approaches to the most common outpatient complaints. The book is meant to help "medical students and house officers learn the ways of ambulatory care." By his own admission, it is not meant to be a comprehensive textbook. Rather, the book attempts to develop a clear clinical approach to common problems. Implicit is the recognition that many different disease processes, both benign and serious, may be manifested by the same common symptoms and signs. The subject of this book is how to optimize benefit to the patient in a cost efficient and diagnostically productive manner.

In his quest to formalize the thinking process that should go on in the internist's mind when confronted by a medical problem, the author largely succeeds in exposing the reader to a well-thought-out system of approaching the analysis and treatment of the problem. His discussions are well supported by literature research. However, the style of introducing this body of academic knowledge is decidedly interpretative and utilitarian. One gets a strong sense that Dr Reilly has assimilated the literature into his approach; the reader does not come away with the burden of sifting through a barrage of conflicting data in order to come up with a methodology. Controversies are placed into the context of a practitioner who cannot afford to be blinded by the lights of the oncoming car, but who must decide on what to do next. The reader comes to appreciate the seasoned clinician behind the book who has grappled with the problems and who is offering his approach based on both the literature and his years of experience. We are reassured that there is a logical approach to sorting out difficult clinical problems. To this end, Dr Reilly's book is ideally suited for medical students and house officers.

The book deals with common problems. Its contents include the following: low back pain, sore throat, diarrhea, headache, knee pain, female urinary tract infections, vulvovaginal disorders, male genitourinary infections, shoulder pain, dizziness, abdominal pain, cough, and chest pain. There are no detailed analyses of disease processes, nor are there extensive discussions of differential diagnoses and therapies. Instead, the reader is offered excellent and well-illustrated reviews of anatomy, physical diagnosis, and carefully constructed decision trees. Costs to the patient and the health care industry for a variety of problems are analyzed with relation to the potential benefits of diagnostic tests and therapeutic plans. The discussions of the economics of health care are both refreshing and crucial at this juncture in American medicine. The treatment plans are based both on accepted practice and on the author's experience. For example, for the management of low back pain, he offers two chiropractic adjustments that he has found useful.

This eclectic approach to patient care and the thoroughness of Dr Reilly's considerations for the care rendered make *Practical Strategies in Outpatient Medicine* a valuable addition to the literature on ambulatory care internal medicine. With the current emphasis on the outpatient setting and the need to train future physicians to be competent in this important arena of health care delivery, Dr Reilly's book will certainly be a tremendous aid, if not the bible, for outpatient medicine students.

BENJAMIN CHU, MD
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

ANNUAL PROGRESS IN CHILD PSYCHIATRY AND CHILD DEVELOPMENT 1985

Edited by Stella Chess, MD, and Alexander Thomas, MD. 665 pp, illustrated. New York, Brunner/Mazel Publishers, 1986. $40.00 (hardcover)

This annual has appeared since 1968 and has become an accepted reference work for child psychiatrists. Not only are articles covering a wide range of clinical issues presented, but equal attention is given to the child development literature which is the basic science of child psychiatry. The volume is edited by Stella Chess and Alexander Thomas, who are well known for their pioneering study of the role of temperament in normal personality development and psychopathology. This volume at first glance may be seen by the majority of the readers of this journal as the distillation of the ideas of a narrow specialty and hence of little appeal. That conclusion would be unfortunate because of the importance of the ideas presented.

If read according to the following directions, this volume could be of interest to many readers. Unless one is prepared to spend a lot of time with it, it should be taken out of a medical library and sampled over an evening or so. Rather than trying to go through all of the 36 articles, the several pages of introduction to each particular section should be read. These introductions review the state of the art in each area and examine the significance of each article. The sections cover basic themes such as developmental, social, and clinical issues; infancy; and family studies; but changing topics such as maternal employment outside the home and developmental disorders are also considered. Within each topic particular articles can be skimmed by reading their introduction and conclusion. Each is interesting in a narrow sense but almost all have implications for social policy.

The editors take special delight in tangling with cherished and long-held ideas. One example is the conviction that extreme poverty and chaotic family life produce a self-perpetuating underclass, that the children of this underclass are doomed by the sins of their parents, and that money put into this group is unlikely to yield cost-efficient results. Long and Vaillant in their article, "Natural History of Male Psychological Health, XI: Escape From the Underclass," challenge this idea. In a long term prospective study, with controls, they found that a majority did escape and find adequate jobs.

Another sacred idea is that it is harmful to children for their mothers to work outside the home. In the section, "Maternal Employment Outside the Home," the past 25 years of careful research is reviewed and it is shown that children of working mothers are not harmed if satisfactory substitute care is provided. A survey of the attitudes of health professionals, however, has shown their views to be more related to their own personal characteristics. For example, older physicians who have children and whose wives do not work are most likely to have opinions contrary to these research findings.

The section on developmental issues is the heart of this volume, and the article by Clarke and Clarke, "Constancy and Change in the Growth of Human Characteristics," is most important. The idea that is challenged, and this is what Chess and Thomas have been unhappy about for years, has been the primary view in this century. That view holds that early experience is dominant and maintains a continuity. Time has shown that development is a complex interweaving of biological and social factors, chance, and the impact of the individual on his own environment. These continually interacting variables alternate between constancy and change. Such ideas pervade all the articles in this volume and also have an importance far beyond the field of child psychiatry. Such ideas would be the reward of spending time with this volume.

STEPHEN L. BENNETT, MD
Harlem Hospital Center
New York, NY 10037

BOOKS RECEIVED

AIDS

AIDS and the Employer. Guidelines on the Management of AIDS in the Workplace. Edited by Leon J. Warshaw, MD. 86 pp. New York, The New York Business Group on Health, Inc, 1985. $30.00 (softcover)

AIDS. Etiology, Diagnosis, Treatment and Prevention. Edited by Vincent T. DeVita, Jr, MD, Samuel Hellman, MD, and Steven A. Rosenberg, MD. 352 pp, illustrated. Philadelphia, JB Lippincott Co, 1985. $39.75 (hardcover)

ANESTHESIOLOGY

Anesthesia for the Uninterested. Second Edition. By John D. Tolmie, MD, and Alexander A. Birch, MD. 263 pp, illustrated. Rockville, Md, Aspen Publishers, Inc, 1986. $19.95 (paperback)

Anesthesia for Obstetrics. Second Edition. Edited by Sol M. Shnider, MD, and Gershon Levinson, MD. 566 pp, illustrated. Baltimore, Williams & Wilkins, 1987. $65.95 (hardcover)

CARDIOLOGY

Cardiology 1986. Edited by William C. Roberts, MD. 468 pp, illustrated. New York, Yorke Medical Books, 1986. $55.00 (hardcover)

The International Textbook of Cardiology. Edited by Tsung O. Cheng, MD. 1,297 pp, illustrated. New York, Pergamon Press, Inc, 1986. $140.00 (hardcover)

CARDIOVASCULAR SURGERY

Cardiac Surgery. Improving Results with Coronary Artery Bypass Grafting. Edited by Jack G. Copeland, MD, and Steven Goldman, MD. 142 pp, illustrated. Philadelphia, Hanley & Belfus, Inc, 1986. $34.00 (hardcover)

Biologic Bioprosthetic Valves. Proceedings of the Third International Symposium. Edited by Endre Bodnar, MD, and Magdi Yacoub, FRCS. 928 pp, illustrated. New York, Yorke Medical Books, 1986. $125.00 (hardcover)

CHEST SURGERY

Atlas of Chest Wall Reconstruction. By Alan E. Sefer, MD, Goeffrey M. Graeber, MD, and Gary G. Wind, MD. 260 pp, illustrated. Rockville, Md, Aspen Publishers, Inc, 1986. $98.00 (hardcover)

EMERGENCY MEDICINE

Case Studies in Pediatric Emergency Medicine. Edited by Anthony J. Haftel, MD. 309 pp. Rockville, Md, Aspen Publishers, Inc, 1986. $33.50 (hardcover)

GENETICS

The New Genetics and Clinical Practice. Second Edition. By D. J. Weatherall, FRS. 206 pp, illustrated. New York, Oxford University Press, 1985. $15.95 (softcover)

HEALTH CARE

Clinical Practice and Cost Containment. A Physician's Perspective. By Henry A. Shenkin, MD. 144 pp. Westport, Conn, Praeger Publishers, 1986. $39.95 (hardcover)

Physician Assistants. Present and Future. Models of Utilization. Edited by Sarah F. Zarbock, PA-C, Kenneth Harbert, PA-C. 140 pp, illustrated. New York, Praeger Publishers, 1986. $35.00 (hardcover)

The Medicare System of Prospective Payment. Implications for Medical Education and Practice. Edited by Mohan L. Garg, ScD, and Barbara M. Barzansky, PhD. 229 pp. New York, Praeger Publishers, 1986. $38.95 (hardcover)

Cost Containment and DRGs. A Guide to Prospective Payment. By Allen D. Spiegel, PhD, and Florence Kavaler, MD. 565 pp, illustrated. Owings Mills, Md, National Health Publishing, 1986. $49.00 (hardcover)

HISTORY OF MEDICINE

Italian Broadsides Concerning Public Health. Documents from Bologna and Brescia in the Mortimer and Anna Neinken Collection. New York Academy of Medicine. Edited by Saul Jarcho, MD. 421 pp. Mount Kisco, NY, Futura Publishing Co, Inc, 1986. $49.50 (hardcover)

INFECTIOUS DISEASES

Complications of Viral and Mycoplasmal Infections in Rodents to Toxicology Research and Testing. Edited by Thomas E. Hamm, Jr. 191 pp, illustrated. New York, Hemisphere Publishing Corp, 1986. $49.95 (hardcover)

Rabies—The Facts. By Colin Kaplan, G. S. Turner, and D. A. Warrell. 126 pp, illustrated. New York, Oxford University Press, 1986. $13.95 (hardcover)

INTERNAL MEDICINE

The Practicing Physician's Approach to Headache. Edited by Seymour Diamond, MD, and Donald J. Dalessio, MD. 240 pp, illustrated. Baltimore, Williams & Wilkins, 1986. $38.50 (hardcover)

LEGAL MEDICINE

Consent and Confidentiality in the Health Care of Children and Adolescents. A Legal Guide. By James M. Morrissey, Adele D. Hofmann, and Jeffrey C. Thrope. 273 pp. New York, The Free Press, 1986. $30.00 (hardcover)

MEDICAL EDUCATION

Continuing Medical Education. A Primer. Edited by Adrienne B. Rosof and William C. Felch, MD. 213 pp, illustrated. New York, Praeger Publishers, 1986. $29.95 (hardcover)

MEDICAL ETHICS

Modern Medicine and Jewish Ethics. By Fred Rosner, MD. 405 pp. Hoboken, NJ, KTAV Publishing House, Inc, 1986. $22.50 (hardcover)

MISCELLANEOUS

The Road Back to Health. Coping with the Emotional Side of Cancer. By Neil A. Fiore, PhD. 224 pp. New York, Bantam Books, 1984. $4.50 (paperback)

The Secret Strength of Depression. Revised Edition. By Frederic F. Flach, MD. 249 pp. New York, Bantam Books, 1986. $3.95 (paperback)

Taking the World in for Repairs. By Richard Selzer. 239 pp. New York, William Morrow & Co, Inc, 1986. $15.95 (cloth)

Special Care. Medical Decisions at the Beginning of Life. By Fred M. Frohock. 263 pp. Chicago, University of Chicago Press, 1986. $19.95 (hardcover)

Slide Interpretation in Oral Diseases and the Oral Manifestations of Systemic Diseases. By Crispian Scully and Jonathan Shepherd. 156 pp, illustrated. New York, Oxford University Press, 1986. $27.95 (softcover)

Behavioral Counseling in Medicine. Strategies for Modifying At-Risk Behavior. By Michael L. Russell, PhD. 327 pp, illustrated. New York, Oxford University Press, 1986. $29.95 (hardcover)

Personal Money Management for Physicians. Fourth Edition. By Lawrence Farber. 286 pp, illustrated. Oradell, NJ, Medical Economics Books, 1986. $27.95 (hardcover)

Inside Doctoring. Stages and Outcomes in the Professional Development of Physicians. Edited by Robert H. Coombs, PhD, D. Scott May, MD, and Gary W. Small, MD. 299 pp. New York, Praeger Publishers, 1986. $14.95 (softcover)

Three Forms of Sudden Death: and Other Reflections on the Grandeur and Misery of the Body. By F. Gonzalez-Crussi, MD. 207 pp. New York, Harper & Row Publishers, 1986. $15.95 (hardcover)

Webster's Medical Desk Dictionary. Edited by Roger W. Pease, Jr, PhD. 790 pp. Springfield, Mass, Merriam-Webster, Inc, 1986. $18.95 (hardcover)

Saving Face. A Dermatologist's Guide to Maintaining a Healthier and Younger-Looking Face. By Nelson Lee Novick, MD. 258 pp, illustrated. New York, Franklin Watts, Inc, 1986. $16.95 (hardcover)

Surgeon! A Year in the Life of an Inner-City Doctor. By Richard T. Caleel, MD, with John Littell. 280 pp. New York, Rawson Associates, 1986. $15.95 (hardcover)

A Family Affair. Helping Families Cope with Mental Illness: A Guide for the Professions. Formulated by the Committee on Psychiatry and the Community, Group for the Advancement of Psychiatry. 102 pp. New York, Brunner/Mazel Publishers, 1986. $12.50 (paperback)

Medical Abbreviations: 4200 Conveniences at the Expense of Communications and Safety. Third Edition. By Neil M. Davis, MS, Pharm D. 102 pp. Huntingdon Valley, Pa, Neil M. Davis Associates, 1987. $4.95 (paperback)

Manhattan Country Doctor. By Milton Slocum, MD. 271 pp. New York, Charles Scribner's Sons, 1986. $16.95 (hardcover)

NEUROLOGY

Brain Matters: Stories of a Neurologist and His Patients. By Bruce H. Dobkin, MD. 202 pp. New York, Crown Publishers, Inc, 1986. $17.95 (hardcover)

NEUROSURGERY

Spine. Failed Back Surgery Syndrome: Evaluation and Treatment. Edited by Arthur H. White, MD. 179 pp, illustrated. Philadelphia, Hanley & Belfus, Inc, 1986. $30.00 (hardcover)

NUTRITION

The Dachman Diet for Kids. A Complete Guide to Healthy Weight Loss. By Ken Dachman. 224 pp. New York, World Almanac Publications, 1986. $15.95 (hardcover)

Bonnie Prudden's After Fifty Fitness Guide. By Bonnie Prudden. 373 pp, illustrated. New York, Random House, 1986. $19.95 (hardcover)

OBSTETRICS AND GYNECOLOGY

Night Calls. The Personal Journey of an OB/GYN. By Henry Eisenberg, MD, Arlene Eisenberg, and Howard Eisenberg. 281 pp. New York, Arbor House Publishing Co, 1986. $16.95 (hardcover)

Atlas of the Human Oocyte and Early Conceptus. By Lucinda L. Veeck, MLT, ASCP. 331 pp, illustrated. Baltimore, Williams & Wilkins, 1986. $110.00 (hardcover)

ONCOLOGY

Pathology of Neoplasia in Children and Adolescents. By Milton Finegold, MD. 512 pp, illustrated. Philadelphia, WB Saunders Co, 1986. $60.00 (hardcover)

OTOLARYNGOLOGY

Toxicology of the Nasal Passages. Edited by Craig S. Barrow, PHD. 317 pp, illustrated. New York, Hemisphere Publishing Corp, 1986. $61.95 (hardcover)

PHARMACOLOGY

Applied Pharmacokinetics. Principles of Therapeutic Drug Monitoring. Second Edition. Edited by William E. Evans, Pharm D, Jerome J. Schentag, Pharm D, William J. Jusko, PHD. 1,272 pp, illustrated. Spokane, Wash, Applied Therapeutics, Inc, 1986. $60.00 (hardcover)

PLASTIC SURGERY

An Anthology of Plastic Surgery. Edited by Harry Hayes, Jr, MD. 338 pp, illustrated. Rockville, Md, Aspen Publishers, Inc, 1986. $55.00 (hardcover)

PSYCHIATRY

Mental Retardation. Nature, Cause and Management. Second Edition. By George S. Baroff. 541 pp, illustrated. New York, Hemisphere Publishing Corp, 1986. $44.95 (hardcover)

RADIOLOGY

Computed Tomographic Angiography of the Mediastinum. By Lee Chiu, MD, William Stanford, MD, and Vicky Yiu-Chiu, MD. 214 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1986. $57.50 (hardcover)

SPORTS MEDICINE

Exercise and Sports Sciences Reviews. Volume 14. Edited by Kent B. Pandolf, PHD. 457 pp, illustrated. New York, Macmillan Publishing Co, 1986. $39.95 (hardcover)

SURGERY

Advances in Upper Extremity Surgery and Rehabilitation. Edited by John A. Boseick, Jr, MD. 303 pp, illustrated. Rockville, Md, Aspen Publishers, Inc, 1986. $43.50 (hardcover)

Basic Surgery. By John A. McCredie, MCH, Gerard P. Burns, MCH. 656 pp, illustrated. New York, Macmillan Publishing Co, 1986. $29.95 (softcover)

TOXICOLOGY

Casarett and Doull's Toxicology. The Basic Science of Poisons. Third Edition. By Curtis D. Klaassen, PHD, Mary O. Amdur, PHD, and John Doull, MD. 974 pp, illustrated. New York, Macmillan Publishing Co, 1986. $49.95 (hardcover)

How the new tax law will affect physicians

The Tax Reform Act of 1986 threatens the deductibility of membership dues for professional societies and of expenses associated with continuing education courses. Attorneys Jack R. Bierig and John P. Simon of the law firm of Sidley & Austin (Chicago, Ill) have prepared a report detailing the effects of the new tax law.

The authors summarize the changes in the tax law as follows: "It will substantially restrict the ability of employed individuals to deduct dues and expenses to the extent that they are not reimbursed by the employer. It will also reduce the amount which all taxpayers can deduct for meal and entertainment expenses, including those incurred in connection with attending a professional education seminar. Finally, it will eliminate deductions for expenses incurred in attending investment or financial planning seminars unrelated to the individual's profession or business."

Prior to January 1, 1987—when the revised tax law went into effect—medical society dues and expenses related to attendance at educational seminars were fully deductible. If the physician's employer or partnership paid for these items, the payer could deduct these expenses. Under the new law, an employed physician can only deduct the amount of professional dues and educational expenses that exceeds 2% of his or her adjusted gross income. However, these items are still fully deductible if paid for by the physician's employer. Consequently, Bierig and Simon anticipate that physicians will seek payment of these expenses or reimbursement by their employers.

In addition, the authors note that while the registration fees and the transportation and lodging costs associated with attendance at professional seminars will remain fully deductible, only 80% of the cost of food and entertainment will be deductible.

As of January 1, the maximum tax bracket has dropped from 50% to 38½%. Therefore, deductions are more valuable in 1986 than they will be in 1987. The authors recommend that "for physicians in high tax brackets, it may make sense to pay 1987 expenses in 1986 to the extent legitimately possible."

FDA approval announced for psoriasis drug

The Food and Drug Administration (FDA) recently approved the drug etretinate for the treatment of patients with severe or disabling forms of psoriasis. In clinical trials with the drug, patients experienced a reduction in scaling, redness, and inflammation as well as a decrease in the thickness of the skin lesions. The drug has been shown to be effective in two refractory forms of the disease: erythrodermic and pustular psoriasis.

Etretinate, which is chemically related to vitamin A, can cause major birth defects, and the FDA has included a strong warning against its use by women who are pregnant or who are planning to become pregnant. (The drug has been detected in patients' blood for almost three years after they stopped taking it). Other side effects seen in individuals taking the drug include liver damage as well as corneal and skeletal changes.

Hoffman La-Roche will market etretinate under the brand name Tegison.

HIV antibodies in commercial intravenous gammaglobulin products

Antibody against human immunodeficiency virus (HIV)—the virus that causes AIDS—is present in a variety of commercial blood products, including gammaglobulin preparations. HIV transmission has been shown following infusion of blood components and clotting factor concentrates, but viral transmission has not been reported following gammaglobulin therapy.

Wood et al (*Ann Intern Med* 1986; 105:536–538) tested commercial preparations of intravenous gammaglobulin for evidence of antibody against HIV and viral activity. They also screened serum samples from immunodeficient patients who received intravenous gammaglobulin for the presence of antibody against HIV. Testing methods included enzyme-linked immunosorbent assay (ELISA) and Western blot analysis. The commercial preparations evaluated included reduced and alkylated gammaglobulin lots and pH4/pepsin-treated lots.

The investigators found HIV antibody in ten out of ten lots of reduced and alkylated gammaglobulin and in four out of eight pH4/pepsin-treated lots on ELISA analysis. Western blotting revealed antibody in eight out of ten reduced and alkylated lots and in three out of eight lots of pH4/pepsin-treated gammaglobulin. The authors attribute these differences to the increased sensitivity and reduced specificity of the ELISA. Serum analysis following infusion of gammaglobulin produced the following results: one third of the samples were seropositive by ELISA, and one sixth yielded positive findings on Western blot analysis. No viral activity was detected in the seropositive lots of gammaglobulin.

From these findings the authors concluded that although patients who receive intravenous gammaglobulin containing HIV antibody may become "transiently seropositive," the antibody does not appear to be associated with viral infection.

The immune system's response to cancer

In a recent issue of the *Archives of Surgery* (1986; 121:1237–1241), Harvey W. Baker, MD, from the Oregon Health Sciences University, discussed the role of the body's immune system in responding to and controlling neoplastic disease. He questioned the view of a tumor as an autonomous growth, supporting instead the belief that the host's response to the cancerous tissue can greatly affect progression of the disease.

Baker noted that there have been reports of spontaneous regression of almost every type of benign and malignant lesion. In addition, regression of primary tumors has been reported, while metastases from those tumors may

---

NEWS BRIEFS are compiled and written by Vicki Glaser.

remain or may appear at a later time. To further support the role of the host immune system in combatting cancer, Baker cited examples of prolonged patient survival with no therapy or following unsuccessful attempts at treatment.

Antitumor immune responses appear to be initiated by antigens on the surface of tumor cells. These antigens are probably not specific to the tumor, but are also expressed at a low level by normal host cells. Recognition of tumor-associated antigens stimulates T lymphocytes to produce a variety of lymphokines, including the interferons, interleukin-2, and tumor necrosis factor, that can lead to the destruction of tumor cells.

Although past attempts at immunotherapy have produced "few encouraging results," Baker foresees a bright future for the new breed of immunotherapeutic agents including the lymphokines, soluble tumor extracts, vaccines composed of tumor cell factors, and monoclonal antibodies that can direct cytotoxic drugs to cancerous tissue and can block immunosuppressive factors.

FROM THE LIBRARY

DIETHYLSTILBESTROL AS AN AID IN THE TREATMENT OF PSORIASIS

Theories as to the cause of psoriasis are numerous and, as in any disease of unknown etiology, the treatment is varied and not very satisfactory. Out of the maze of clinical and laboratory findings there is much to indicate a relationship between the endocrine system and psoriasis.

The value of sunlight in psoriasis has long been known, and the fact that the disease is rare in the Negro suggests a relationship between melanin on one hand, and the adrenals or gonads on the other.

Reynolds states that "if hypogonadal individuals (man or woman) are exposed to sunlight they fail to tan readily although an invisible change takes place in the tissues. Thus, if androgens or estrogens are injected subsequent to exposure to ultraviolet light, pigmentation of the skin occurs without further exposure to light. Injections of the hormones into nonphotosensitized individuals fail to elicit pigmentation. The relation of this phenomenon to the mechanism of carotin and melanin is unknown. It is suggestive that sex hormones have a sterol nucleus somewhat similar to that of adrenal cortical hormone in the absence of which coloration of the skin characteristic of Addison's disease takes place."

Fat metabolism has long been believed to play a role in psoriasis, although the findings of a hypercholesteremia have been contradictory.

Gruneberg and others believe the adrenal plays an important role in psoriasis. LeWinn and Urbach in a recent article sum up the evidence in the following manner: Definite decreases in urinary vitamin C excretion, the decrease in the amount of sulfur excreted in the urine, and the increase in the blood potassium level all found in psoriasis, infer a relationship between this disease and the adrenal cortex.

Unfortunately, the therapeutic application of this relationship has produced no uniformly successful result, and treatment is still mainly directed at the local condition of the skin rather than by the use of any systemic therapy. . . .

Lawrence Frank, MD
(*NY State J Med* 1947; 47:1790–1791)

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

John Frederick Argue, MD, Wilson. Died September 28, 1986; age 76. Dr Argue was a 1935 graduate of University of Buffalo School of Medicine. He was a Fellow of the American Academy of Family Practice, a Diplomate of the American Board of Family Practice, and a member of the Medical Society of the County of Niagara and the Medical Society of the State of New York.

Anne Magdalen Bahlke, MD, Albany. Died August 6, 1986; age 83. She was a 1936 graduate of Johns Hopkins University School of Medicine, Baltimore. Dr Bahlke was a Fellow of the American College of Preventive Medicine and a Diplomate of the American Board of Preventive Medicine, Inc. Her memberships included the American Public Health Association, the Medical Society of the County of Albany, and the Medical Society of the State of New York.

John Percy Baker, MD, Huntington. Died October 4, 1986; age 92. He was a 1921 graduate of Columbia University College of Physicians and Surgeons, New York. Dr Baker was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Otolaryngology. His memberships included the American Academy of Otolaryngology; the American Laryngological, Rhinological and Otological Society; the American Academy of Facial Plastic and Reconstructive Surgery; the American Broncho-Esophagological Association; the Medical Society of the County of Kings; and the Medical Society of the State of New York.

Valer Barbu, MD, New York. Died August 25, 1986; age 94. Dr Barbu was a 1915 graduate of Eotvos Lorand Tudomanyegyetem Orvostudományi Fakultas, Budapest. Dr Barbu was a Diplomate of the American Board of Psychiatry and Neurology, and a member of the American Psychiatric Association, the Academy of Psychoanalysis, the New York County Medical Society, and the Medical Society of the State of New York.

Alfred Joseph Barra, MD, Forest Hills. Died September 29, 1986; age 79. He was a 1931 graduate of Georgetown University School of Medicine, Washington, DC. Dr Barra was a Qualified Fellow of the International College of Surgeons and a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

George Dows Cannon, MD, New York. Died August 31, 1986; age 84. Dr Cannon was a 1934 graduate of Rush Medical College, Chicago. He was a member of the Academy of Medicine, the American Academy of Family Practice, the New York County Medical Society, and the Medical Society of the State of New York.

Arthur Henry Carson, MD, Elmhurst. Died October 14, 1986; age 79. He was a 1930 graduate of Tufts University School of Medicine, Boston. Dr Carson was a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Urology. He was a member of the American Urological Association, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Thomas Joseph Costigan, MD, Westwood, New Jersey. Died July 31, 1986; age 83. Dr Costigan was a 1930 graduate of Georgetown University School of Medicine, Washington, DC. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Clifton Louis Dance, MD, Staten Island. Died September 5, 1986; age 93. He was a 1918 graduate of Wayne State University College of Medicine, Detroit. Dr Dance was a Qualified Fellow of the International College of Surgeons and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

C. Pinckney Deal, MD, New York. Died October 21, 1986; age 55. Dr Deal was a 1958 graduate of Harvard Medical School, Boston. Dr Deal was a member of the New York State Society of Internal Medicine, the American Society of Internal Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Joseph P. Demeri, MD, Kew Gardens. Died October 20, 1986; age 65. He was a 1945 graduate of New York Medical College, Flower and Fifth Avenue Hospitals, New York. Dr Demeri was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Orthopedic Surgery. His memberships included the Academy of Medicine, the American Academy of Orthopaedic Surgeons, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Miguel Grausman Elias, MD, New York. Died July 28, 1986; age 90. Dr Elias was a 1921 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American College of Surgeons and a member of the Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Almer Tristan George, MD, Glenmont. Died September 21, 1986; age 76. Dr George was a 1937 graduate of New York University College of Medicine, New York. Dr George was a member of the New York State Society of Occupational Medicine, the American Occupational Medical Association, the American Academy of Compensation Medicine, Inc, the Medical Society of the County of Albany, and the Medical Society of the State of New York.

Nathan Gordon, MD, Los Angeles, California. Died September 29, 1986; age 68. Dr Gordon was a 1942 graduate of the University of the Witwatersrand Medical School, Johannesburg. He was a Diplomate of the American Board of Family Practice and a member of the American Academy of Family Practice, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Israel Gottlieb, MD, Miami Beach, Florida. Died March 15, 1986; age 98. He was a 1915 graduate of New York University College of Medicine, New York. Dr Gottlieb was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the Academy of Medicine, the American Academy of Neurology, the American Psychiatric Association, the Bronx County Medical Society, and the Medical Society of the State of New York.

Zafar Haider, MD, New Hartford. Died October 19, 1986; age 51. Dr Haider was a 1959 graduate of King Edward Medical College, Lahore, Punjab. Dr Haider was a Diplomate of the American Board of Pathology and a member of the Medical Society of the County of Oneida and the Medical Society of the State of New York.

Anwar J. Hanania, MD, New York. Died October 22, 1986; age 78. Dr Hanania was a 1935 graduate of the Université de Paris Faculté de Médecine. Dr Hanania was a Diplomate of the American Board of Obstetrics and Gynecology and a member of the American Fertility Society, the New York County Medical Society, and the Medical Society of the State of New York.

Renee Koehler Imberman, MD, New York. Died August 28, 1986; age 76. Dr Imberman was a 1935 graduate of Medizinische Fakultät der Universität, Wien. Dr Imberman was a member of the New York County Medical Society and the Medical Society of the State of New York.

Samuel Kampf, MD, Scarsdale. Died September 24, 1986; age 75. He was a 1940 graduate of Université de Lausane Faculté de Médecine. Dr Kampf was a member of the American Academy of Family Practice, the Bronx County Medical Society, and the Medical Society of the State of New York.

Mortimer A. Lasky, MD, Rockville, Maryland. Died August 16, 1986; age 82. Dr Lasky was a 1926 graduate of State University of New York Health Science Center at Brooklyn, New York. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Merny M. Laster, MD, Baldwin. Died September 24, 1986; age 75. Dr Laster was a 1936 graduate of Columbia University College of Physicians and Surgeons, New York. Dr Laster was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Tiffany Lawyer, Jr, MD, Bronxville. Died September 30, 1986; age 71. Dr Lawyer was a 1942 graduate of Albany Medical College of Union University. Dr Lawyer was a Diplomate of the American Board of Psychiatry and Neurology and a member of the Academy of Medicine, the New York Neurological Society, the American Neurological Association, the American Academy of Neurology, the American Psychiatric Association, the Association for Research in Nervous and Mental Disease, the Bronx County Medical Society, and the Medical Society of the State of New York.

Sidney Lecker, MD, New York. Died September 21, 1986; age 49. He was a 1962 graduate of McGill University Faculty of Medicine, Montreal, Canada. Dr Lecker was a Diplomate of the American Board of Psychiatry and Neurology and a member of the New York County Medical Society and the Medical Society of the State of New York.

H. George Liebmann, MD, New York. Died September 11, 1986; age 90. Dr Liebmann was a 1920 graduate of Julius-Maximilians-Universität Medizinische Fakultät, Wurzburg, Bavaria. He was a Fellow of the American College of Obstetricians and Gynecologists and a member of the New York County Medical Society and the Medical Society of the State of New York.

John Loesch, MD, Hurley. Died August 28, 1986; age 89. He was a 1924 graduate of Deutsche Universität Medizinische Fakultät, Prague. Dr Loesch was a Fellow of the American College of Chest Physicians and a Diplomate of the American Board of Pathology. His memberships included the American Thoracic Society, the American Association of Pathologists and Bacteriologists, the Medical Society of the County of Ulster, and the Medical Society of the State of New York.

Joseph Bruce Logue, MD, Brooklyn. Died October 1, 1986; age 91. Dr Logue was a 1918 graduate of Medical College of Georgia, Augusta. He was a Fellow of the American College of Surgeons and a member of the New York County Medical Society and the Medical Society of the State of New York.

Felix Peter Loiacono, MD, Danville, California. Died September 1986; age 82. Dr Loiacono was a 1933 graduate of Creighton University School of Medicine, Omaha. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Edward H. Mandel, MD, New York. Died October 13, 1986; age 65. He was a 1945 graduate of New York Medical College, Flower and Fifth Avenue Hospitals, New York. Dr Mandel was a Fellow of the American College of Physicians and a Diplomate of the American Board of Dermatology, Inc. His memberships included the Academy of Medicine, the New York State Dermatological Society, the American Dermatological Association, the Society for Investigative Dermatology, the American Academy of Dermatology, the New York County Medical Society, and the Medical Society of the State of New York.

Myer Michael Medine, MD, New York. Died October 8, 1986; age 74. Dr Medine was a 1936 graduate of McGill University Faculty of Medicine, Montreal, Canada. He was a Fellow of the American College of Surgeons, a Diplomate of the American Board of Ophthalmology, and a member of the American Academy of Ophthalmology and Otolaryngology, the New York County Medical Society, and the Medical Society of the State of New York.

Americus J. Paganelli, MD, Germantown. Died September 9, 1986; age 82. He was a 1931 graduate of Università di Napoli Facoltà di Medicina e Chirurgia. Dr Paganelli was a Qualified Fellow of the International College of Surgeons and a member of the New York County Medical Society and the Medical Society of the State of New York.

Paul Schultze, Jr, MD, Menands. Died July 14, 1986; age 81. Dr Schultze was a 1932 graduate of Albany Medical College of Union University. He was a Fellow of the American College of Obstetricians and Gynecologists and a member of the Medical Society of the County of Albany and the Medical Society of the State of New York.

Joseph Schwartzwald, MD, Boynton Beach, Florida. Died October 5, 1986; age 75. He was a 1937 graduate of Medizinische Fakultät der Universität, Wien. Dr Schwartzwald was a Fellow of the American College of Anesthesiologists. His memberships included the American Society of Anesthesiologists, Inc, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Harry Sonnenschein, MD, Lakehurst, New Jersey. Died September 23, 1986; age 81. Dr Sonnenschein was a 1928 graduate of State University of New York Health Science Center at Brooklyn, New York. He was a Diplomate of the American Board of Pediatrics and a member of the American Academy of Pediatrics, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Louis Stix, MD, Mill Neck. Died October 26, 1986; age 84. Dr Stix was a 1931 graduate of Columbia University College of Physicians and Surgeons, New York. He was a member of the Academy of Medicine, the American Psychiatric Association, the Academy of Psychoanalysis, the Nassau County Medical Society, and the Medical Society of the State of New York.

Maurice Joel Stone, MD, Bronx. Died September 1, 1986; age 90. He was a 1920 graduate of Columbia University College of Physicians and Surgeons, New York. Dr Stone was a member of the American Academy of Family Practice, the Bronx County Medical Society, and the Medical Society of the State of New York.

Irving J. Thorne, MD, New York. Died August 6, 1986; age 79. Dr Thorne was a 1934 graduate of Harvard Medical School, Boston. He was a Fellow of the American College of Surgeons, a Qualified Fellow of the International College of Surgeons, and a Diplomate of the American Board of Surgery. Dr Thorne's memberships included the New York State Surgical Society, the Pan American Medical Association, the Aerospace Medical Association, the New York County Medical Society, and the Medical Society of the State of New York.

Raul Arturo Trillo, MD, Howard Beach. Died October 27, 1986; age 65. He was a 1946 graduate of Universidad de la Habana Facultad de Medicina y Farmacia, Cuba. Dr Trillo was a Fellow of the American College of Surgeons, a Diplomate of the American Board of Surgery, and a member of the New York County Medical Society and the Medical Society of the State of New York.

Robert Wachen, MD, Brooklyn. Died October 23, 1986; age 86. He was a 1924 graduate of Universität Heidelberg Medizinische Fakultät, Baden. Dr Wachen was a Fellow of the American Academy of Family Practice, the American College of Cardiology, and the American College of Angiology. His memberships included the American Geriatrics Society, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1983; 83:1089–1094). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith[2] reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al*. Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83: 925–927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337–338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, P.O. Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

Schedule of Sessions 69
Officers, Councilors, and Trustees 71
Members of the House 74
Reference Committees 78
Nominating Committee 80
Report of the House Committee on Bylaws 81

HOUSE OF DELEGATES

March 12 through 15, 1987
New York Hilton

SCHEDULE of SESSIONS

Thursday, March 12
Opening Session .... 1:00 p.m.

Friday, March 13
Reference Committee Hearings .... 8:30 a.m.–5:00 p.m.

Saturday, March 14
Second Session .... 9:00 a.m.
Third Session .... 1:30 p.m.

Sunday, March 15
Closing Session .... 9:00 a.m.

A Guide to the 1987 Annual Meeting

Policy-making in The House of Delegates

This section of the New York State Journal of Medicine is a call to the officers, delegates, and members of the Medical Society of the State of New York to attend the 181st Annual Meeting of the Society and the meeting of its House of Delegates; 299 delegates representing 61 county medical societies, 9 district branches, specialty societies, the hospital medical staff section, 12 medical schools, the resident and medical student membership will meet at the Hilton Hotel in New York City on March 12–15, 1987.

The officers, members of the Council and the Board of Trustees, past presidents of the MSSNY, the Commissioner of the New York State Department of Health, and the Past President of the American Medical Association from New York, Gerald D. Dorman, MD, are also official representatives to the House.

Mandated by the Bylaws of the Society, this section lists the names of elected representatives, delegates who are serving on reference committees, and selections of the Nominating Committee for the election of Officers, Councilors, Trustees and AMA Delegates. Also included are the amendments to the Bylaws which will be considered by the House of Delegates.

Since the House of Delegates is the legislative body of the Society, charged with the general management of the State Society and its affairs, members of the Society are urged to use this information to consult with and advise their representatives. Resolutions introduced by the representatives bring members' concerns to the attention of the entire Society. Representatives not only have the responsibility to present the views of their constituents, but also to inform them of the actions taken by this governing body. The House of Delegates also formulates policy for the Society. No officer, councilor, board, commission, committee, or employee may initiate any policy unless such policy has been expressly approved by the House of Delegates, or by the Council of the Society.

If one were to choose the most important work of the House of Delegates, it would have to be that of the reference committees. Any member of the Medical Society of the State of New York has the right to appear before any reference committee and participate in its deliberations. It is also in the reference committees that people outside of the State Society are asked to give their views. Nonmember phyisicians, guests or interested observers who wish to express their opinions may, with permission, come before the reference committee and speak to the business of the committee. I strongly urge members to use this forum to express their views.

Charles N. Aswad, MD, Speaker
House of Delegates

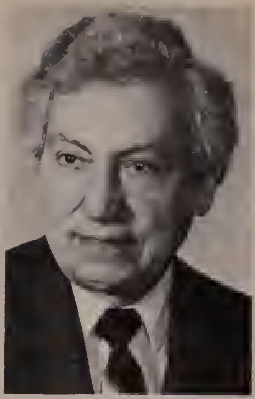
MEDICAL SOCIETY OF THE STATE OF NEW YORK

ORGANIZED 1807

Officers 1986–1987

John A. Finkbeiner

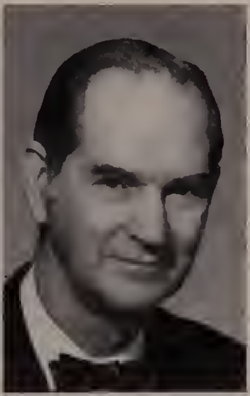
New York
President


Samuel M. Gelfand

Nassau
President-Elect

Daniel F. O'Keeffe

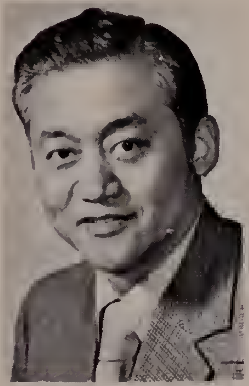
Warren
Past-President


Charles D. Sherman, Jr.

Monroe
Vice-President

John H. Carter

Albany
Secretary


George Lim

Oneida
Assistant Secretary

Morton Kurtz

Queens
Treasurer

Robert A. Mayers

Westchester
Assistant Treasurer

Charles N. Aswad

Broome
Speaker

Seymour R. Stall

Dutchess
Vice-Speaker

Board of Trustees 1986–1987

Ralph M. Schwartz

Kings
Chairman

Edgar P. Berry

New York

Richard D. Eberle

Onondaga

Joseph F. Shanaphy

Richmond

Victor J. Tofany

Monroe

Bernard J. Pisani

New York

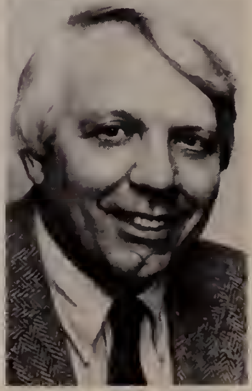
Daniel F. O'Keeffe

Warren

Councilors 1986–1987

David M. Benford

New York



Duane M. Cady

Onondaga

James M. Flanagan

Wayne

M. Theodore Tanenhaus

Kings

James H. Cosgriff, Jr.

Erie

Richard A. Hughes

Warren

Antonio F. LaSorte

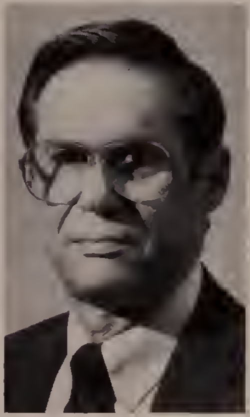
Broome

Sidney Mishkin

Nassau

Robert E. Fear

Suffolk

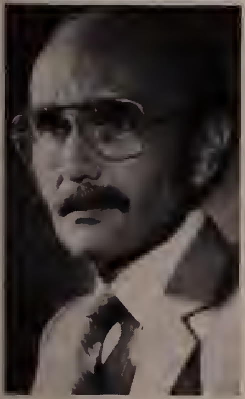


Stanley L. Grossman

Orange

Thomas D. Pemrick

Rensselaer



Ralph E. Schlossman

Queens

Kathleen E. Squires

New York
Resident

Frances Hartenstein-Collichio

Monroe
Medical Student

1987 HOUSE OF DELEGATES MEMBERS

The following is a list of members of the 1987 House of Delegates of the Medical Society of the State of New York.

Officers 1986–1987
President—John A. Finkbeiner, New York
President-Elect—Samuel M. Gelfand, Nassau
Vice-President—Charles D. Sherman, Jr., Monroe
Secretary—John H. Carter, Albany
Assistant Secretary—George Lim, Oneida
Treasurer—Morton Kurtz, Queens
Assistant Treasurer—Robert A. Mayers, Westchester
Speaker—Charles N. Aswad, Broome
Vice Speaker—Seymour R. Stall, Dutchess

Councilors
Term Expires 1987
David M. Benford, New York
Duane M. Cady, Onondaga
James M. Flanagan, Wayne
M. Theodore Tanenhaus, Kings

Term Expires 1988
James H. Cosgriff, Jr., Erie
Richard A. Hughes, Warren
Antonio F. LaSorte, Broome
Sidney Mishkin, Nassau

Term Expires 1989
Robert E. Fear, Suffolk
Stanley L. Grossman, Orange
Thomas D. Pemrick, Rensselaer
Ralph E. Schlossman, Queens

Student Councilor
Term Expires 1987
Frances Hartenstein-Collichio, Monroe

Resident Councilor
Term Expires 1987
Kathleen E. Squires, New York

Trustees
Ralph M. Schwartz, Kings, *Chairman*
Edgar P. Berry, New York
Richard D. Eberle, Onondaga
Joseph F. Shanaphy, Richmond
Victor J. Tofany, Monroe
Bernard J. Pisani, New York
Daniel F. O'Keeffe, Warren

Past Presidents
1959–1960—Henry I. Fineberg, Suffolk
1960–1961—Norman S. Moore, Tompkins
1961–1962—John M. Galbraith, Nassau
1966–1967—James M. Blake, Schenectady
1969–1970—Walter T. Heldmann, Richmond
1970–1971—Walter Scott Walls, Erie
1971–1972—George Himler, New York
1972–1973—Edward Siegel, Clinton
1974–1975—Lynn R. Callin, Monroe
1975–1976—Ralph S. Emerson, Nassau
1976–1977—George L. Collins, Jr., Erie
1977–1978—Carl Goldmark, Jr., New York
1978–1979—George T. C. Way, Dutchess
1979–1980—G. Rehmi Denton, Albany
1980–1981—Ralph M. Schwartz, Kings
1982–1983—Richard D. Eberle, Onondaga
1983–1984—Bernard J. Pisani, New York
1984–1985—Allison B. Landolt, Westchester
1985–1986—Daniel F. O'Keeffe, Warren

Past President of the American Medical Association
Gerald D. Dorman, New York

Vice Speaker of the American Medical Association
John L. Clowe, Schenectady

Commissioner, New York State Department of Health

District Delegates
(*Elected Delegates of District Branches*)
First—Roger D. Billig, Richmond
Second—Robert Bruce Bergmann, Nassau
Third—Thomas W. Greenlees, Schoharie
Fourth—Michael J. Lynch, Washington
Fifth—Frederic A. Stone, Jefferson
Sixth—Vincent I. Maddi, Broome
Seventh—Verne M. Marshall, Ontario
Eighth—Robert James Reszel, Niagara
Ninth—Francis J. McMahon, Dutchess

Specialty Society Delegates
New York State Society of Anesthesiology—Edward C. Sinnott, Nassau
New York State Society of Dermatology—David Sibulkin, New York
New York Chapter American College of Emergency Physicians—Ralph Altman, New York
New York State Academy of Family Physicians—James R. Nunn, Erie
New York State Society of Internal Medicine—Robert M. Kohn, Erie
New York State Neurosurgical Society, Inc.—Russel H. Patterson, Jr., New York
American College of Obstetricians and Gynecologists—District II—Leonard S. Weiss, Orange
New York Occupational Medical Association, Inc.—Thomas J. Doyle, Queens

New York State Ophthalmological Society—Frank J. Piper, Onondaga
New York State Society of Orthopaedic Surgeons, Inc.—Leo A. Green, Queens
New York State Society of Otolaryngology—Head and Neck Surgery—Fouad B. Michael, Nassau
New York State Society of Pathologists—Edward C. Zaino, Nassau
American Academy of Pediatrics—District II—Philip W. H. Eskes, Queens
New York Society of Physical Medicine and Rehabilitation, Inc.—Gabriel S. Rinzler, Westchester
New York Regional Society of Plastic and Reconstructive Surgery—Philip C. Bonanno, Westchester
New York State Psychiatric Association—Harold E. Berson, Kings
New York State Chapter—American College of Radiology—Victor A. Panaro, Erie
New York State Society of Surgeons, Inc.—Theodore C. Max, Oneida
New York State Urological Society, Inc.—

Delegates from Component County Medical Societies

Albany (*4*)
John W. Abbuhl, Albany
William O'Dwyer, Latham
James H. Puleo, Albany
Nathan P. Reed, Albany

Allegany (*1*)
Gary W. Ogden, Andover

Bronx (*10*)
John P. Albanese, Bronx
Marcos A. Charles, New Rochelle
Dattatraya G. Lanjewar, Pelham Manor
Charles LaVerdi, Bronx
Hugo M. Morales, Bronx
Raul Pohorille, New Rochelle
Govindappa Rangaiah, Bronx
Howard S. Schwartz, Bronx
Tullio F. Tartaglia, Rye
Juana Toporovsky, Bronx

Broome (*4*)
Louis W. Giordano, Endicott
Theodore R. Poritz, Binghamton
Jeffrey A. Ribner, Binghamton
Irwin J. Rosenberg, Endicott

Cattaraugus (*1*)
Waleed Saleh, Olean

Cayuga (*1*)
Richard J. Freeman, Auburn

Chautaugua (*2*)
James G. Dahlie, Jamestown
Robert W. Scott, Jamestown

Chemung (*2*)
Donald L. Brooks, Elmira
Gerald P. Schneider, Elmira

Chenango (*1*)
Norman C. Lyster, Norwich

Clinton (*2*)
Soham S. Patel, Plattsburgh

Columbia (*1*)
Richard B. Weininger, Hudson

Cortland (*1*)
Patrick J. Hayes, Cortland

Delaware (*1*)
Gary Preiser, Walton

Dutchess (*4*)
Eric R. Brocks, Fishkill
Stephen R. Chernay, Fishkill
Arthur W. Menken, Poughkeepsie
Pinaki Ray, Poughkeepsie

Erie (*9*)
Thomas W. Bradley, Kenmore
Anthony J. Federico, Buffalo
Edmond J. Gicewicz, Buffalo
Allen L. Lesswing, Snyder
Milford C. Maloney, Buffalo
Leo E. Manning, Amherst
M. Luther Musselman, Buffalo
James F. Phillips, Buffalo
Anthony P. Santomauro, Tonawanda

Essex (*1*)

Franklin (*1*)
Alfred A. Hartmann, Sr., Malone

Fulton (*1*)
Armand J. D'Errico, Gloversville

Genesee (*1*)
Bruce E. Baker, Le Roy

Greene (*1*)
Karuvath Enu, Catskill

Herkimer (*1*)
Krishna K. Vadlamudi, Ilion

Jefferson (*2*)
W. Bruce Carter, Watertown
David O. VanEenenaam, Watertown

Kings (*19*)
Grace A. Altenau, Brooklyn
Lawrence Ames, Brooklyn
Norman S. Blackman, Brooklyn
Randall D. Bloomfield, Brooklyn
Joseph R. Brennan, Brooklyn
George W. Calvosa, Brooklyn
Vernal G. Cave, Brooklyn
Duncan W. Clark, Brooklyn
Paul Finkelstein, Brooklyn

Robert E. Gordon, Brooklyn
Ladislav P. Hinterbuchner, Brooklyn
Donald Mandel, Brooklyn
A. W. Martin Marino, Jr., Brooklyn
Martin Markowitz, Brooklyn
Bentley D. Merrim, Brooklyn
Hubert S. Pearlman, Brooklyn
Leon M. Rothman, New York
Veronica Santilli, Brooklyn
Leo J. Swirsky, Brooklyn

Lewis (*1*)
John C. Herrman, Lowville

Livingston (*1*)
Sheppard Arluck, Geneseo

Madison (*1*)
Leonard A. Argentine, Oneida

Monroe (*7*)
Marilyn R. Brown, Rochester
William A. Dolan, Rochester
Warren Glaser, Rochester
William A. Kern, Rochester
Vito P. Laglia, Rochester
Mario B. Lo Monaco, Rochester
Joyce M. McChesney, Rochester

Montgomery (*1*)
Feori F. Pipito, Amsterdam

Nassau (*11*)
Lee S. Binder, Valley Stream
Morton Goldfarb, Massapequa Park
Jeffrey T. Kessler, Great Neck
Paul C. Lehmuller, Levittown
Arnold D. Lurie, Baldwin
Alfred W. Marks, Rockville Centre
Felix A. Monaco, Rockville Centre
John A. Ostuni, Freeport
Neal A. Sckolnick, Rockville Centre
Arthur J. Wise, Manhasset
Jerome Zwanger, Massapequa

New York (*23*)
Arnold L. Bachman, New York
Melvin H. Becker, Manhasset
Robert S. Bernstein, New York
Irwin J. Cohen, New York
John W. Cornwall, New York
Hugh Clark Davidson, New York
Wilbur J. Gould, New York
George Hyams, New York
Virginia Kanick, New York
Vance Lauderdale Jr, New Jersey
Samuel H. Madell, New York
Aaron G. Meislin, New York
George W. Melcher, New York
Alfred Moldovan, New York
Richard B. Nolan, New York
Donna B. O'Hare, New York
Stuart I. Orsher, New York
Richard N. Pierson, Jr., New York
William B Rawls, Pleasant Valley
Isadore Rosenfeld, New York
Albert M. Schwartz, New York
Clifford L. Spingarn, New York
Meyer Texon, New York

Niagara (*3*)
Frank P. Altieri, Lewiston
Claus Michael Fichte, Niagara Falls
James J. Kropelin, Niagara Falls

Oneida (*4*)
Gerald C. Gant, New Hartford
Robert M. George, Utica
Neville W. Harper, Rome
Robert S. Manogue, Utica

Onondaga (*6*)
Daniel L. Dombroski, Syracuse
Daniel S. Fuleihan, Syracuse
John A. Hoepner, Syracuse
Richard A. Konys, Syracuse
Arthur Lehrman, Syracuse
George P. Tilley, Syracuse

Ontario (*2*)
Kenneth T. Steadman, Geneva
Thomas A. McCormick, Geneva

Orange (*4*)
Jacob J. Barie, Middletown
Stanley F. Brunn, Port Jervis
Michael A. Parmer, Port Jervis
Seymour J. Weiner, Port Jervis

Orleans (*1*)
David B. L. Meza III, Albion

Oswego (*1*)
Warren Le Roy Hollis, Lacona

Otsego (*1*)
Rodman D. Carter, Cooperstown

Putnam (*1*)
Hari P. Chakravorty, Mahopac

Queens (*16*)
Marvin L. Blumberg, Jamaica
Daniel Chansky, Forest Hills
John W. V. Cordice, Jr., St. Albans
Daniel G. Deckler, Richmond Hill
Alexander M. De la Garza, Flushing
Robert M. Farrell, Flushing
Fred N. Flatau, Flushing
Irving G. Frohman, Rockaway Beach
Lorraine M. Giordano, Jamaica Estates
Edith Gutman, Beechhurst
Nicetas H. Kuo, Great Neck
Maxwell J. Marder, Forest Hills
Frank Ratner, Flushing

Hilda Ratner, Beechhurst
Sheldon D. Sax, Whitestone
Ezra A. Wolff, Manhasset

Rensselaer (*2*)
William M. McCarthy, Troy
William A. Whyland, Troy

Richmond (*4*)
Robert Bonvino, Staten Island
B. Basil Hyman, Staten Island
Gerald J. Lustig, Staten Island
Robert J. Silich, Staten Island

Rockland (*4*)
Herbert Z. Geller, New City
Michael A. Kalvert, Bardonia
Roy H. Lieberman, Nanuet
Stanley H. Oransky, Bardonia

St. Lawrence (*1*)
Henry J. Dobies, Massena

Saratoga (*1*)
Allen T. Jones, Saratoga Springs

Schenectady (*3*)
Richard H. Lange, Schenectady
John M. Spring, Schenectady
Donald Wexler, Schenectady

Schoharie (*1*)
Frederick J. Michel, Cobleskill

Schuyler (*1*)

Seneca (*1*)
Donald Bidwell, Seneca Falls

Steuben (*2*)
Jack D. O'Neil, Corning
C. Susan Rainwater, Wayland

Suffolk (*11*)
William H. Bloom, Bay Shore
Stephen I. Braitman, Islip Terrace
Clive D. Caplan, Islip Terrace
Frank R. Collier, Port Jefferson
John G. Egner, West Islip
Robert E. Fear, Southampton
Elise H. Korman, Southampton
Phillip I. Levitan, Smithtown
Nathaniel B. Messinger, Islip Terrace
Stanley A. Steckler, Smithtown
William H. Weir, Jr., Riverhead

Sullivan (*1*)
Josef Richter, Ferndale

Tioga (*1*)
P. V. Abraham, Waverly

Tompkins (*2*)
Roger H. Perry, Ithaca

Ulster (*2*)
John V. Ioia, Kingston
Geraldine T. Keyes, Stone Ridge

Warren (*2*)
Franklin A. DePeters, Glens Falls
Robert A. Reid, Glens Falls

Washington (*1*)
Arnold B. Wise, Cambridge

Wayne (*1*)
Norman R. Loomis, Ontario

Westchester (*9*)
Ann C. Cea, Rye Brook
Richard L. Fenton, Tarrytown
Thomas A. Lansen, White Plains
Armond V. Mascia, Tarrytown
Stanley M. Mendelowitz, Tarrytown
Richard L. Petrillo, Mount Vernon
John A. Ramsdell, White Plains
Thomas D. Rizzo, Bronxville
William J. Walsh, White Plains

Wyoming (*1*)

Yates (*1*)

Medical School Delegates (12)
Albany Medical College—Samuel S. Ciccio, Albany
Albert Einstein College of Medicine of Yeshiva University of New York—Melvin N. Zelefsky, Bronx
Columbia University College of Physicians and Surgeons—Linda D. Lewis, New York
Cornell University Medical College—Jack Richard, New York
Mount Sinai School of Medicine of the City University of New York—David A. Dreiling, New York
New York Medical College—Michael Friedland, Westchester
New York University of Medicine—Stanford Wessler, New York
State University of New York Health Science Center at Brooklyn—Stanley Wallace, Kings
State University of New York at Buffalo School of Medicine—Robert Scheig, Erie
State University of New York at Stony Brook School of Medicine—Dorothy Spiegel Lane, Suffolk
State University of New York Health Science Center at Syracuse—John Bernard Henry, Onondaga
University of Rochester School of Medicine and Dentistry—Richard B. Hornick, Monroe

Medical Student Delegates (3)
Stuart Gitlow, New York
Robert T. Geertman, Westchester
Victor S. Sloan, Westchester

Resident Membership Delegate (1)

Hospital Medical Staff Delegate (1)
Richard J. Dwane, Cayuga

REFERENCE COMMITTEES

Reports of Officers and Administrative Matters

John A. Ramsdell, Westchester, *Chairman*
William A. Dolan, Monroe
John Bernard Henry, State University of New York Health Science Center at Syracuse
Virginia Kanick, New York
John A. Ostuni, Nassau

ANNUAL REPORTS REFERRED TO COMMITTEE:

President
President-Elect
Secretary
Treasurer
Board of Trustees
Budget and Finance
Executive Vice-President
New York Delegation to the AMA
District Branches
Liaison Committee with the Nursing Profession
Physicians' Home
Administrative Committee for the MSSNY Pension Program
Membership
General Insurance
Membership Programs
Publications, Library, and Archives
Résumé of Instructions from the 1986 House of Delegates
Ad Hoc Advisory Committee on Real Estate

Bylaws

Randall D. Bloomfield, Kings, *Chairman*
Milford C. Maloney, Erie
Theodore C. Max, NYS Society of Surgeons
Stanley H. Oransky, Rockland
Hilda Ratner, Queens

ANNUAL REPORT REFERRED TO COMMITTEE:

Résumé of Instructions from the 1986 House of Delegates

Governmental Affairs and Legal Matters

Jeffrey A. Ribner, Broome, *Chairman*
Eric R. Brocks, Dutchess
Fred N. Flatau, Queens
Samuel H. Madell, New York
Anthony P. Santomauro, Erie

ANNUAL REPORTS REFERRED TO COMMITTEE:

Ethics
Judicial Council
State Legislation
Federal Legislation
New York State Association of the Professions
Professional Medical Liability Insurance and Defense Board
Conduct of OPMC Investigations
Résumé of Instructions from the 1986 House of Delegates
Ad Hoc Committee on Medical Liability

Medical Services

Dattatraya G. Lanjewar, The Bronx, *Chairman*
Ralph Altman, NYS Chapter American College of Emergency Physicians
Sheppard Arluck, Livingston
Robert S. Bernstein, New York
Joyce M. McChesney, Monroe

ANNUAL REPORTS REFERRED TO COMMITTEE:

Health Care Delivery Systems
Hospital and Professional Relations
Interspecialty
Medicaid
Medical Care Insurance
Social Security Disability Benefits Program, Committees to Provide Input Into
Workers' Compensation and Occupational Health
Résumé of Instructions from the 1986 House of Delegates

Public Health and Education

Michael L. Friedland, New York Medical College, *Chairman*
Leo Arthur Green, New York State Society of Orthopaedic Surgeons, Inc.
Richard A. Konys, Onondaga
Kenneth B. Levites, Suffolk
James H. Puleo, Albany

ANNUAL REPORTS REFERRED TO COMMITTEE:

Accident and Injury Prevention
Aging and Nursing Homes
Alcoholism
Cancer
Cardiovascular Disease
Child Abuse
Drug Abuse
Emergency Medical Services
Home Health Care and Hospice
Maternal and Child Health
Mental Health
Physical Medicine and Rehabilitation
Preventive Medicine
School Health and Sports Medicine

Bioethical Issues
Continuing Medical Education
Forensic Medicine
Information Technology In Medicine
Medical School Relations
Physicians' Health
Scientific Program
Résumé of Instructions from the 1986 House of Delegates
Ad Hoc Committee on the Gellhorn Report

CONVENTION COMMITTEES

Credentials

A. W. Martin Marino, Kings, *Chairman*
Verne M. Marshall, Seventh District
Arthur W. Menken, Dutchess
Gary Preiser, Delaware
Irwin J. Rosenberg, Broome

Tellers

Elise H. Korman, Suffolk, *Chairman*
Jacob J. Barie, Orange
Hari P. Chakravorty, Putnam
Arnold D. Lurie, Nassau
Veronica Santilli, Kings

Sergeant at Arms

Henry J. Dobies, St. Lawrence, *Chairman*
Robert Bonvino, Richmond
John W. Cornwall, New York
Victor A. Panaro, NYS Chapter, American College of Radiology
C. Susan Rainwater, Steuben

Rules and Order of Business

Nathan P. Reed, Albany, *Chairman*
Ann C. Cea, Westchester
Alexander M. De la Garza, Queens
Richard John Dwane, Hospital Medical Staff Section
James J. Kropelin, Niagara

NOMINATING COMMITTEE

To the House of Delegates, Ladies and Gentlemen:

In accordance with Article XI, Section 2 of the Bylaws of the Medical Society of the State of New York, the Nominating Committee met at the Society's headquarters, 420 Lakeville Road, Lake Success, New York, on Thursday, October 2, 1986 at 10:30 a.m.

Members of the committee present were:
Member-at-Large—**Daniel F. O'Keeffe, M.D.**, Warren, *Chairman*
First District Branch—**Stanley Luftschein, M.D.**, The Bronx
Second District Branch—**Robert E. Fear, M.D.**, Suffolk
Third District Branch—**Thomas D. Pemrick, M.D.**, Rensselaer
Fourth District Branch—**Robert A. Reid, M.D.**, Warren
Fifth District Branch—**Richard D. Eberle, M.D.**, Onondaga
Sixth District Branch—**Jeffrey A. Ribner, M.D.**, Broome
Seventh District Branch—**James M. Flanagan, M.D.**, Wayne
Eighth District Branch—**James J. Kropelin, M.D.**, Niagara
Ninth District Branch—**Seymour R. Stall, M.D.**, Dutchess
Member-at-Large—**David M. Benford, M.D.**, New York

After careful consideration of the recommendations submitted by the district branches, the county medical societies, and the resident and student membership in response to a memorandum, dated June 9, 1986, and nominations from the floor, your committee respectfully nominated the following candidates for election on March 15, 1987:

President—**Samuel M. Gelfand, M.D.**, Nassau
President-Elect—**Charles D. Sherman, Jr., M.D.**, Monroe
Vice-President—**Joseph F. Shanaphy, M.D.**, Richmond
Secretary—**John H. Carter, M.D.**, Albany
Assistant Secretary—**George Lim, M.D.**, Oneida
Treasurer—**Morton Kurtz, M.D.**, Queens
Assistant Treasurer—**Robert A. Mayers, M.D.**, Westchester
Speaker—**Charles N. Aswad, M.D.**, Broome
Vice-Speaker—**Seymour R. Stall, M.D.**, Dutchess
Trustee (*one for five years*)—**Allison B. Landolt, M.D.**, Westchester
Councilors (*four for three years*)—**David M. Benford, M.D.**, New York
Duane M. Cady, M.D., Onondaga
William A. Dolan, M.D., Monroe
Robert E. Gordon, M.D., Kings
Student Councilor (one for one year)—**Michael Paciorek**, Onondaga
Resident Councilor (one for one year)—**Kathleen Squires, M.D.**, New York

Delegates to the American Medical Association for two years commencing January 1, 1988:
David M. Benford, M.D., New York
John H. Carter, M.D., Albany
Duncan W. Clark, M.D., Kings
Irwin J. Cohen, M.D., New York
James H. Cosgriff, Jr., M.D., Erie
G. Rehmi Denton, M.D., Albany
John A. Finkbeiner, M.D., New York
James M. Flanagan, M.D., Wayne
Samuel M. Gelfand, M.D., Nassau
Robert M. George, M.D., Oneida
Morton Kurtz, M.D., Queens
Allison B. Landolt, M.D., Westchester
Antonio F. LaSorte, M.D., Broome
George Lim, M.D., Oneida
David B. L. Meza, III, M.D., Orleans
Daniel F. O'Keeffe, M.D., Warren
Stuart Orsher, M.D., New York
Bernard J. Pisani, M.D., New York
Charles D. Sherman, Jr., M.D., Monroe
Seymour R. Stall, M.D., Dutchess
M. Theodore Tanenhaus, M.D., Kings
Victor J. Tofany, M.D., Monroe

The eleven nominees receiving the largest number of votes will be delegates; the second eleven will be alternates.

Respectfully submitted,
Daniel F. O'Keeffe, M.D., *Chairman*

HOUSE COMMITTEE ON BYLAWS

To the House of Delegates, Ladies and Gentlemen:

The members of the House Committee on Bylaws are as follows:

George Lim, M.D., Chairman	Oneida
Stanley F. Brunn, M.D.	Orange
Rodman D. Carter, M.D.	Otsego
Robert M. Kohn, M.D.	Erie
Sidney Mishkin, M.D.	Nassau
James R. Nunn, M.D.	Erie
Donna B. O'Hare, M.D.	New York
Hilda Ratner, M.D.	Queens
Stanley A. Steckler, M.D.	Suffolk
John A. Finkbeiner, M.D., President, ex officio	New York
John H. Carter, M.D., Secretary, ex officio	Albany
Charles N. Aswad, M.D., Speaker, ex officio	Broome
Donald F. Foy	Executive Vice President, ex officio
Robert J. O'Connor, M.D.,	Deputy Executive Vice President ex officio
Donald R. Moy, J.D.	General Counsel, ex officio

A meeting of the Reference Committee on Bylaws was held at the Hilton Hotel in New York City on March 7, 1986. The Reference Committee prepared a transcript of testimony and a summary of its recommendations to the House Committee on Bylaws. The input received from the meeting of the Reference Committee on Bylaws proved to be of great assistance to the House Committee on Bylaws when it met on November 6, 1986.

In all, four resolutions, asking for amendments to the Bylaws, were referred to the Committee for its consideration.

The following actions were taken with respect to the resolutions referred to the Committee:

Resolution 86-1, Exceptions to Deadline for Introduction of Resolutions

Introduced by Robert M. Kohn, M.D., Delegate, New York State Society of Internal Medicine

Whereas, There are currently no exceptions for specialty societies to introduce resolutions past the deadline of thirty days before the opening of the House of Delegates; and

Whereas, The AMA allows exceptions to its deadline for the introduction of resolutions from the resident physician section, the medical student section, the section on medical schools, and the hospital medical staff section, as well as a constituent society or a specialty society whose House of Delegates or comparable policy making body adjourns during one week preceding thirty days prior to commencement of a convention; and

Whereas, Resolution 85-62 which will be considered by the 1986 House of Delegates does not propose such exceptions; and

Whereas, Separate meetings of the resident and student membership over thirty days prior to a House of Delegates meeting would be costly; and

Whereas, The Medical Society of the State of New York utilized the AMA mechanism for the introduction of resolutions last year when its House of Delegates meeting occurred ten days prior to the AMA meeting; therefore be it

Resolved, That Article V, Section 2, paragraph 1 under the heading "Speaker" of the Bylaws of the Medical Society of the State of New York be amended by the following addition to the last sentence:

". . . with the following exceptions: the resident membership, medical student membership, the hospital medical staff section, and a component county medical or specialty society whose House of Delegates or comparable policy making body adjourns during or one week preceding thirty days prior to the annual meeting of the House of Delegates of the Medical Society of the State of New York may introduce resolutions at any time before the first day of the annual meeting of the House of Delegates."

Article V, Section 2, paragraph 1 under the heading "Speaker" would then read as follows: (additions italicized, deletions in brackets)

"The Speaker shall preside at all meetings of the House of Delegates. He shall appoint all committees to serve during the meeting of the House of Delegates at least thirty days in advance of the meeting. All resolutions submitted by component county medical societies, district branches, [scientific sections] *specialty societies*, and medical schools to be presented to the House of Delegates should be forwarded to the speaker at least forty-five days in advance of the annual meeting of the House and referred by him to the appropriate reference committees; and all resolutions sent in subsequent to forty-five days should be presented to the House and referred to the appropriate reference committees *with the following exceptions: the resident membership, medical student membership, the hospital medical staff section, and a component county medical or specialty society whose House of Delegates or comparable policy making body adjourns during or one week preceding thirty days prior to the annual meeting of the House of Delegates of the Medical Society of the State of New York may introduce resolutions at any time before the first day of the annual meeting of the House of Delegates.*"

NOTE: Last year, Resolution 85-62 was adopted, which amended the paragraph entitled "Speaker" under Article V, Section 2. In order to quote this paragraph as it now reads, the resolved portion of Resolution 86-1 should read as follows:

Resolved, That Article V, Section 2, paragraph 1 under the heading "Speaker" of the Bylaws of the Medical Society of the State of New York be amended by the following addition to the last sentence:

". . . with the following exceptions: the resident membership, medical student membership, the hospital medical staff section, and a component county medical or specialty society whose House of Delegates or comparable policy making body adjourns during or one week preceding thirty days prior to the annual meeting of the House of Delegates of the Medical Society of the State of

New York may introduce resolutions at any time before the first day of the annual meeting of the House of Delegates."

Article V, Section 2, paragraph 1 under the heading "Speaker" would then read as follows: (additions italicized)

"The speaker shall preside at all meetings of the House of Delegates. He shall serve on the Executive Committee with voice but without vote. He shall appoint all committees to serve during the meeting of the House of Delegates at least thirty days in advance of the meeting. All resolutions submitted by members of the House of Delegates (pursuant to Article III, Section 3, paragraph 3) to be presented to the House of Delegates shall be forwarded to the speaker at least thirty days in advance of the annual meeting of the House of Delegates and referred by him to the appropriate reference committees, *with the following exceptions: the resident membership, medical student membership, the hospital medical staff section, and a component county medical or specialty society whose House of Delegates or comparable policy making body adjourns during or one week preceding thirty days prior to the annual meeting of the House of Delegates of the Medical Society of the State of New York may introduce resolutions at any time before the first day of the annual meeting of the House of Delegates.*"*

The House Committee on Bylaws believes that an equitable procedure was adopted last year through the approval of Resolution 85-62. Pursuant to Resolution 85-62, Article III, Section 3 of the MSSNY Bylaws requires resolutions to be presented to the House of Delegates to be forwarded to the speaker at least thirty days in advance of the annual meeting. A procedure is provided whereby resolutions may be presented by a voting delegate any time prior to the final day of the annual meeting of the House of Delegates. In addition, a procedure is provided whereby resolutions of an emergency nature may be presented on the final day of the annual meeting. The House Committee on Bylaws does not believe it is necessary or prudent to expand the exceptions to the "thirty day" rule that are already included in Article III, Section 3.

The House Committee on Bylaws, therefore, recommends that Resolution 86-1 **NOT BE ADOPTED.**

Resolution 86-2, Full Time, Active Duty Military Physician Dues Structure

Introduced by Medical Society of Jefferson County

Whereas, Fort Drum, New York, located in Jefferson County, is one of the largest military installations in the United States and is the headquarters for the 10th Mountain Division, U.S. Army; and

Whereas, Currently six (6) full-time, active duty military physicians are stationed at Fort Drum and upwards of approximately twenty five (25) to thirty (30) military physicians will be stationed at Fort Drum within two years; and

Whereas, It is obviously of great importance to attract these physicians to our ranks as early as possible; therefore be it

Resolved, That the Medical Society of the State of New York establish a full-time, active duty military physician dues structure of one-half (½) of regular dues structure for those full time, active duty military physicians stationed in the State of New York; and be it further

Resolved, That Article XV "Dues" of the Bylaws of the Medical Society of the State of New York be amended by adding a new paragraph after paragraph five (5) to read: (additions italicized)

"Dues for full-time, active duty military physicians stationed in the State of New York will be one-half the amount levied on all other active members."

The House Committee on Bylaws believes that Article XV of the Bylaws already includes sufficient reduced dues categories. In addition, the MSSNY Bylaws provide for dues remission where a member shows hardship. It is believed that if reduced dues are provided for active duty military physicians, it will only be a matter of time before other classes of physicians who perceive themselves as having reduced or limited incomes will be calling for equal treatment.

The House Committee on Bylaws, therefore, unanimously recommends that Resolution 86-2 **NOT BE ADOPTED.**

Resolution 86-4, Reduced Dues for Members of Another General Medical Society With a National/State/County Structure

Introduced by New York County Medical Society

Whereas, Minority physicians who are members of another general medical society with a national, state and county structure appear to be under-represented in the Medical Society of the State of New York; and

Whereas, Many of these physicians are discouraged from participation because of the cost of membership in two medical societies; and

Whereas, It is the purpose of this resolution to devise a way to attract these under-represented physicians to membership; therefore be it

Resolved, That Article XV (Dues) paragraph 3 of the Bylaws of the Medical Society of the State of New York be amended to provide that physicians who are members of another general medical society with a national/state/county structure pay reduced dues based on the following formula: first year of membership $75; second year of continuous membership $150; third year of continuous membership $225; full dues fourth year of continuous membership. Amended Article XV, Paragraph 3 will now read (additions italicized):

"The dues and assessments of those active members who have been graduated from medical or osteopathic college not more than five calendar years, not counting active service in the armed forces of the United States or in the United States Public Health Service, *with the exception of physicians who are members of another general medical society with a national, state, county structure*, shall be one-third of the amount levied on all other active members. The dues and assessments of those active members who have not completed their continuous residency training shall be one-tenth the amount levied on all other active members. In no case may an active physician continue to pay such reduced dues more than seven years after the date of graduation from medical or osteopathic school, *with the exception, as noted above, of physicians who are members of another general medical society with a national/state/county structure who will pay reduced dues based on the following formula regardless of the date of graduation from medical or osteopathic school: first year of membership $75; second year of continuous membership $150; third year of continuous membership $225; fourth year of continuous membership full dues.* The dues and assessments of all active members entering practice for the first time, who are not otherwise eligible for reduced dues as set forth in prior provisions in this paragraph, shall be one half the amount levied on all other active members for the first two years of practice; full dues shall be paid after the first two years of practice. Once an active member pays full dues, he shall no longer qualify for half-dues status.

---

* Furthermore, for purposes of clarity, if Resolution 86-1 is adopted, the House Committee on Bylaws believes that the exceptions should be enumerated under Article III, Section 3, paragraphs 3–6.

The House Committee on Bylaws recognizes the good intentions of the resolution. There was much testimony heard at the meeting of the Reference Committee on Bylaws that efforts must be made to encourage members of other "general" medical societies to join the Medical Society of the State of New York. There is a strong consensus among those who testified at the Reference Committee on Bylaws meeting, the members of the Reference Committee and the members of the House Committee on Bylaws, however, that the lowering of Medical Society of the State of New York dues for members of other general medical societies should not be used as an inducement to get such individuals to join the Medical Society.

The House Committee on Bylaws, therefore, unanimously recommends that Resolution 86-4 **NOT BE ADOPTED.**

Resolution 86-9, Past Executive Vice-President of the State Society as a Member of the House of Delegates for Life

Introduced by the Council

Whereas, Any member of the Medical Society of the State of New York who has served as executive vice-president of the State Society has gained valuable experience in dealing with the policies and issues concerning the medical profession; and

Whereas, This experience can be of great value to the House of Delegates of the Medical Society of the State of New York in its deliberations; therefore be it

Resolved, That the Bylaws of the Medical Society of the State of New York be amended to provide that any past executive vice-president of the Medical Society of the State of New York shall be a member of the House of Delegates of the State Society for life, provided that the individual is a member of the Medical Society of the State of New York, resides in the State of New York, and is not otherwise a member of the House of Delegates for life in accordance with this section; and be it further

Resolved, That Article III, Section 1 of the Bylaws of the Medical Society of the State of New York be amended by adding a new clause to read:

"(h) any past executive vice-president of the State Society who shall be a member for life, provided that individual is a member of the Medical Society of the State of New York, resides in the State of New York and is not otherwise a member of the House of Delegates for life in accordance with this section."

Article III, Section 1 shall then read: (additions italicized): "The House of Delegates shall be composed of: (a) duly designated delegates from the component county medical societies; (b) officers of the Medical Society of the State of New York, councilors, and trustees; (c) a duly designated delegate from each district branch; (d) a duly designated delegate from each recognized specialty society; (e) duly designated delegates from the student membership; (f) the Commissioner of Health of the State of New York or a deputy designated by him, provided that any representative shall be a member of the State Society; (g) past-presidents of the State Society and any past president of the American Medical Association, provided that individual is a member of the Medical Society of the State of New York who shall be members for life; (*h*) *any past executive vice-president of the State Society, who shall be a member for life, provided that individual is a member of the Medical Society of the State of New York, resides in the State of New York and is not otherwise a member of the House of Delegates for life in accordance with this section;* (i) a representative from each of the medical schools in New York State, provided said representative is a member of the Medical Society of the State of New York; (j) a delegate representing the resident membership; (k) a delegate representing the hospital medical staff section and; (l) elected officers, trustees, and speakers of the American Medical Association, provided those individuals are members of the Medical Society of the State of New York."

The House Committee on Bylaws believes that this resolution allows the House of Delegates to retain the experience and expertise of any past executive vice-president of the State Society who is a member of the State Society.

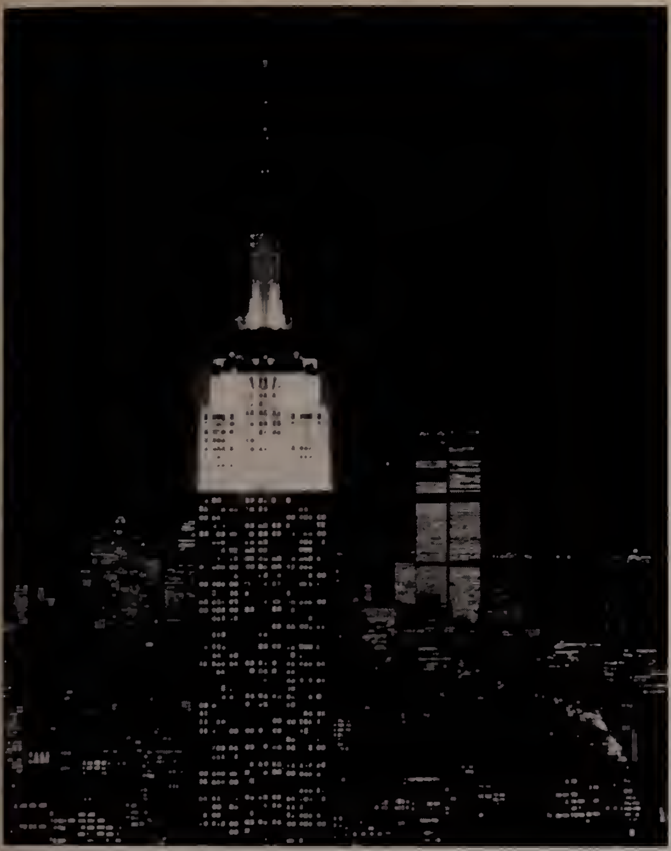
Accordingly, the House Committee on Bylaws unanimously recommends that Resolution 86-9 **BE ADOPTED.**

Your chairman would like to thank, in addition to the members of the Committee and the physicians who testified before it, those members of staff who have helped this Committee; Donald R. Moy, J.D., General Counsel, Ellen Panzeca, J.D., Legal Assistant, and Ms. Kathleen Curran for her secretarial assistance.

Respectfully submitted,

George Lim, M.D., *Chairman*

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807



COURTESY N.Y. CONVENTION & VISITORS BUREAU

Medical Society of the State of New York

ANNUAL CME ASSEMBLY

Clinical Dilemmas & Controversies

March 13–15, 1987

and the

Annual Meeting of the House of Delegates

March 12–15, 1987

The New York Hilton
New York City

HIGHLIGHTS . . .

- 22 Sessions
- Panel Discussions
- Technical Exhibits
- Two Receptions
- President's Reception & Dinner Dance

CLIP & MAIL

Director of Front Office Operations
The New York Hilton
1335 Avenue of the Americas
New York, N.Y. 10019

Phone: 212-586-7000

for hotel use only

NAME ____________________ TITLE ____________

FIRM ____________________ PHONE ____________

STREET ________________________________

CITY/STATE/ZIP ________________________________

REMARKS ________________________________
(please print)

NOTE: Reservations must be received no later than February 19, 1987 and will be held only until 6 p.m. on day of arrival unless later hour is specified.

please print

ARRIVAL DATE ____________________

☐ 2-4 p.m. ☐ 4-6 p.m. ☐ 6-8 p.m. ☐ after 8 p.m.

DEPARTURE DATE ____________________
(checkout time is 1 p.m.)

HAVE A CREDIT CARD? You can register at our QUICK CHECK desk. If you wish to take advantage of this credit card PAYMENT GUARANTEED reservation, please complete the information below.

☐ AMERICAN EXPRESS
☐ DINERS CLUB
☐ CARTE BLANCHE

Expiration Date ____________

____
Credit Card No.

Signature ____________________________

MEDICAL SOCIETY OF THE STATE OF NEW YORK
Annual Meeting of the House of Delegates
March 12–15, 1987
Annual CME Assembly
March 13–15, 1987

Circle Preferred rate:

Singles:	$130	$145	$155
Doubles/Twins:	$155	$170	$180
Executive Tower:	$200 Single/Double		
Suites: (P&1):	$360	$395	
Suites: (P&2):	$460	$495	

Rates subject to 8¼% N.Y. State sales tax; $2.00 per room per night, and 5% N.Y.C. occupancy taxes. (If room at the rate requested is unavailable, one at the nearest available rate will be reserved.)

GENERAL INFORMATION

All physicians, members of the allied professions, and their guests are invited to attend the Medical Society of the State of New York's Annual CME Assembly to be held March 13–15, 1987, at the New York Hilton. The two and one-half days of meetings will provide the individual physician with a possible 18 credits out of a total of 93 Category 1, CME credit hours being offered. The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians. Over 125 outstanding speakers, including a number from out-of-state, will present papers at 20 sessions.

The theme of the CME Sessions will be "Clinical Dilemmas and Controversies." A few of the subjects to be presented will be, Terrorism & Medicine; Computers in Medicine; AIDS in Pregnancy; Humanism and Medical Ethics in Our Technological Age; Intensive Care for AIDS Victims; Thoracic Aortic Balloon Occlusion: Methods and Indications; and the Role of the Physician as the Patient's Advocate. For details, see the Preliminary Program which appears in the December 1986 issue of the *New York State Journal of Medicine*. Our program will offer the clinician timely approaches in diagnosis and treatment of diseases in a wide range of medical specialties.

Here are comments from some of the attendees at the 1986 CME Assembly: "The sessions were educational, and the topics were timely," "There was an excellent choice of qualified speakers," "I found the material was practical, concise and clinically useful." Don't miss this opportunity to gain category 1 credits over a weekend when you are free from office hours. Also, bring your family to New York City to enjoy the sights. There is no registration fee for MSSNY members or members of any other state medical society.

Professional representatives from more than 60 companies will be in the Rhinelander Gallery Exhibit Hall on Friday and Saturday to discuss their products and services. The Technical exhibits will include drug products, office management systems, medical and surgical suppliers, health insurance plans, pension plans and more.

SPECIAL EVENTS

Thursday, March 12

The Annual Dinner Dance, in honor of our President, John Finkbeiner, M.D., will be held in the Trianon Ballroom of the New York Hilton. The reception will be held in the Mercury Ballroom starting at 7:00 p.m. All attendees are welcome. Tickets are $75.00 per person and should be ordered in advance from the Medical Society.

UNITED NATIONS HEADQUARTERS — *Courtesy N.Y. Convention & Visitors Bureau*

Friday, March 13 and Saturday, March 14

A reception for physicians, their guests, the allied professions and exhibitors will be held both days in the Exhibit Hall between 5:00 p.m. and 6:00 p.m. A free drawing for a color TV set will take place at both receptions.

HOTEL RESERVATIONS

Physicians must make their own reservations with the New York Hilton no later than February 19, 1987. After that date, rooms are on an availability basis only. If making reservations by telephone, please mention that you are attending the Medical Society Convention for billing at the special rate.

REGISTRATION

CME Registration will take place in the Exhibit Hall on Friday, March 13, from 8:00 a.m. to 5:30 p.m., and Saturday, March 14 from 8:30 a.m. to 5:30 p.m., and on Sunday, March 15, registration will take place on the 2nd floor Promenade from 8:30 a.m. to 12:30 p.m.

House of Delegates registration will take place on the 2nd and 3rd floor Promenades at the following hours: Wednesday, March 11, from 12 Noon to 6:00 p.m.; Thursday, March 12, Friday, March 13, and Saturday, March 14, from 8:30 a.m.–5:30 p.m.; and Sunday, March 15, from 8:30 a.m. to 12 Noon. All MSSNY members are invited to attend the meetings.

CME MEETING HOURS

Friday, March 13 —	9am – 12:30pm
	2pm – 5pm
Saturday, March 14	10am – 1pm
	2pm – 5pm
Sunday, March 15	10am – 1pm

EXHIBIT HOURS

Friday, March 13 —	9am – 6pm
Saturday, March 14	9am – 6pm

1987 ANNUAL CME ASSEMBLY

Medical Society of the State of New York
The New York Hilton, New York City

Clinical Dilemmas and Controversies

FRIDAY, MARCH 13

9:00 a.m. - 1:00 p.m. Plenary Session: Clinical and Ethical Dilemmas, *Gramercy A & B*

2:00 p.m. - 5:00 p.m. Chest Diseases, *Regent Parlor*
Cardiovascular Diseases, *Murray Hill B*
Pediatrics/Orthopedic Surgery, *Murray Hill A*
Neurology/Alcoholism/Physical Medicine & Rehabilitation, *Nassau A*
Emergency Medicine/Critical Care/Otolaryngology, *Gramercy B*
Medical Liability, *Gramercy A*

SATURDAY, MARCH 14

10:00 a.m. - 1:00 p.m. Internal Medicine (All Day), *Regent Parlor*
Ophthalmology (All Day), *Sutton Parlor Center*
Psychiatry (All Day), *Murray Hill B*
Hand Surgery/Orthopedic Surgery/Physical Medicine & Rehabilitation (All Day), *Murray Hill A*
Urology/Pathology, *Nassau A*
Dermatology, *Nassau B*

2:00 p.m. - 5:00 p.m. Internal Medicine, *Regent Parlor*
Ophthalmology, *Sutton Parlor Center*
Psychiatry, *Murray Hill B*
Hand Surgery/Orthopedic Surgery/Physical Medicine & Rehabilitation, *Murray Hill A*
Computers in Medicine, *Madison Suite*
Terrorism and Medicine, *Sutton Parlor South*

SUNDAY, MARCH 15

10:00 a.m. - 1:00 p.m. Allergy and Immunology, *Murray Hill B*
General Surgery/Radiology/Anesthesiology, *Murray Hill A*
Obstetrics and Gynecology/Women's Medical Society of New York State, *Gramercy B*
Family Practice/Orthopedic Surgery, *Nassau A*
Neurosurgery, *Morgan Suite*

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis. The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians.

◆

Technical Exhibits

The Brooklyn Bridge (foreground) and the Manhattan Bridge spanning the East River

Advance Registration Form

1987 Annual CME Assembly, March 13–15
The New York Hilton, New York City

Complete and return this form to the Medical Society address as indicated below.

Name______________________________
PLEASE PRINT LAST FIRST

Address______________________________

City/State Zip______________________________

Medical Specialty______________________________

REGISTRATION FEE *Please check categories*

☐ Non-Member Physician* $50.00
☐ Allied Profession $10.00

☐ Nurse
☐ Physician Assist.
☐ Dentist

☐ Lab Technician
☐ PT/OT
☐ Other__________ *Specify*

NO FEE *Please check category*

☐ Active Member*
☐ MSSNY Life Member
☐ Resident/Intern

☐ Medical Students
☐ Medical Assist.
☐ Speaker

☐ Spouse/Family
☐ News Media/Medical Writer

REMITTANCE ENCLOSED $__________

* of any state medical society

Please make check payable to:
Medical Society of the State of New York, and return this form with remittance to: Division of Meeting Services/MSSNY, 420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042.

Important: Your ID Badge will be held at the MSSNY Registration Desk located in the Exhibit Hall, on the second floor of the New York Hilton.

SW, Suite 300 East, Washington, DC 20024-2588. Tel: 1-800-533-8811.

Feb 7–14. **4th Annual Office Based Sports Medicine.** Park City. Info: University of California, San Francisco, Extended Programs in Medical Education, Room 569-U, San Francisco, CA 94143. Tel: (415) 476-4251.

Feb 22–27. **Diagnostic Imaging: Update 1987.** 23½ Cat 1 Credits. Park City. Info: Radiology Postgraduate Education, University of California, Third & Parnassus Ave, Room C324, San Francisco, CA 94143. Tel: (415) 476-5731.

AROUND THE WORLD

AUSTRALIA/NEW ZEALAND

Feb 9–25. **Imaging Solutions to Clinical Problems.** 25 Cat 1 Credits. Aboard the Royal Odyssey—Destination Australia and New Zealand. Classes at ports of call in Australia, New Zealand, and Tasmania. Info: Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (414) 641-9773.

INDIA

Feb 22–27. **2nd International Conference on Surgery in the Tropics.** Bombay. Info: Dr D. S. Saksena, General Secretary, Second International Conference on Surgery in the Tropics, Bombay Hospital Institute of Medical Sciences, Bombay Hospital Ave, Bombay, 400 020, India. Tel: 297100/296500. Cable: SWASTHYA.

MEXICO

Feb 23–28. **Neuroradiology and Head and Neck Radiology, Computed Tomography and Magnetic Resonance Imaging.** Acapulco Princess Hotel, Acapulco. Info: Betty Frost, Program Coordinator, Continuing Education Office, SUNY Health Science Center at Syracuse, 750 East Adams St, Syracuse, NY 13210. Tel: (315) 473-4606.

SWITZERLAND

Feb 8–15. **Current Topics on Infectious Diseases.** 24 Cat 1 Credits. Conference Center in Davis. Info: Continuing Edu-

(*continued on p 18A*)

Medical Society of the
State of New York

Annual Dinner Dance
in honor of
John A. Finkbeiner, M.D.
President

Thursday
March 12, 1987
Trianon Ballroom
The New York Hilton
53rd Street & Avenue of Americas

Cocktail Hour
7 P.M.

Superlative
Dinner

Subscription $75.00 per person

Reservation Form

Make check payable to:
Medical Society of the State of New York
Attn: Div. of Management & Meeting Services
420 Lakeville Road, P.O. Box 5404
Lake Success, New York 11042

Please send me ______ tickets for the
Annual Dinner Dance on March 12, 1987
Check enclosed for $________

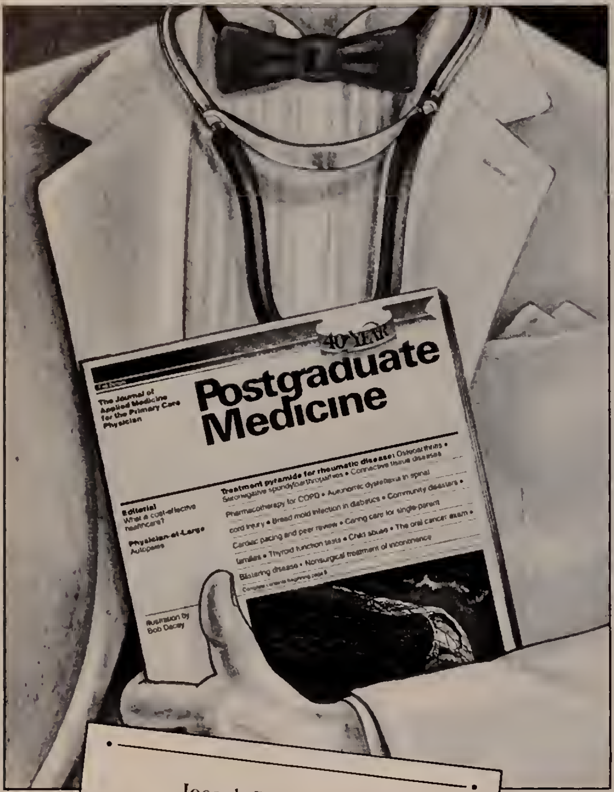
PLEASE NOTE: Reservations and ticket sales will close at 12 Noon on Thursday, March 12, one hour before the House Convenes.

Name____________________

Address____________________

____________________Zip____________

FAMOUS PHYSICIANS ARE SHOWING UP FOR OUR 40th YEAR CELEBRATION.


40 YEAR
Postgraduate Medicine
The Journal of Applied Medicine for the Primary Care Physician
Editorial
Physician-at-Large
Treatment pyramid for rheumatic disease
Illustration by Bob Dacey

Joseph B. Kirsner, M.D.
Donald E. DeWitt, M.D.
W. Jack Stelmach, M.D.
Robert E. Rosenthal, M.D.
James D. Mills, M.D.
Thomas J. McDonald, M.D.

Read Every Issue From Cover to Cover:

January
Symposium: Biliary Tract Disease
Special Article: Gastroenterology 1947-1987 . . . . Joseph B. Kirsner, M.D.

February 1
Symposium: Focus on Family Practice . . . . Donald E. DeWitt, M.D. coordinator
Special Article: Family Practice 1947-1987 . . . . W. Jack Stelmach, M.D.

February 15
Symposium: Emergency Medicine . . . . Robert E. Rosenthal, M.D. coordinator
Special Article: Emergency Medicine 1947-1987 . . . . James D. Mills, M.D.

March
Symposium: Otolaryngology in Primary Care . . . . Thomas J. McDonald, M.D. coordinator
Special Article: Otolaryngology 1947-1987 . . . . Thomas J. McDonald, M.D.

---

40 YEAR
Postgraduate Medicine

Every Issue is a Celebration of Clinical Information

AN APPROACH TO LYMPHOMAS FROM BOTH SIDES OF THE ATLANTIC

Dr. Karl Lennert (Kiel, W. Germany) • Dr. Philip Lieberman (New York, U.S.A.)

AN UPDATE ON SOCIO-ECONOMIC MATTERS FOR PATHOLOGISTS

2-DAY WEEKEND SLIDE SEMINARS & LECTURES

N.Y. State Society of Pathologists
Nassau County Society of Pathologists, Garden City, L.I. N.Y.

FRIDAY-SUNDAY MARCH 13-15, 1987 ($125 Including Lunch)

FOR INFORMATION: Nathan S. Taylor, M.D., Pathology Dept., Rochester General Hospital, Rochester, NY 14621, (716) 338-4121.

March-October 1987 Tues. Eves. 4-7 p.m.

EMERGENCY MEDICINE BOARD REVIEW #312

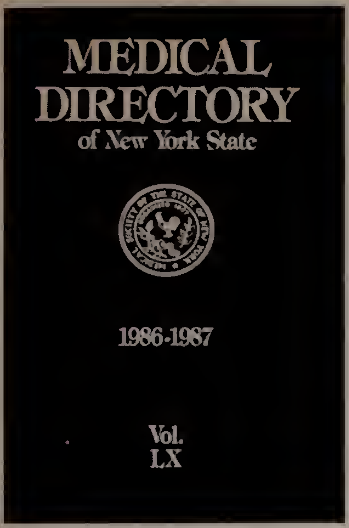
NYU Medical Center

TUITION: $1200
Accreditation: 63 AMA Category I credit hours.
Pending: 63 AAFP and 63 ACEP credit hours.

For New York metropolitan area physicians: Twenty one weekly 3-hour sessions spread over a 21 week period to prepare for the written portion (Part I) of the Emergency Medicine Board Examination, plus two, 2-hour ("1 on 1") patient management encounters for each registrant that simulates and prepares candidates for the oral portion (Part II) of the Board Examinations.

Information available on #315 EMERGENCY MEDICINE, May 11-14, 1987 Monday-Friday

FOR FURTHER INFORMATION: NYU Post-Graduate Medical School, 550 First Avenue, New York, N.Y. 10016 (212) 340-5295 (24-hour service)



ANNOUNCING THE 1986-87 Edition of the MEDICAL DIRECTORY of New York State

Published biennially, by the Medical Society of the State of New York, this important 920 page, reference source is now available.

The Directory lists over 27,000 New York State Physicians, both members of the Medical Society, and non-members who chose to be listed for a fee. It includes the following data.

SAMPLE LISTINGS

★**FARNSWORTH, Peter Birger,** 420 Lakeville Rd 11042. Tel 488-6100. MCGILL 59, D-P (PDC). Am Ac Ped, Dir Div Scientific Activities Med Soc SNY. Also North Salem (AMA)

★**FINEBERG, Henry Irwin,** 420 Lakeville Rd 11042. Tel 488-6100. YALE 27. Am Thor. Am Pub Hlth, Ac Med. Exec Vice Pres Emerit Med Soc SNY. Also Dix Hills.

★**LAWRENCE Jr, George J,** 420 Lakeville Rd 11042. Tel 488-6100. PENN 38. D-OG. SL. FACOG. FACS. Exec Vice Pres Emerit Med Soc SNY. Hon Chair. O&G Flushing. Also Glen Cove (AMA)

★**O'CONNOR, Robert James,** 33 Valencia Ave 10301. Tel 447-7242. STATE UNIV BKLN. D-OG SL. FACOG. FICS. Am Abdominal Sur. Am Ac Comp Med. Attd O&G Staten Isl. Cons O&G USPHSH SI. Attd Gyn Sea View. Also Lake Success. (MEM-AMA)

□

FLUSHING HOSPITAL AND MEDICAL CENTER

45th Ave. and Parsons Blvd. Flushing. 11353. Tel 6705000. Voluntary, General. 424 Beds. Maintains Registered School of Nursing. Exec. Dir. Philip A. Palese, Dep. Exec. Dir. Michael Marigliano, Asso. Dir. Gen. Serv. Morton Beckmen, Asso. Dir. Nursing Serv. Mrs. Mary G. Gamer, R.N., Asso. Dr. Fiscal Serv. Alen Currier, Dir. Corporate and Med. Stsff. Serv. Patricia DelGiorno.

Emeritus Staff

DENTISTRY—L. Schwartz
MEDICINE—J. P. Burghardt, E. J. Carey, B. A. Glass, J. Iraci, S. Iraci, H. Kalenderian, P. J. Lacovara, T. Nyilas, L. Richeimer, W. Seitz.
  ALLERGY—A. Notaro.
  ENDROCRINOLOGY—C. Delit.
  GASTROENTEROLOGY—A. Weiss.
  NEUROLOGY—S. Karlan
OBSTETRICS & GYNECOLOGY—E. Kiss, V. A. PArnell, M. Roland.

- Cities, towns and counties in which the physicians practice
- Hospital affiliations, Fellowships, and Board certifications
- Medical School and date of graduation
- Up to three office addresses and telephone numbers
- Separate list of hospitals with medical staffs identified.

Of course, the Directory is distributed free to members of the State Medical Society who will automatically receive the Directory when published. A special price of $40 per copy is offered to non-member physicians who chose to be listed, and to all others the price is $70 per copy. The Directory is the most comprehensive, up-to-date desk reference source on physicians in New York State, and is constantly utilized by physicians, libraries, and others in the health care field.

MEDICAL DIRECTORY ORDER FORM for non-members, or members requesting additional copies. (1986-87 Edition)

Name____

Address____

City/State/Zip____________________________________

Please send me the following:

( ) copies @ $40 $__________
(*Limit three copies per listed physician*)
Plus applicable sales tax __________
Total enclosed $__________

( ) copies @ $70 $__________
Plus applicable sales tax __________
Total enclosed $__________

Make check payable to: Medical Society of the State of New York, and mail to: Accounting Dept. Medical Society of the State of New York, 420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042.

4th Annual International
Breast Cancer Seminar
Presented in Cooperation with
the American Cancer Society

MIAMI
March 12 - 14, 1987
HOTEL INTER•CONTINENTAL MIAMI

Join 15 leading specialists from all over the world at this post graduate seminar. They'll be addressing the latest developments in the diagnosis and treatment of all forms of breast disease.

Participants Include:
ITALY: **Umberto Veronesi, M.D.** Director N.C.I., Milan
ENGLAND: **John Hayward, M.D.** Director Imperial Cancer Research, London
JAPAN: **Rikiya Abe, M.D.** Prof. of Surgery, Fukushima Medical College
FRANCE: **Jean Yves Petit, M.D.** Chief Reconstructive Breast Surgeon, Institute Gustave Roussy
CHINA: **Zhen Zhou Shen, M.D.** Chief Breast Surgeon, Cancer Hospital, Shanghai
U.S.A.
Marc Lippman, M.D. Chief Med. Breast Cancer Section, N.C.I., Bethesda
David Kinne, M.D. Chief Breast Service, Memorial Sloan-Kettering, N.Y.
Jay Harris, M.D. Clinical Director Radiation Therapy, Harvard Med. School
Alfred Ketcham, M.D. Prof. of Surgery and Oncology, University of Miami
Arthur Donovan, M.D. Prof. of Surgery USC School of Med. Los Angeles
Barbara Danoff-Fowble, M.D. Assistant Prof. Radiation Therapy, Fox Chase Cancer Center, Philadelphia
Jeffery Schlom, PhD. Chief Tumor Immunology & Biology, N.C.I., Bethesda
Raphael S. Good, M.D. Prof. Psychiatry & O.B. Gyn. University of Miami
Robert Gale, M.D. Assistant Prof. of Medicine, UCLA, Los Angeles
C.D. Haagensen, M.D. Prof. Emeritus Clinical Surgery, Columbia University

Approved for 15 hours of Category I Credit.

For more information on the **Fourth Annual International Breast Cancer Seminar**, mail to: **MIAMI CANCER CONFERENCE, INC.**
P.O. Box 56-2110
Miami, Florida 33256
(305) 557-1600

Name ______________________

Address ______________________

City/State/Zip ______________________

Registration Fee $325

FIGHT CANCER. EAT YOUR VEGETABLES.

There's strong evidence your greengrocer has access to cancer protection you won't find in any doctor's office.

Like broccoli, peaches, spinach, tomatoes, citrus fruits and various other types of fruits and vegetables. They may help reduce the risk of some forms of cancer.

Write for more information.

AMERICAN CANCER SOCIETY®

cation S/P, University of California, Box 0446, San Francisco, CA 94143-0446.

VIRGIN ISLANDS

Feb 16–18. **Management of the Postmenopausal Patient.** *Feb 19–21.* **Antenatal Fetal Assessment: State of the Art.** Frenchman's Reef, St Thomas. Info: ACOG Registrar, 600 Maryland Ave, SW, Suite 300 East, Washington, DC 20024-2588. Tel: 1-800-533-8811.

Added Control For America's Coughs

The expectorant action of guaifenesin plus the Advantage of Codeine!

Robitussin and codeine. Each 5 ml (*1 teaspoonful*) contains: Guaifenesin, USP, 100 mg; Codeine Phosphate, USP, 10 mg (*warning: may be habit forming*); alcohol, 3.5 percent; in a palatable, aromatic syrup

A·H·ROBINS

Pharmaceutical Division, Richmond, Virginia 23261-6609

©A.H. Robins Company 1985

Robitussin A-C® Ⓒ

MEDICAL & SURGICAL PROBLEMS IN WORKERS' COMPENSATION

Sponsored by: The American Academy of Legal & Industrial Medicine, Inc.
(Formerly the American Academy of Compensation Medicine, Inc.)

Co-sponsored by: New York University Post-Graduate Medical School

March 18-19, 1987, Sheraton Centre Hotel, 811 Seventh Ave. @ 53rd St., NYC, NY

Accreditation: As an organization accredited for continuing medical education, the New York University Post-Graduate Medical School certifies that this continuing medical education offering meets the criteria for 14 credit hours in Category I of the Physician's Recognition Award of the American Medical Association. The New York University Post—Graduate Medical School is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to sponsor continuing medical education for physicians.

WEDNESDAY, MARCH 18TH:	THURSDAY, MARCH 19TH:
Assessment and Management of Pain Moderator: Mathew HM Lee, MD	**Disability Evaluation** Moderator: Robert Katz, MD
Illicit Drugs in Industry Moderator: Peter Aldin, MD	**Industry-Induced Illness** Moderator: Morton Gluck, MD

***For information and application please write:* Mathew H. M. Lee, M.D., F.A.C.P., Program Chairman, American Academy of Legal & Industrial Medicine, Inc., P.O. Box 10, Island Station, Roosevelt Island, NY 10044. Phone (212) 750-6781.**

MISCELLANEOUS—CONT'D

HAVE YOU RECEIVED AN INQUIRY FROM THE OFFICE OF PROFESSIONAL MEDICAL CONDUCT? For consultation, contact Susan Kaplan, Attorney-at-Law, (212) 877-5998. Practice limited to the crucial legal problems directly affecting your license to practice medicine—with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professions), and as an Assistant District Attorney in Nassau County. Susan Kaplan, Esq., 165 West End Avenue, Suite 27P, New York, N.Y. 10023.

COMPUTERIZE YOUR BILLING—Remarkably easy-to-use software automatically prints bills, statements, insurance forms. Menu-driven. Reports ageing balances, referral sources, income by time period, procedure & more. Installs automatically. IBM and compatibles. Solo practice only $399. Group practice, only $499. Free information. Demonstration disk with complete manual, $10. Call or write REM Systems, Inc., Dept. NY1, 281 Dug Hill Road, Brownsboro, AL 35741 (205) 534-3457. VISA, MC Accepted.

50% OFF PREVIOUSLY OWNED MEDICAL—Laboratory, x-ray, ultrasound equipment in excellent condition. We buy, sell, broker, repair. Ask about our Holter Scanning Services. Call anytime: Medical Equipment Resale, 24026 Haggerty, Farmington Hills, MI 48018. (313) 477-6880.

MISCELLANEOUS—CONT'D

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta, GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

WEIGHT LOSS TUTORIAL, NO FEE for physicians and their staff. Individual help in setting up a medically documented, effective, economical, uncomplicated weight loss program in your office practice. Contact Allan Lazar, M.D., 420 Grand Avenue, Englewood, N.J. 07631. (201) 568-3742.

EXPERIENCED MEDICAL DISCIPLINARY ATTORNEY. Litigator/trial attorney experienced in representing physicians at hearings before the Department of Health and the Office of Professional Medical Conduct. Michael S. Kelton, Esq., c/o Lippman & Krasnow, P.C., 475 Fifth Avenue, Suite 1212, New York, N.Y. 10017, telephone number (212) 532-0710.

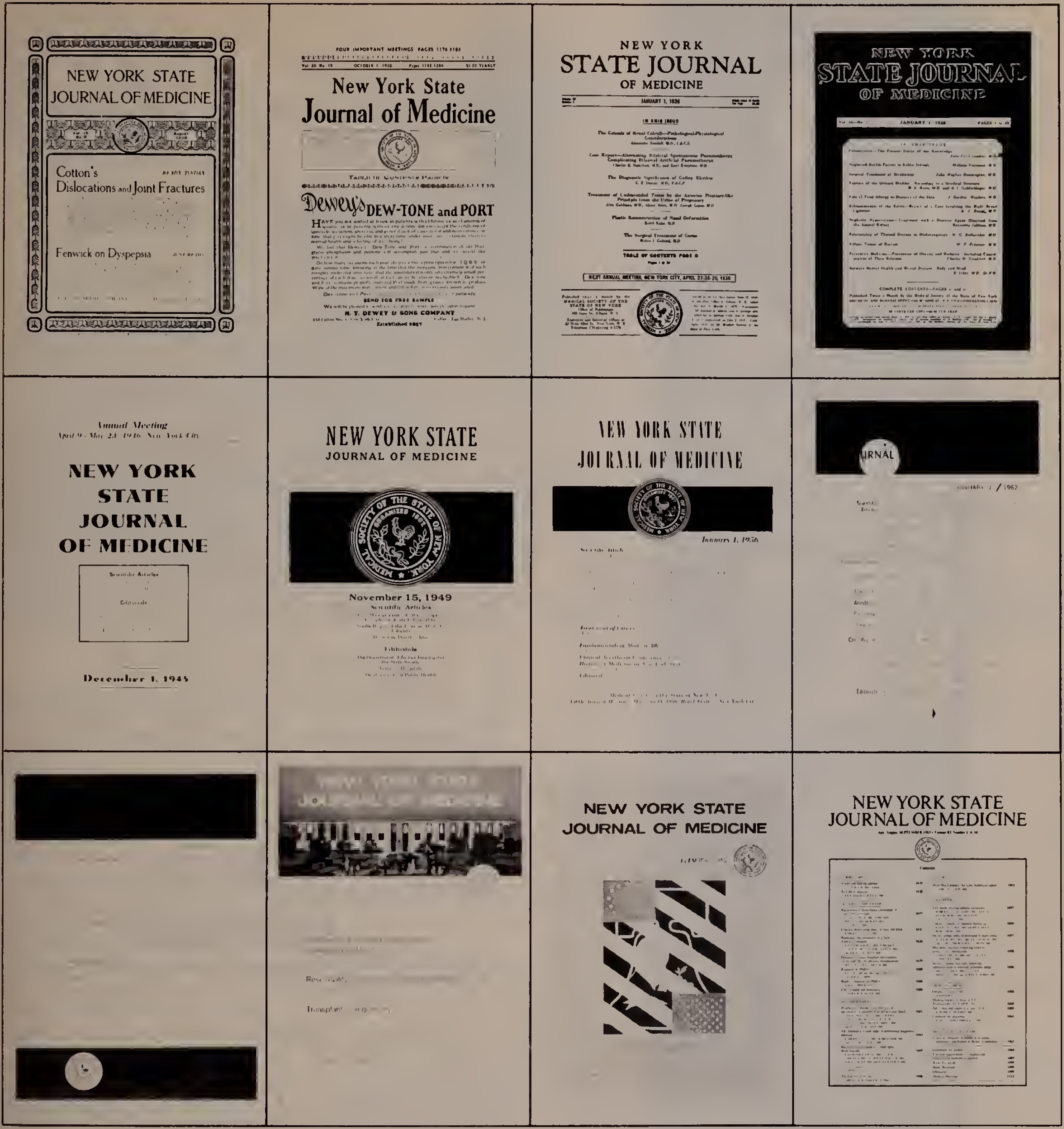
DOCTOR, DO YOU NEED AN EQUALIZER? Are Medicare and third party claims and payments giving you the blues? Are your letters lost, calls ignored, and EOBs imply incompetence/dishonesty? Whether you're participating or not let T.P.E. save your time, staff and sanity. Former Medical Director & Attorney for Empire Blue Cross Blue Shield/Medicare will be your Third Party Equalizers! Call T.P.E. at (516) 764-2800 today!

MISCELLANEOUS—CONT'D

PEYRONIE'S DISEASE—Wanted to consult a specialists in treatment, in Metropolitan area. Contact Dept. #429 c/o NYSJM.

PROFESSIONAL MISCONDUCT ATTORNEYS. William L. Wood Jr., formerly Executive Director of the New York State Office of Professional Discipline and Anthony Z. Scher, formerly Director of Prosecutions, announce the opening of their law office, Wood & Scher. Our recent tenure as chief enforcement officers has given us experience which will allow us to represent physicians in professional misconduct proceedings, malpractice licensure, license restoration, controlled drug proceedings, reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practitioners. Wood and Scher, Attorneys at Law, One Chase Road, Scarsdale, New York 10583. Telephone 914-723-3500.

Cover to cover . . . . always good reading.



In 1987, the *New York State Journal of Medicine* promises to provide timely and stimulating commentary, challenging research papers, analysis of clinical trends, debate on topical issues, and in-depth coverage of medical news. The journal will make a welcome gift to a colleague or student in the United States or abroad.

A free copy of the July 1985 issue, a special issue on smoking, will be included with each subscription while the supply lasts.

SUBSCRIPTION ORDER FORM Clip and mail

Circulation Department
New York State Journal of Medicine
420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042

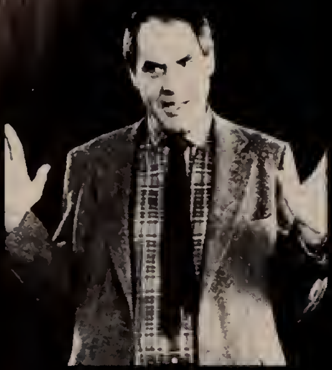
Name____

Address______________________________________

____

	1 Year	2 Years	3 Years
U.S. & Canada	☐ $25	☐ $45	☐ $60
Foreign	☐ $30	☐ $55	☐ $75

Payment required with order.



TAKE CARE OF YOUR LUNGS.

THEY'RE ONLY HUMAN.

I'm not the old Robert Klein you know and love. I'm the new Robert Klein. I gave up smoking and I feel like a new man. My doctor asked me to take a deep breath and I vacuumed half his office! Seriously, smoking does damage your lungs with each and every puff. I may be crazy, but I'm not crazy enough to keep smoking. I quit and so can you. You'll see!

Robert Klein

AMERICAN LUNG ASSOCIATION®
The Christmas Seal People®

Doctor . .

Do you, or a physician you know, have a problem with alcohol, drugs, or emotional illness?

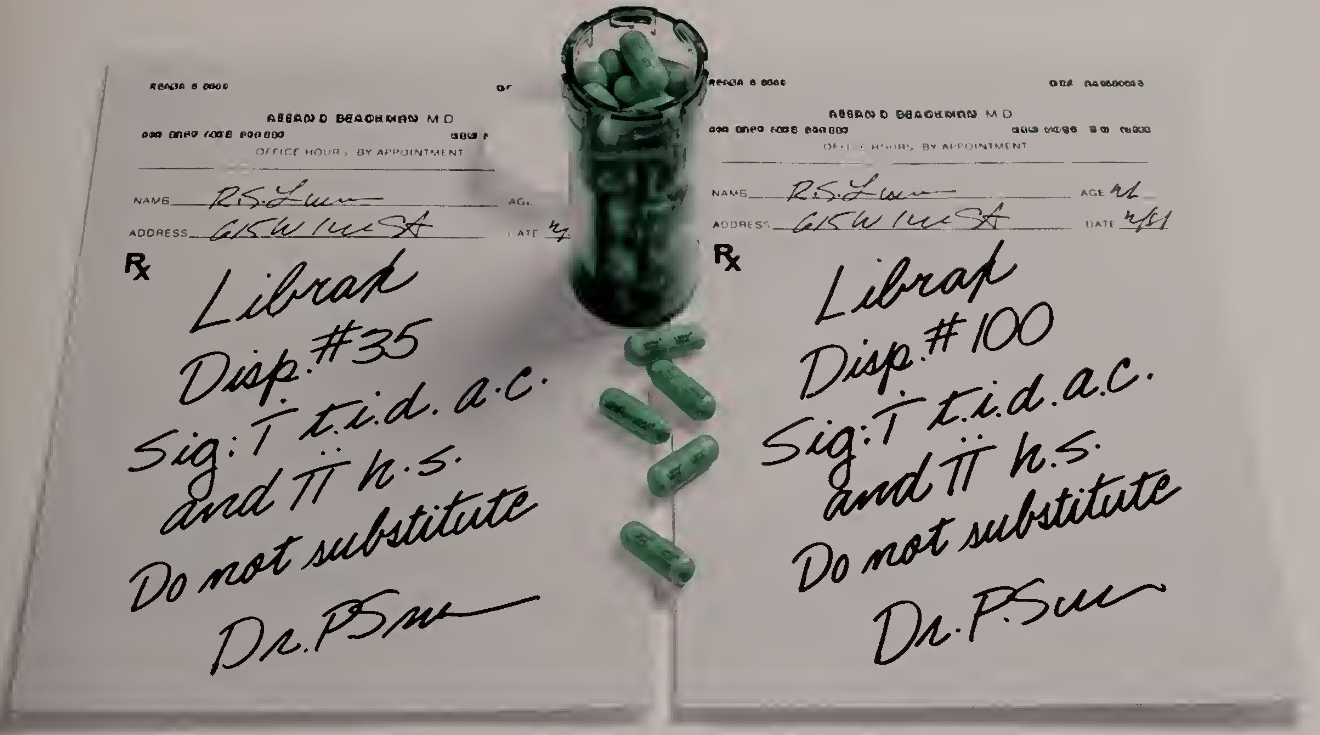
If so, please contact the Medical Society of the State of New York's Committee for Physicians' Health, whose function is to assist, *confidentially.*

Telephone: 516-488-7777.

Index to Advertisers

St. Clare's Hospital 7A
Classified Advertising 6A, 16A, 20A
Eli Lilly and Company 10A
Marion Laboratories 1A, 2A
Physician's Reciprocial Insurers 8A
Postgraduate Medicine 16A
A. H. Robins Company 9A, 19A
Roche Laboratories .... 4A, 5A, 3rd & 4th Cover
Upjohn Company .... 2nd Cover

SPECIFY **LIBRAX**®



OFFICE HOURS, BY APPOINTMENT

NAME R.S. Lum
ADDRESS 615 W 1..St

℞ Librax
Disp. #35
Sig: i t.i.d. a.c.
and ii h.s.
Do not substitute
Dr. P.Sm

OFFICE HOURS, BY APPOINTMENT

NAME R.S. Lum AGE 41
ADDRESS 615 W 1..St DATE 4/81

℞ Librax
Disp. #100
Sig: i t.i.d. a.c.
and ii h.s.
Do not substitute
Dr. P.Sm

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide.

Please consult complete prescribing information, a summary of which follows:

* **Indications:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:
"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl/Roche) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.
Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

ROCHE®
Roche Products Inc.
Manati, Puerto Rico 00701

ROCHE®

FLARE-UP

SPASM AND PAIN CAN SIGNAL FUNCTIONAL GI DISORDERS*

Patients experiencing symptoms of irritable bowel syndrome* or duodenal ulcer* can often have emotional stress operating in the background. When you prescribe Librax for these patients, they receive treatment for both the emotional and the somatic elements to help relieve the anxiety/pain cycle.

Librax provides the well-known antianxiety action of Librium® (chlordiazepoxide HCl/Roche), a benzodiazepine with an established record of safety after use in thousands of patients worldwide. Also included are the proven antispasmodic and antisecretory actions of Quarzan® (clidinium bromide/Roche), the component which helps to reduce colonic spasm and hypersecretion and helps also to alleviate the pain they cause.

LIBRAX: FOR THE DUAL PROBLEMS OF FUNCTIONAL GI DISORDERS.

SPECIFY ADJUNCTIVE

LIBRAX®

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide.

ANTIANXIETY
ANTISECRETORY
ANTISPASMODIC

*Librax has been evaluated as possibly effective as adjunctive therapy in the treatment of duodenal ulcer and the irritable bowel syndrome.

Copyright © 1983 by Roche Products Inc. All rights reserved.

Please see summary of prescribing information on following page.

STACKS

NEW YORK STATE JOURNAL OF MEDICINE

FEBRUARY 1987 • Volume 87, Number 2

Medical Society of the State of New York • Organized 1807

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA
M.D.S.
FEB 18 1987

Contents

COMMENTARIES

Evaluating the impact of the 1986 New York State liability legislation 85
DONALD F. FOY

Ruptured diaphragm: A diagnostic dilemma 87
ELISABETH HEIBERG, MD

Tuberculosis of the knee 88
ALJE VENNEMA, MD

HISTORY

Arthur Donaldson Smith, MD: Physician, explorer, naturalist, and diplomat—Part I 90
PASCAL JAMES IMPERATO, MD

SPORTS MEDICINE

The exercise prescription 106
RICHARD B. BIRRER, MD, JOHN A. DALLER, MS

Treatment of acute sports injuries 113
JOHN BAUM, MD

The child and adolescent in sports 116
THOMAS L. WICKIEWICZ, MD

Cardiac evaluation of school sports participants 120
NORMAN B. SCHELL, MD, MARJORIE L. KING, MD

CASE REPORTS

Diaphragmatic hernia mimicking pyopneumothorax and colonic mass 122
ROBERT G. SPERBER, MD

Tuberculosis of the knee presenting as internal derangement 123
BURTON L. BERSON, MD; DOV J. BERKOWITZ, MD; MARVIN S. GILBERT, MD; MILTON C. ENGEL, MD; HANS E. SCHAPIRA, MD

Electrocardiographic anterior wall pseudoinfarction pattern caused by a metastatic brain tumor 125
RICK CONETTA, MD, BERNARD GITLER, MD

LETTERS TO THE EDITOR

Physicians and access to health care 128
RANDALL D. BLOOMFIELD, MD

The jury is a crowd 128
ALBERT W. COOK, MD

Relation of stroke to cocaine abuse 128
LOUIS L. CREGLER, MD, HERBERT MARK, MD

AN APPRECIATION

Our referees 130

BOOK REVIEWS 132

NEWS BRIEFS 136

OBITUARIES 137

MEDICAL MEETINGS AND LECTURES 10A

Annual CME Assembly, March 13–15, 1987, New York Hilton, New York City
(See December 1986 issue for Preliminary Program)

Library of the College Physician
of Philadelphia
19 S. 22nd St. Dec. 87
Phila, PA 19103

Motrin® 800 TABLETS mg

ibuprofen

Extra strength
Convenience
Economy

Upjohn

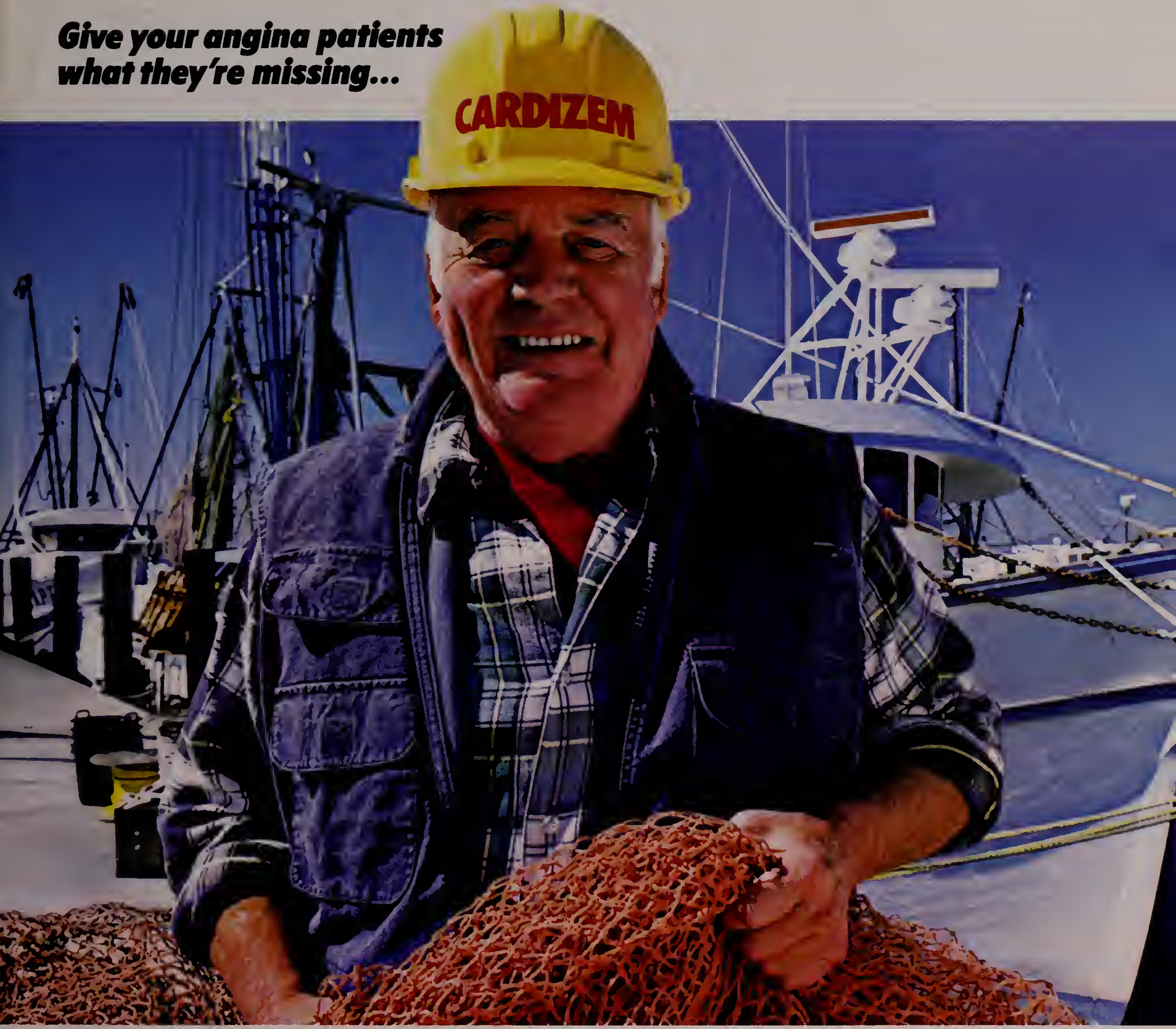
A Century of Caring

1886·1986

©1986 The Upjohn Company

J-6138 January 1986

Give your angina patients what they're missing...



CARDIZEM®: FEW SIDE EFFECTS

diltiazem HCl/Marion

- **Antianginal action includes dilatation of coronary arteries, a decrease in vascular resistance/afterload, and a reduction in heart rate**
- **Proven efficacy when used alone in angina[1]**
- **Compatible with other antianginals[2,3*]**
- **A safe choice for angina patients with coexisting hypertension, asthma, COPD, or PVD[4,5]**

***See Warnings and Precautions.**

Please see brief summary of prescribing information on the next page.

CARDIZEM® FEW SIDE EFFECTS IN ANTIANGINAL THERAPY

diltiazem HCl/Marion

60 mg tid or qid

Brief Summary
Professional Use Information

CARDIZEM®
(diltiazem HCl) 30 mg and 60 mg Tablets

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. ***Cardiac Conduction.*** *CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1,243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.*
2. ***Congestive Heart Failure.*** *Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt).*
   Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. ***Hypotension.*** *Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.*
4. ***Acute Hepatic Injury.*** *In rare instances, significant elevations in enzymes such as alkaline phosphatase, CPK, LDH, SGOT, SGPT, and other symptoms consistent with acute hepatic injury have been noted. These reactions have been reversible upon discontinuation of drug therapy. The relationship to CARDIZEM is uncertain in most cases, but probable in some. (See PRECAUTIONS.)*

PRECAUTIONS

General. *CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.*

Drug Interaction. *Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)*

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. *A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.*

Pregnancy. *Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.*

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. *Diltiazem is excreted in human milk. One report suggests that concentrations in breast milk may approximate serum levels. If use of CARDIZEM is deemed essential, an alternative method of infant feeding should be instituted.*

Pediatric Use. *Safety and effectiveness in children have not been established.*

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences as well as their frequency of presentation are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%). In addition, the following events were reported infrequently (less than 1%):

Cardiovascular:	*Angina, arrhythmia, AV block (first degree), AV block (second or third degree — see conduction warning), bradycardia, congestive heart failure, flushing, hypotension, palpitations, syncope.*
Nervous System:	*Amnesia, gait abnormality, hallucinations, insomnia, nervousness, paresthesia, personality change, somnolence, tinnitus, tremor.*
Gastrointestinal:	*Anorexia, constipation, diarrhea, dysgeusia, dyspepsia, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH (see hepatic warnings), vomiting, weight increase.*
Dermatologic:	*Petechiae, pruritus, photosensitivity, urticaria.*
Other:	*Amblyopia, dyspnea, epistaxis, eye irritation, hyperglycemia, nasal congestion, nocturia, osteoarticular pain, polyuria, sexual difficulties.*

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: alopecia, gingival hyperplasia, erythema multiforme, and leukopenia. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established. *Issued 7/86*

See complete Professional Use Information before prescribing.

References: 1. *Pepine CJ, Feldman RL, Hill JA, et al: Clinical outcome after treatment of rest angina with calcium blockers: Comparative experience during the initial year of therapy with diltiazem, nifedipine, and verapamil. Am Heart J 1983, 106(6):1341-1347.* ***2.*** *Shapiro W: Calcium channel blockers: Actions on the heart and uses in ischemic heart disease. Consultant 1984;24(Dec):150-159.* ***3.*** *Johnston DL, Lesoway R, Humen DP, et al: Clinical and hemodynamic evaluation of propranolol in combination with verapamil, nifedipine and diltiazem in exertional angina pectoris: A placebo-controlled, double-blind, randomized, crossover study. Am J Cardiol 1985;55:680-687.* ***4.*** *Cohn PF, Braunwald E: Chronic ischemic heart disease, in Braunwald E (ed): Heart Disease: A Textbook of Cardiovascular Medicine, ed 2. Philadelphia, WB Saunders Co, 1984, chap 39.* ***5.*** *Schroeder JS: Calcium and beta blockers in ischemic heart disease: When to use which. Mod Med 1982;50(Sept):94-116.*

Another patient benefit product from
PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC.
KANSAS CITY, MO 64137

0246M6

NEW YORK STATE JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
FRANK C. MAXON, JR, MD
ELIZA H. CALDWELL, MD
JOHN T. PRIOR, MD
STANFORD WESSLER, MD

Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Consulting Editor and Book Review Editor	RICHARD B. BIRRER, MD
Consulting Editor	NAOMI R. BLUESTONE, MD, MPH
Consulting Editor	CARL POCHEDLY, MD
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Managing Editor	CAROL L. MOORE
Librarian	ELLA ABNEY
Assistant Librarian	ELEANOR BURNS
Consulting Medical Writer	VICKI GLASER
Editorial Assistant	MILDRED J. ARFMANN
Secretary	ELIZABETH J. SOMERS
Advertising Director	ROBERT MILLER

ASSOCIATE EDITORIAL BOARD 1986

MICHAEL E. BERLOW, MD
RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MYLES S. GOMBERT, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
JAMES M. MORRISSEY, ESQ
STEPHEN NORDLICHT, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. United Media Associates, Inc, 16 Bruce Park Ave, Greenwich, CT 06830. Telephone: (203) 661-9702.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

JOHN A. FINKBEINER, MD,	*President*
DANIEL F. O'KEEFFE, MD,	*Past-President*
SAMUEL M. GELFAND, MD,	*President-Elect*
CHARLES D. SHERMAN, MD,	*Vice-President*
JOHN H. CARTER, MD,	*Secretary*
GEORGE LIM, MD,	*Assistant Secretary*
MORTON KURTZ, MD,	*Treasurer*
ROBERT A. MAYERS, MD,	*Assistant Treasurer*
CHARLES N. ASWAD, MD,	*Speaker*
SEYMOUR R. STALL, MD,	*Vice-Speaker*

Councilors

Term Expires 1987

DAVID M. BENFORD, MD,	New York
DUANE M. CADY, MD,	Onondaga
JAMES M. FLANAGAN, MD,	Wayne
M. THEODORE TANENHAUS, MD,	Kings
KATHLEEN E. SQUIRES, MD,	New York

(*Councilor representing the resident physician membership*)

FRANCES HARTENSTEIN-COLLICHIO,	Monroe

(*Councilor representing the medical student membership*)

Term Expires 1988

JAMES H. COSGRIFF, JR, MD,	Erie
RICHARD A. HUGHES, MD,	Warren
ANTONIO F. LASORTE, MD,	Broome
SIDNEY MISHKIN, MD,	Nassau

Term Expires 1989

ROBERT E. FEAR, MD,	Suffolk
STANLEY L. GROSSMAN, MD,	Orange
THOMAS D. PEMRICK, MD,	Rensselaer
RALPH E. SCHLOSSMAN, MD,	Queens

Trustees

RALPH M. SCHWARTZ, MD,	Kings
EDGAR P. BERRY, MD,	New York
RICHARD D. EBERLE, MD,	Onondaga
JOSEPH F. SHANAPHY, MD,	Richmond
VICTOR J. TOFANY, MD,	Monroe
BERNARD J. PISANI, MD,	New York
DANIEL F. O'KEEFFE, MD,	Warren

Executive Vice-President	DONALD F. FOY
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Vice-President Emeritus	GEORGE J. LAWRENCE, JR, MD
Director, Division of Policy Coordination	IRMA A. ERICKSON
Director, Division of Scientific Publications	PASCAL JAMES IMPERATO, MD
Director, Division of Communications	EDWARD A. HYNES
Director, Division of Computer Services	IVAN H. NAJMAN
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Governmental Affairs	GERARD L. CONWAY, ESQ
General Counsel	DONALD R. MOY, JD
Director, Division of Management and Meeting Services	ROBERT MILLER
Director, Division of Membership Support Services	EUNICE M. SKELLY
Director, Division of Scientific and Educational Activities	PETER B. FARNSWORTH, MD
Director, Division of Socio-Medical Economics	MARIO V. MENGHINI
Medical Director, Committee for Physicians' Health	BARTON A. HARRIS, MD
Consultant	EDWARD SIEGEL, MD

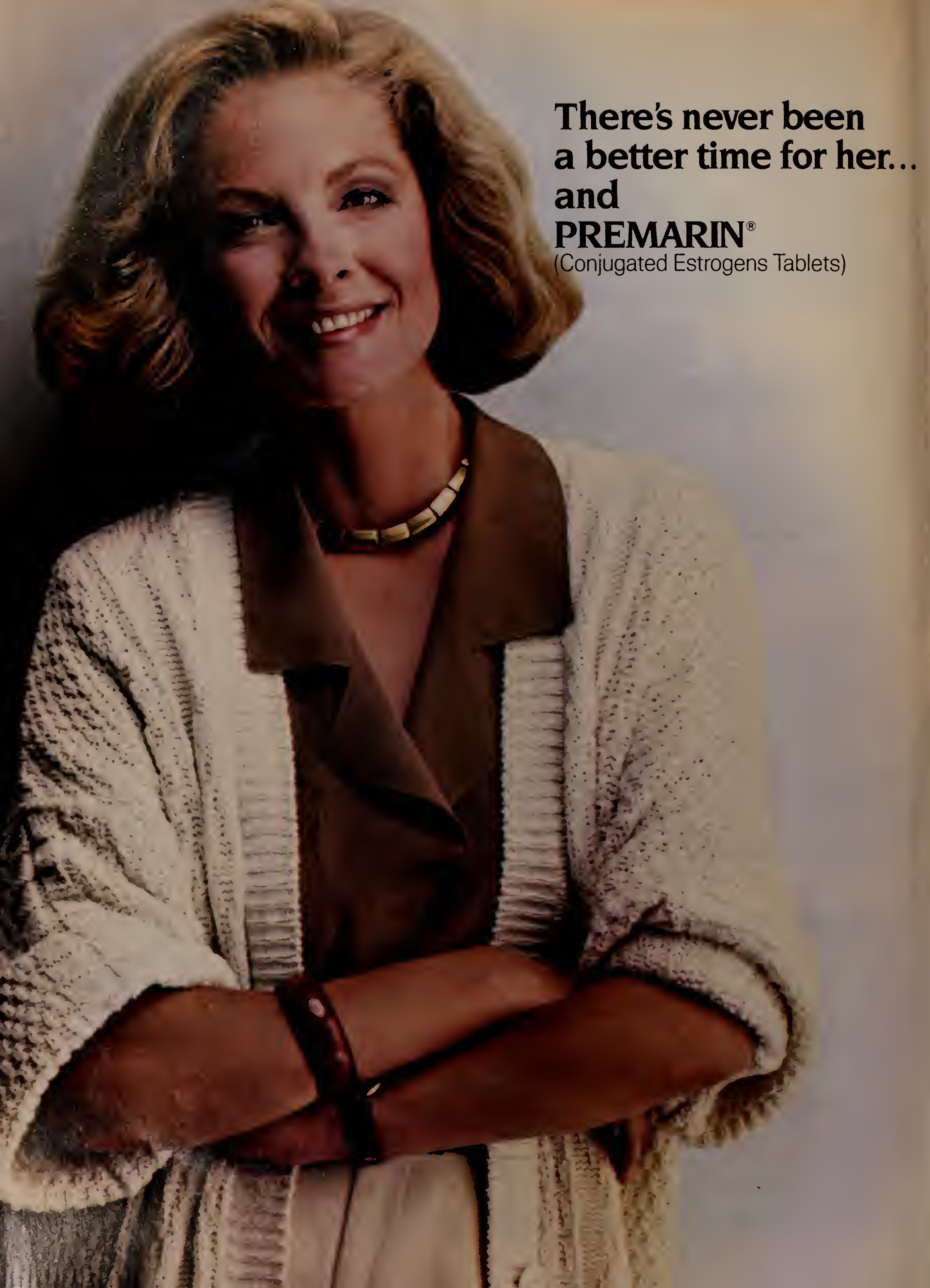
The New York State Journal of Medicine (ISSN 0028-7628) is published monthly, with a special issue added in April, by the Medical Society of the State of New York.

Copyright © 1986, Medical Society of the State of New York. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.


There's never been a better time for her… and PREMARIN®
(Conjugated Estrogens Tablets)

Now the evidence looks better than ever

Significantly reduced risk of endometrial hyperplasia

Endometrial hyperplasia was significantly reduced when progestin was added to PREMARIN therapy for more than ten days a month.[1-4] The risk of endometrial hyperplasia may also be reduced through cyclic administration of unopposed, low-dose PREMARIN.

Effect on lipids—an important feature

PREMARIN used alone does not adversely affect lipid levels. In fact, a clinical study has shown a significant increase in HDL cholesterol—from 49.7 mg/dL to 56.4 mg/dL—and decrease in LDL cholesterol—from 165.1 mg/dL to 138.1 mg/dL—after one year of therapy with PREMARIN, 0.625 mg.[5]

Low-dose control of menopausal symptoms*

PREMARIN effectively relieves vasomotor symptoms, such as hot flashes. When estrogen deficiency is limited to atrophic vaginitis, PREMARIN® (conjugated estrogens) Vaginal Cream restores the vaginal environment to its premenopausal state.

The most widely used, most extensively studied estrogen worldwide.

PREMARIN®

(Conjugated Estrogens Tablets)

Most trusted for more reasons

---

*PREMARIN is indicated for moderate-to-severe vasomotor symptoms.

Please see following page for brief summary of prescribing information.

For moderate-to-severe vasomotor symptoms

PREMARIN®
(Conjugated Estrogens Tablets)

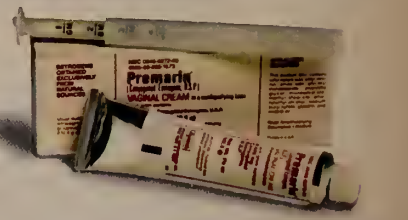
0.3 mg 0.625 mg 0.9 mg 1.25 mg 2.5 mg

The appearance of these tablets is a trademark of Ayerst Laboratories.

For atrophic vaginitis

PREMARIN®
(Conjugated Estrogens)

Vaginal Cream
0.625mg/g



BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION AND PATIENT INFORMATION, SEE PACKAGE CIRCULARS.)*

PREMARIN® Brand of conjugated estrogens tablets, USP
PREMARIN® Brand of conjugated estrogens Vaginal Cream in a nonliquefying base

1. ESTROGENS HAVE BEEN REPORTED TO INCREASE THE RISK OF ENDOMETRIAL CARCINOMA

Three independent case control studies have reported an increased risk of endometrial cancer in postmenopausal women exposed to exogenous estrogens for more than one year. This risk was independent of the other known risk factors for endometrial cancer. These studies are further supported by the finding that incidence rates of endometrial cancer have increased sharply since 1969 in eight different areas of the United States with population-based cancer reporting systems, an increase which may be related to the rapidly expanding use of estrogens during the last decade. The three case control studies reported that the risk of endometrial cancer in estrogen users was about 4.5 to 13.9 times greater than in nonusers The risk appears to depend on both duration of treatment and on estrogen dose. In view of these findings, when estrogens are used for the treatment of menopausal symptoms, the lowest dose that will control symptoms should be utilized and medication should be discontinued as soon as possible. When prolonged treatment is medically indicated, the patient should be reassessed on at least a semiannual basis to determine the need for continued therapy. Although the evidence must be considered preliminary, one study suggests that cyclic administration of low doses of estrogen may carry less risk than continuous administration; it therefore appears prudent to utilize such a regimen. Close clinical surveillance of all women taking estrogens is important. In all cases of undiagnosed persistent or recurring abnormal vaginal bleeding, adequate diagnostic measures should be undertaken to rule out malignancy. There is no evidence at present that "natural" estrogens are more or less hazardous than "synthetic" estrogens at equiestrogenic doses.

2. ESTROGENS SHOULD NOT BE USED DURING PREGNANCY.

The use of female sex hormones, both estrogens and progestogens, during early pregnancy may seriously damage the offspring. It has been shown that females exposed in utero to diethylstilbestrol, a non-steroidal estrogen, have an increased risk of developing in later life a form of vaginal or cervical cancer that is ordinarily extremely rare. This risk has been estimated as not greater than 4 per 1,000 exposures. Furthermore, a high percentage of such exposed women (from 30% to 90%) have been found to have vaginal adenosis, epithelial changes of the vagina and cervix. Although these changes are histologically benign, it is not known whether they are precursors of malignancy. Although similar data are not available with the use of other estrogens, it cannot be presumed they would not induce similar changes. Several reports suggest an association between intrauterine exposure to female sex hormones and congenital anomalies, including congenital heart defects and limb reduction defects. One case control study estimated a 4 7-fold increased risk of limb reduction defects in infants exposed in utero to sex hormones (oral contraceptives, hormone withdrawal tests for pregnancy, or attempted treatment for threatened abortion). Some of these exposures were very short and involved only a few days of treatment. The data suggest that the risk of limb reduction defects in exposed fetuses is somewhat less than 1 per 1,000. In the past, female sex hormones have been used during pregnancy in an attempt to treat threatened or habitual abortion. There is considerable evidence that estrogens are ineffective for these indications, and there is no evidence from well controlled studies that progestogens are effective for these uses. If PREMARIN is used during pregnancy, or if the patient becomes pregnant while taking this drug, she should be apprised of the potential risks to the fetus, and the advisability of pregnancy continuation.

DESCRIPTION: PREMARIN (conjugated estrogens, USP) contains a mixture of estrogens, obtained exclusively from natural sources, blended to represent the average composition of material derived from pregnant mares' urine It contains estrone, equilin, and 17α-dihydroequilin, together with smaller amounts of 17α-estradiol, equilenin, and 17α-dihydroequilenin as salts of their sulfate esters Tablets are available in 0.3 mg, 0.625 mg, 0.9 mg, 1.25 mg, and 2.5 mg strengths of conjugated estrogens Cream is available as 0.625 mg conjugated estrogens per gram.

INDICATIONS AND USAGE: PREMARIN (conjugated estrogens tablets, USP): Moderate-to-severe *vasomotor* symptoms associated with the menopause. (There is no evidence that estrogens are effective for nervous symptoms or depression without associated vasomotor symptoms and they should not be used to treat such conditions.) **Osteoporosis (abnormally low bone mass)**. Atrophic vaginitis. Kraurosis vulvae Female castration

PREMARIN (conjugated estrogens) Vaginal Cream is indicated in the treatment of atrophic vaginitis and kraurosis vulvae. PREMARIN HAS NOT BEEN SHOWN TO BE EFFECTIVE FOR ANY PURPOSE DURING PREGNANCY AND ITS USE MAY CAUSE SEVERE HARM TO THE FETUS (SEE BOXED WARNING).

Concomitant Progestin Use: The lowest effective dose appropriate for the specific indication should be utilized. Studies of the addition of a progestin for 7 or more days of a cycle of estrogen administration have reported a lowered incidence of endometrial hyperplasia Morphological and biochemical studies of the endometrium suggest that 10 to 13 days of progestin are needed to provide maximal maturation of the endometrium and to eliminate any hyperplastic changes. Whether this will provide protection from endometrial carcinoma has not been clearly established There are possible additional risks which may be associated with the inclusion of progestin in estrogen replacement regimens. (See PRECAUTIONS) The choice of progestin and dosage may be important; product labeling should be reviewed to minimize possible adverse effects.

CONTRAINDICATIONS: Estrogens should not be used in women (or men) with any of the following conditions: 1. Known or suspected cancer of the breast except in appropriately selected patients being treated for metastatic disease 2 Known or suspected estrogen-dependent neoplasia 3 Known or suspected pregnancy (See Boxed Warning) 4. Undiagnosed abnormal genital bleeding 5. Active thrombophlebitis or thromboembolic disorders 6 A past history of thrombophlebitis, thrombosis, or thromboembolic disorders associated with previous estrogen use (except when used in treatment of breast or prostatic malignancy).

WARNINGS: Long-term continuous administration of natural and synthetic estrogens in certain animal species increases the frequency of carcinomas of the breast, cervix, vagina, and liver. There are now reports that estrogens increase the risk of carcinoma of the endometrium in humans. (See Boxed Warning.) At the present time there is no satisfactory evidence that estrogens given to postmenopausal women increase the risk of cancer of the breast, although a recent study has raised this possibility There is a need for caution in prescribing estrogens for women with a strong family history of breast cancer or who have breast nodules, fibrocystic disease, or abnormal mammograms A recent study has reported a 2- to 3-fold increase in the risk of surgically confirmed gallbladder disease in women receiving postmenopausal estrogens.

Adverse effects of oral contraceptives may be expected at the larger doses of estrogen used to treat prostatic or breast cancer or postpartum breast engorgement; it has been shown that there is an increased risk of thrombosis in men receiving estrogens for prostatic cancer and women for postpartum breast engorgement. Users of oral contraceptives have an increased risk of diseases, such as thrombophlebitis, pulmonary embolism, stroke, and myocardial infarction. Cases of retinal thrombosis, mesenteric thrombosis, and optic neuritis have been reported in oral contraceptive users An increased risk of postsurgery thromboembolic complications has also been reported in users of oral contraceptives. If feasible. estrogen should be discontinued at least 4 weeks before surgery of the type associated with an increased risk of thromboembolism, or during periods of prolonged immobilization Estrogens should not be used in persons with active thrombophlebitis, thromboembolic disorders, or in persons with a history of such disorders in association with estrogen use. They should be used with caution in patients with cerebral vascular or coronary artery disease. Large doses (5 mg conjugated estrogens per day), comparable to those used to treat cancer of the prostate and breast, have been shown to increase the risk of nonfatal myocardial infarction, pulmonary embolism and thrombophlebitis. When doses of this size are used, any of the thromboembolic and thrombotic adverse effects should be considered a clear risk

Benign hepatic adenomas should be considered in estrogen users having abdominal pain and tenderness, abdominal mass, or hypovolemic shock Hepatocellular carcinoma has been reported in women taking estrogen-containing oral contraceptives Increased blood pressure may occur with use of estrogens in the menopause and blood pressure should be monitored with estrogen use A worsening of glucose tolerance has been observed in patients on estrogen-containing oral contraceptives. For this reason, diabetic patients should be carefully observed Estrogens may lead to severe hypercalcemia in patients with breast cancer and bone metastases

PRECAUTIONS: Physical examination and a complete medical and family history should be taken prior to the initiation of any estrogen therapy with special reference to blood pressure, breasts, abdomen, and pelvic organs, and should include a Papanicolaou smear. As a general rule, estrogen should not be prescribed for longer than one year without another physical examination being performed Conditions influenced by fluid retention such as asthma, epilepsy, migraine, and cardiac or renal dysfunction, require careful observation Certain patients may develop manifestations of excessive estrogenic stimulation, such as abnormal or excessive uterine bleeding. mastodynia, etc. Prolonged administration of unopposed estrogen therapy has been reported to increase the risk of endometrial hyperplasia in some patients. Oral contraceptives appear to be associated with an increased incidence of mental depression. Patients with a history of depression should be carefully observed Preexisting uterine leiomyomata may increase in size during estrogen use The pathologist should be advised of estrogen therapy when relevant specimens are submitted. If jaundice develops in any patient receiving estrogen, the medication should be discontinued while the cause is investigated Estrogens should be used with care in patients with impaired liver function, renal insufficiency, metabolic bone diseases associated with hypercalcemia, or in young patients in whom bone growth is not complete. If concomitant progestin therapy is used, potential risks may include adverse effects on carbohydrate and lipid metabolism.

The following changes may be expected with larger doses of estrogen:

a. Increased sulfobromophthalein retention

b Increased prothrombin and factors VII, VIII, IX, and X, decreased antithrombin 3; increased norepinephrine-induced platelet aggregability.

c. Increased thyroid binding globulin (TBG) leading to increased circulating total thyroid hormone, as measured by PBI, T4 by column, or T4 by radioimmunoassay Free T3 resin uptake is decreased, reflecting the elevated TBG; free T4 concentration is unaltered.

d. Impaired glucose tolerance.

e. Decreased pregnanediol excretion.

f. Reduced response to metyrapone test.

g Reduced serum folate concentration.

h. Increased serum triglyceride and phospholipid concentration. As a general principle, the administration of any drug to nursing mothers should be done only when clearly necessary since many drugs are excreted in human milk

ADVERSE REACTIONS: The following have been reported with estrogenic therapy, including oral contraceptives: breakthrough bleeding, spotting, change in menstrual flow, dysmenorrhea; premenstrual-like syndrome; amenorrhea during and after treatment; increase in size of uterine fibromyomata; vaginal candidiasis, change in cervical erosion and in degree of cervical secretion; cystitis-like syndrome; tenderness, enlargement, secretion (of breasts); nausea, vomiting, abdominal cramps, bloating; cholestatic jaundice; chloasma or melasma which may persist when drug is discontinued; erythema multiforme; erythema nodosum; hemorrhagic eruption; loss of scalp hair, hirsutism; steepening of corneal curvature; intolerance to contact lenses; headache, migraine, dizziness, mental depression, chorea; increase or decrease in weight; reduced carbohydrate tolerance, aggravation of porphyria; edema; changes in libido.

ACUTE OVERDOSAGE: May cause nausea, and withdrawal bleeding may occur in females.

DOSAGE AND ADMINISTRATION:

PREMARIN® Brand of conjugated estrogens tablets, USP

1. *Given cyclically for short-term use only.* For treatment of moderate to severe *vasomotor* symptoms, atrophic vaginitis, or kraurosis vulvae associated with the menopause (0.3 to 1.25 mg or more daily). The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible Administration should be cyclic (eg, three weeks on and one week off). Attempts to discontinue or taper medication should be made at three- to six-month intervals.

2. *Given cyclically* Female castration. Osteoporosis. Female castration—1.25 mg daily, cyclically Adjust upward or downward according to response of the patient. For maintenance, adjust dosage to lowest level that will provide effective control. Osteoporosis —0.625 mg daily Administration should be cyclic (eg, three weeks on and one week off).

Patients with an intact uterus should be monitored for signs of endometrial cancer and appropriate measures taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding.

PREMARIN® Brand of conjugated estrogens Vaginal Cream

Given cyclically for short-term use only. For treatment of atrophic vaginitis or kraurosis vulvae.

The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible.

Administration should be cyclic (eg, three weeks on and one week off).

Attempts to discontinue or taper medication should be made at three-to-six month intervals.

Usual dosage range: 2 to 4 g daily, intravaginally, depending on the severity of the condition.

Treated patients with an intact uterus should be monitored closely for signs of endometrial cancer and appropriate diagnostic measures should be taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding

References:

1. Whitehead MI, Townsend PT, Pryse-Davies J, et al: Effects of estrogens and progestins on the biochemistry and morphology of the postmenopausal endometrium. *N Engl J Med* 1981;305:1599-1605 **2.** Paterson MEL, Wade-Evans T, Sturdee DW, et al: Endometrial disease after treatment with oestrogens and progestogens in the climacteric. *Br Med J* 1980;280:822-824 **3.** Magos AL, Brincat M, Studd JWW, et al: Amenorrhea and endometrial atrophy with continuous oral estrogen and progestogen therapy in postmenopausal women. *Obstet Gynecol* 1985; 67: 496-499. **4.** Whitehead MI, Lane G, Siddle N, et al: Avoidance of endometrial hyperstimulation in estrogen-treated postmenopausal women *Semin Reprod Endocrinol* 1983;1:1;41-52 **5**. Barnes RB, Roy S, Lobo RA: Comparison of lipid and androgen levels after conjugated estrogen or depo-medroxyprogesterone acetate treatment in postmenopausal women *Obstet Gynecol* 1985,66:216-219

© 1986 Ayerst Laboratories

Ayerst®
AYERST LABORATORIES
New York, NY 10017

T6194/886

Photography by Ken Nahoum

AIDS is a complex disease, assaulting the psyche as well as the body. The Spellman Center for the Treatment of Persons With AIDS treats both. A comprehensive medical facility, Spellman is devoted to the care of those suffering from Acquired Immune Deficiency Syndrome.

Respect for individual dignity guides the Spellman program. Staffed by trained personnel dedicated to working with AIDS patients, the Center includes family and friends in the management of the patient's health crisis. Spellman also operates a 24-hour information hotline at 1-800-433-AIDS.

For criteria on admission and referrals, please contact the Medical Director, at 1-212-307-0735. Spellman is a division of St. Clare's Hospital and Health Center, designated the first AIDS treatment center by New York State.

The Spellman Center
for the Treatment of Persons With AIDS
St. Clare's Hospital and Health Center

SC

415 W 51st St
New York, NY 10019

An Affiliate of New York Medical College

An AZT Clinical Site

Medical experts,
providers of care

Classified Advertising

PHYSICIANS WANTED

ARIZONA BASED PHYSICIAN RECRUITMENT firm has opportunities Coast to Coast. "Professionals working with Professionals". Over 13 years experience. Call (602) 990-8080, or send CV to: Mitchell & Associates, Inc., P.O. Box 1804, Scottsdale, AZ 85252.

EMERGENCY MEDICINE POSITIONS. Full/part time emergency medicine physicians sought by multi-state professional association. Openings in metropolitan NY, NJ, PA, MD, DC, FL and throughout U.S. Contact or send CV to Liberty Healthcare Corporation, 399 Market Street, Suite 400, Philadelphia, PA 19106, (215) 592-7400 or outside PA (800) 331-7122.

GENERAL SURGERY—Solo General Surgical Practice looking for a young associate to begin Spring/Summer 1987. General and some vascular surgery. Delightful upstate location. Prefer candidates completing residency or with recent training. Send confidential letter and C.V. to P.O. Box 505, New Hartford, New York 13413.

DO YOU WANT TO SHARE A THRIVING DERMATOLOGY PRACTICE: Do you enjoy year around outdoor recreation? You should contact us—a progressive multi-specialty group of 68 physicians in a pleasant, growing community. We offer a liberal financial package and outstanding benefits. Send curriculum vitae and references to: Michael T. Anderson, M.D., Willmar Medical Center, 101 Willmar Ave. SW, Willmar, MN 56201.

PHYSICIANS needed for 75 children's private residential camps July and/or August. Contact: Association of Independent Camps (JM), 43 West 23rd Street—Sixth Floor, New York, N.Y. 10010 (212) 645-6620.

BE A "WINTER TEXAN" INTERNIST. Enjoy the warm, beautiful Rio Grande Valley while practicing internal medicine with an internist. Texas license essential. Salary, living accommodations, and malpractice insurers. Send curriculum vitae to Luis F. Arango, M.D., P.A., 104 South Bryan Road, Mission, Texas 78572 or call (512) 585-2783 for more information.

CAMP WAYNE, Northeast Pennsylvania, Wayne County, 2½ hours from New York City. Physician 2–4 weeks between June 26 and August 21. Professional staff, nurses, private lake, tennis, arts, sports, fine facilities, family accommodations. (516) 599-4562. 570 Broadway, Lynbrook, New York 11563.

PEDIATRICIAN, Board eligible/certified, to work in a community health center. Salary commensurate with experience. Send CV to Edward Lebowitz, MD, Acting Medical Director, Jordan Health Center, 82 Holland Street, Box 876, Rochester, New York 14603, EOE, M/F.

PHYSICIANS WANTED—CONT'D

HEALTH COMMISSIONER: Physician needed to administer and manage Rural Orange County, NY (50 miles north of NYC) Public Health Department. Send resume and salary history to: Commissioner of Personnel, Orange County Government Center, 255-275 Main St., Goshen, NY 10924. An Equal Opportunity Employer M/F.

NEEDED NY STATE LICENSED pediatrician, obs-gynecologist dermatologist, ophthalmologist, psychiatrist and dentist. Part-time/full-time. Excellent neighborhood, good growth potential. Write Lovina Medical Offices, 1423 Glover Street, Bronx, NY 10462 or call (212) 597-8383.

GEORGIA, Director: Low volume Emergency Department in pleasant, smaller community. Located 1½ hours from Georgia's Golden Isles. Competitive compensation, benefit package, no on-call responsibilities. Professional liability insurance procured on your behalf. For more information contact: Sheila Murphy, Coastal Emergency Services, Inc., P.O. Box 925, Augusta, GA 30903; collect (404) 724-3368.

NEW YORK, WESTERN: Seeking primary care trained physicians for full time Emergency Department positions. Moderate volume. Attractive hourly compensation, plus malpractice insurance, Director position available. Contact: Emergency Consultants, Inc., 2240 S. Airport Road, Room 42, Traverse City, MI 49684: 1-800-253-1795 or in Michigan 1-800-632-3496.

POSITIONS WANTED

OPHTHALMOLOGIST, 32, well trained and very surgically oriented, seeks employment with ultimate association in group practice in New York State. Reply Dept. 434 c/o NYSJM.

RADIOLOGIST, A.B.R. & A.B.N.M. is seeking a salaried position with a group or a hospital for 4–5 years. Group partnership not a consideration. Expertise in Angiography including neuro (20 years). Special competence in nuclear cardiology. Mammography & xeromammography (10 years), cat-head & body (6 years). Artrography, special procedures, ultrasonography & diagnostic radiology. Location limited to 100 mile radius of New York City. Reply to Dept. 432 c/o NYSJM.

PRACTICES AVAILABLE

FOR SALE—Long established Primary Care Internal Medicine practice and office building. Medium sized Finger Lakes City. Ten minutes to 200 bed hospital. Three rental apartments in office building. Will stay on and introduce to patients. Terms generous. Reply Dept. 433 c/o NYSJM.

FAMILY PRACTICE AND OFFICE BUILDING in $3 million, 5 acre professional complex for sale. 1800 square feet, fully equipped, 5 exam rooms, beautifully landscaped. Room for two physicians. Located in Modesto with easy access to San Francisco. Available now. Contact Emile Lengyel, M.D., 4141 Carver Road, Modesto, CA 95356, (209) 522-2544.

PRACTICES AVAILABLE—CONT'D

"SELLING PRACTICES IS OUR ONLY BUSINESS"

Want to maximize the return on your investment? Before you buy or sell, call for an appraisal to assure the best financial and transfer terms. We guide you through the entire sales process from initial meeting to closing. Serving NY, CT, NJ and MA.

Countrywide Business Brokerage, Inc.
319 East 24th St., Suite 23-G
New York, NY 10010
(212) 686-7902 (203) 869-3666

6-YEAR INTERNAL MEDICINE PRACTICE FOR SALE in Dutchess County. Good location, near two hospitals, in rapidly growing area. Leaving area, reasonable sale price. Reply Dept. #431 c/o NYSJM.

REAL ESTATE FOR SALE OR RENT

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

EAST SIXTIES, PROFESSIONAL BUILDING. The preferred location for internists, cardiologist, rheumatologist. 7 treatment rooms, 2 reception areas to share with three other internists. Share NYC licensed lab, radiology, thermography. Full or part time. (212) 838-2860.

EAST 76th STREET BETWEEN PARK & MADISON. Charmingly appointed Internist's office. Own street entrance. Ideal for psychiatrist, surgeon or specialist. Excellent rent for current market. Available Tuesday, Thursday and Friday. Hours flexible. (212) 988-7816.

FULL OR PART-TIME consultation and waiting room for psychiatrist in newly renovated brownstone. Available Mid-February. 92nd Street between Lexington and Park, (212) 289-7166.

BUILDING FOR RENT, newly renovated, suitable for professional office. Located in the fastest growing tourist/vineyard area on Long Island. Great fishing and boating. Highly visible corner plus parking. Rent $825/month, call (516) 883-1058.

EQUIPMENT

DISCOUNT HOLTER SCANNING SERVICES starting at $35.00. Spacelabs holter recorder (cassette) available from $1,275.00. Cardionostic holter recorder (cassette) available from $1,125.00. Smallest and lightest holters update. Fast service (24–48 hours) turn over. Hookup kits starting at $4.95. Special introductory offer of three free tests with any purchase or lease of the recorder. Cardiologist overread available for $15.00. If interested call (301) 870-3626.

Added Control For America's Coughs

The expectorant action of guaifenesin plus the Advantage of Codeine!

Robitussin and codeine. Each 5 ml (*1 teaspoonful*) contains: Guaifenesin, USP, 100 mg; Codeine Phosphate, USP, 10 mg (*warning: may be habit forming*); alcohol, 3.5 percent; in a palatable, aromatic syrup

A·H·ROBINS

Pharmaceutical Division, Richmond, Virginia 23261-6609

©A.H Robins Company 1985

Robitussin A-C® Ⓒ

PRI
PHYSICIANS RECIPROCAL INSURERS

Offering New York State Physicians the Broadest
Claims-Made Professional Liability Coverage

OUR UNIQUE PROGRAM INCLUDES...

■ SELECTIVE UNDERWRITING	A preferred risk group
■ STATEWIDE DISTRICT REPRESENTATIVES	Knowledgeable and sensitive to physicians' concerns
■ GROUP PRACTICE COVERAGE	Additional layer of protection at nominal cost
■ NEW DOCTOR & PART-TIME RATES	Aware of the special needs and unique situations of our fellow professionals
■ AND MORE....	

FOR ADDITIONAL INFORMATION CONTACT
PHYSICIANS RECIPROCAL INSURERS
111 East Shore Road, Manhasset, New York 11030
212/936-3050 • 516/365-6690 • TOLL FREE 800/632-6040

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices unless the information is submitted at least three months prior to the event.*

MARCH 1987

AROUND THE STATE

BRONX

Mar 25. **Current Issues in Forensic Serology: The Applications of DNA to the Forensic Sciences.** Contact: Mark L. Taff, MD, Nassau County Medical Examiner's Office, PO Box 160, East Meadow, NY 11554. Tel: (516) 542-2400.

MANHATTAN

Mar 6–7. **3rd Annual Live Ophthalmic Plastic Surgery Workshop: Eyelid, Socket & Lacrimal Surgery:** *Mar 21.* **Degenerative Diseases of the Retina: Diagnosis, Prognosis & Treatment.** Manhattan Eye, Ear and Throat Hospital. Contact: Martha Klapp, Manhattan, Eye, Ear and Throat Hospital, 210 East 64 St, New York, NY 10021. Tel: (212) 605-3762.

Mar 8–13. **Anesthesiology Comprehensive Review VII.** 47½ Cat 1 Credits. Marriot Marquis. *Mar 16–20.* **Seminar in Advanced Rheumatology.** 24½ Cat 1 Credits. NYU Medical Center. Contact: NYU Medical Center Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Mar 12–14. **14th Annual Conference on Dyslexia.** New York Penta Hotel. Info: New York Branch of the Orton Dyslexia Society, 80 Fifth Ave, Rm 903, New York, NY 10011. Tel: (212) 691-1930.

Mar 18. **The New Economics of Medical Care: The Identifier Concept.** 6 Cat 1 Credits. The Warwick Hotel. Contact: Ann J. Boehme, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

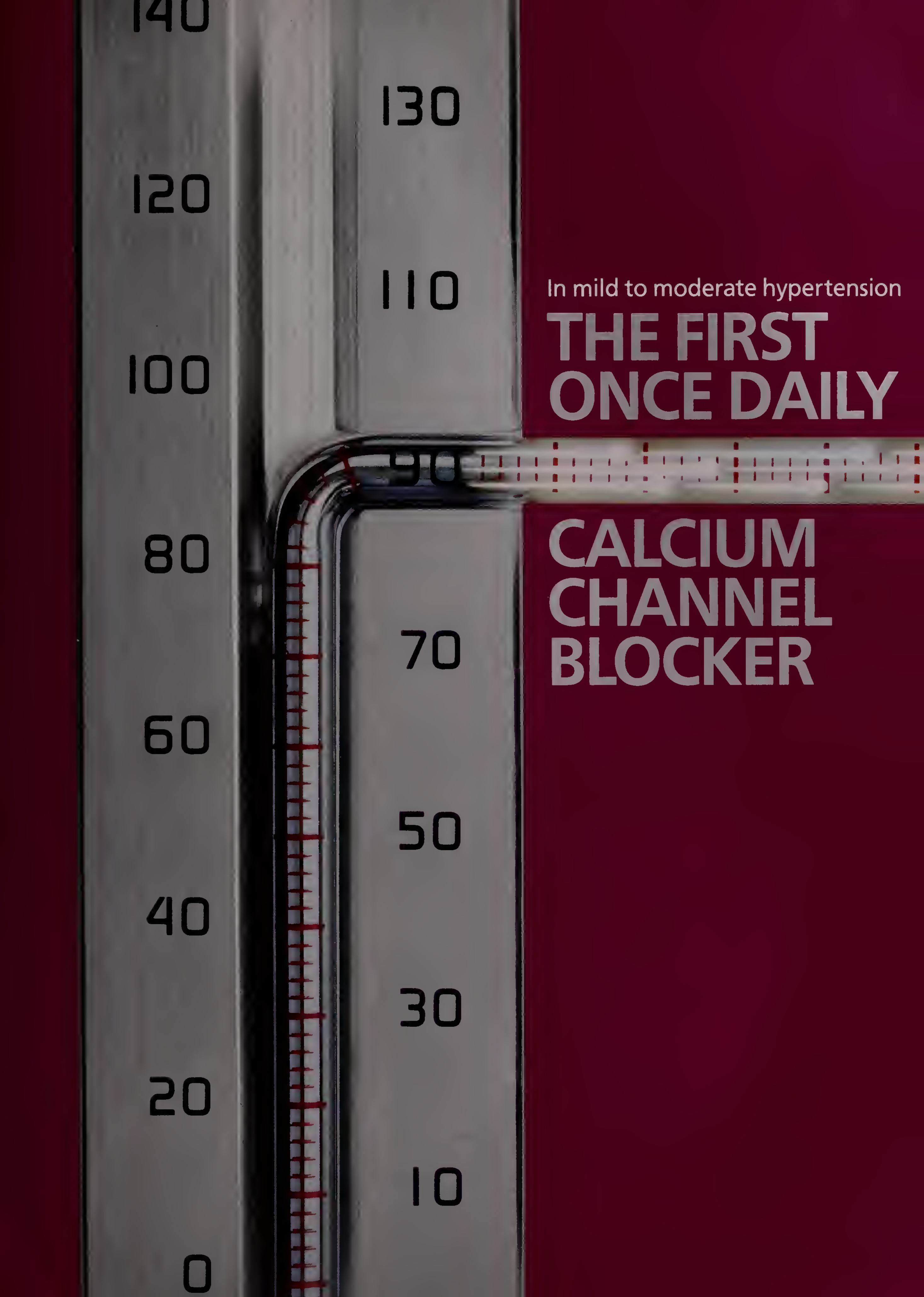
Mar 20. **Allergic, Immunological and Environmental Influences on Lung Disease.** Beth Israel Medical Center. Contact: Beverly R. Baptiste, Medical Education, Beth Israel Medical Center, First Ave at 16th St, New York, NY 10002.

Mar 26–27. **4th World Conference on Infectious Diseases and Human Infertility.** 14 Cat 1 Credits. Einhorn Auditorium, Lenox Hill Hospital. Contact: Masood A. Khatamee, MD, Program Chairman, Fertility Research Foundation, 1430 Second Ave, New York, NY 10021.

NASSAU

Mar 13–14. **People, Skills and Money: Building Comprehensive Community Services for Infants, Toddlers and Their Families.** 14 Cat 1 Credits. Garden City Hotel, Garden City. Info: Ann J. Boehme, Associate Director for Continuing Education, Schneider Children's Hospital, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

(*continued on p 13A*)


140
130
120
110
100
90
80
70
60
50
40
30
20
10
0
In mild to moderate hypertension
THE FIRST ONCE DAILY CALCIUM CHANNEL BLOCKER

NEW

ONCE DAILY

ISOPTIN SR*

(verapamil HCl/Knoll)

240 mg scored, sustained-release tablets

JAMES B.
38, black male, heavy smoker. Prescribed a diuretic by another physician last year for hypertension.

YOUR CONCERNS
Presents with "smoker's cough." Workup reveals a BP of 150/107.

A LOGICAL CHOICE FOR CONTROL OF HIS BP
ISOPTIN® (verapamil HCl/Knoll) because...

- Black hypertensives often have low plasma renin activity and generally do not respond favorably to beta blockers.
- Beta blockers may increase the likelihood of bronchospasm.

ALICE W.
65, diabetic, overweight. Her BP has elevated to 190/98.

YOUR CONCERNS
She's on daily insulin.

A LOGICAL CHOICE FOR CONTROL OF HER BP
ISOPTIN® (verapamil HCl/Knoll) because...

- Unlike most beta blockers and diuretics, ISOPTIN has no adverse effects on serum glucose levels.
- Unlike most beta blockers, ISOPTIN does not mask the symptoms of hypoglycemia.

THOMAS G.
70, asthmatic. In the past, BP adequately controlled with 25 mg hydrochlorothiazide daily.

YOUR CONCERNS
Today patient presents with symptoms of gout. Workup reveals high uric acid level, low serum potassium, and BP elevated to 180/98.

A LOGICAL CHOICE FOR CONTROL OF HIS BP
ISOPTIN® (verapamil HCl/Knoll) because...

- Unlike diuretics, ISOPTIN will not decrease serum potassium levels or elevate uric acid levels.
- Unlike beta blockers, ISOPTIN can be used safely in asthma and COPD patients.

JOHN K.
42, Annual physical uncovered diastolic BP of 102... confirmed on three successive office visits. Unresponsive to nonpharmacologic intervention.

YOUR CONCERNS
Salesman, spends many hours of his working day in car... total cholesterol level 300, HDL 35.

A LOGICAL CHOICE FOR CONTROL OF HIS BP
ISOPTIN® (verapamil HCl/Knoll) because...

- Unlike diuretics, ISOPTIN does not cause urinary urgency.
- Unlike either beta blockers or diuretics, ISOPTIN will not adversely affect his already seriously compromised lipid profile.
- Unlike with propranolol, fatigue and impotence are rarely reported.

Antihypertensive therapy you and your patients can live with

Knoll Pharmaceuticals
A Unit of BASF K&F Corporation
Whippany, New Jersey 07981

BASF Group

knoll®

*A product of Knoll research.

© 1986, BASF K & F Corporation

2538/1-87

Printed in U.S.A.

In mild to moderate hypertension Brief Summary

THE FIRST ONCE DAILY CALCIUM CHANNEL BLOCKER

ISOPTIN® SR
(verapamil HCl/Knoll)
240 mg scored, sustained-release tablets

CONTRAINDICATIONS: 1) Severe left ventricular dysfunction (see WARNINGS), 2) Hypotension (less than 90 mmHg systolic pressure) or cardiogenic shock, 3) Sick sinus syndrome or 2nd or 3rd degree AV block (except in patients with a functioning artificial ventricular pacemaker).

WARNINGS: Heart Failure: ISOPTIN should be avoided in patients with severe left ventricular dysfunction (see DRUG INTERACTIONS). Patients with milder ventricular dysfunction should, if possible, be controlled before verapamil treatment. Hypotension: ISOPTIN (verapamil HCl) may produce occasional symptomatic hypotension. Elevated Liver Enzymes: Elevations of transaminases with and without concomitant elevations in alkaline phosphatase and bilirubin have been reported. Periodic monitoring of liver function in patients receiving verapamil is therefore prudent. Accessory Bypass Tract (Wolff-Parkinson-White): Patients with paroxysmal and/or chronic atrial flutter or atrial fibrillation and a coexisting accessory AV pathway have developed increased antegrade conduction across the accessory pathway producing a very rapid ventricular response or ventricular fibrillation after receiving intravenous verapamil. While this has not been reported with oral verapamil, it should be considered a potential risk. Treatment is usually D.C.-cardioversion. Atrioventricular Block: The effect of verapamil on AV conduction and the SA node may cause asymptomatic 1st degree AV block and transient bradycardia. Higher degrees of AV block, while infrequent (0.8%), may require a reduction in dosage or, in rare instances, discontinuation of verapamil HCl. Patients with Hypertrophic Cardiomyopathy (IHSS): Although verapamil has been used in the therapy of patients with IHSS, severe cardiovascular decompensation and death have been noted in this patient population.

PRECAUTIONS: Impaired Hepatic or Renal Function: Verapamil is highly metabolized by the liver with about 70% of an administered dose excreted in the urine. In patients with impaired hepatic or renal function verapamil should be administered cautiously and the patients monitored for abnormal prolongation of the PR interval or other signs of excessive pharmacological effects (see OVERDOSAGE).

Drug Interactions: Beta Blockers: Concomitant use of ISOPTIN and oral beta-adrenergic blocking agents may be beneficial in certain patients with chronic stable angina or hypertension, but available information is not sufficient to predict with confidence the effects of concurrent treatment in patients with left ventricular dysfunction or cardiac conduction abnormalities. Digitalis: Clinical use of verapamil in digitalized patients has shown the combination to be well tolerated if digoxin doses are properly adjusted. However, chronic verapamil treatment increases serum digoxin levels by 50 to 75% during the first week of therapy and this can result in digitalis toxicity. Upon discontinuation of ISOPTIN (verapamil HCl), the patient should be reassessed to avoid underdigitalization. Antihypertensive Agents: Verapamil administered concomitantly with oral antihypertensive agents (e.g., vasodilators, angiotensin-converting enzyme inhibitors, diuretics, beta blockers, prazosin) will usually have an additive effect on lowering blood pressure. Patients receiving these combinations should be appropriately monitored. Disopyramide: Disopyramide should not be administered within 48 hours before or 24 hours after verapamil administration. Quinidine: In patients with hypertrophic cardiomyopathy (IHSS), concomitant use of verapamil and quinidine resulted in significant hypotension. There has been a report of increased quinidine levels during verapamil therapy. Nitrates: The pharmacologic profile of verapamil and nitrates as well as clinical experience suggest beneficial interactions. Cimetidine: Two clinical trials have shown a lack of significant verapamil interaction with cimetidine. A third study showed cimetidine reduced verapamil clearance and increased elimination to 1/2. Anesthetic Agents: Verapamil may potentiate the activity of neuromuscular blocking agents and inhalation anesthetics. Carbamazepine: Verapamil may increase carbamazepine concentrations during combined therapy. Rifampin: Therapy with rifampin may markedly reduce oral verapamil bioavailability. Lithium: Verapamil may lower lithium levels in patient on chronic oral lithium therapy. Carcinogenesis, Mutagenesis, Impairment of Fertility: There was no evidence of a carcinogenic potential of verapamil administered to rats for two years. Verapamil was not mutagenic in the Ames test. Studies in female rats did not show impaired fertility. Effects on male fertility have not been determined. Pregnancy (Category C): There are no adequate and well-controlled studies in pregnant women. ISOPTIN crosses the placental barrier and can be detected in umbilical vein blood at delivery. This drug should be used during pregnancy, labor, and delivery, only if clearly needed. Nursing Mothers: ISOPTIN is excreted in human milk, therefore, nursing should be discontinued while verapamil is administered. Pediatric Use: Safety and efficacy of ISOPTIN in children below the age of 18 years have not been established.

ADVERSE REACTIONS: Constipation 8.4%, dizziness 3.5%, nausea 2.7%, hypotension 2.5%, edema 2.1%, headache 1.9%, CHF/pulmonary edema 1.8%, fatigue 1.7%, bradycardia 1.4%, 3° AV block 0.8%, flushing 0.1%, elevated liver enzymes (see WARNINGS). The following reactions, reported in less than 1.0% of patients, occurred under conditions (open trials, marketing experience) where a causal relationship is uncertain; they are mentioned to alert the physician to a possible relationship: angina pectoris, arthralgia and rash, AV block, blurred vision, cerebrovascular accident, chest pain, claudication, confusion, diarrhea, dry mouth, dyspnea, ecchymosis or bruising, equilibrium disorders, exanthema, gastrointestinal distress, gingival hyperplasia, gynecomastia, hair loss, hyperkeratosis, impotence, increased urination, insomnia, macules, muscle cramps, myocardial infarction, palpitations, paresthesia, psychotic symptoms, purpura (vasculitis), shakiness, somnolence, spotty menstruation, sweating, syncope, urticaria Treatment of Acute Cardiovascular Adverse Reactions: Whenever severe hypotension or complete AV block occur following oral administration of verapamil, the appropriate emergency measures should be applied immediately, e.g., intravenously administered isoproterenol HCl, levarterenol bitartrate, atropine (all in the usual doses), or calcium gluconate (10% solution). If further support is necessary, inotropic agents (dopamine or dobutamine) may be administered. Actual treatment and dosage should depend on the severity and the clinical situation and the judgment and experience of the treating physician.

OVERDOSAGE: Treatment of overdosage should be supportive. Beta-adrenergic stimulation or parenteral administration of calcium solutions may increase calcium ion flux across the slow channel, and have been used effectively in treatment of deliberate overdosage with verapamil. Clinically significant hypotensive reactions or fixed high degree AV block should be treated with vasopressor agents or cardiac pacing, respectively. Asystole should be handled by the usual measures including cardiopulmonary resuscitation.

Knoll Pharmaceuticals
A Unit of BASF K&F Corporation
Whippany, New Jersey 07981

knoll®

BASF Group
©1986, BASF K&F Corporation 2474/11/86 Printed in U.S.A.

(*Meetings continued from p 10A*)

AROUND THE NATION

CALIFORNIA

Mar 7–8. **Breast Imaging Update.** 13 Cat 1 Credits. Fairmont Hotel, San Francisco. *Mar 9–13.* **Diagnostic Radiology.** 34 Cat 1 Credits. Fairmont Hotel, San Francisco. *Mar 14–15.* **Contemporary Trends in Diagnostic Nuclear Medicine.** Fairmont Hotel, San Francisco. Info: University of California, Extended Programs in Medical Education, Third and Parnassus Ave, San Francisco, CA 94143. Tel: (415) 476-5731.

Mar 8–13. **8th Annual Mammoth Mountain Emergency Medicine Ski Conference.** 20 Cat 1 Credits. Mammoth Lakes. Info: Daniel L. Abbott, MD, Program Director, Medical Conferences, Inc, PO Box 52-B, Newport Beach, CA 92662. Tel: (714) 650-4156.

Mar 12–14. **Monoclonal Antibody Immunoconjugates for Cancer.** 19½ Cat 1 Credits. Hotel Inter-Continental San Diego, San Diego. Info: Cynthia Saxe, Continuing Medical Education, M-017, University of California, San Diego, La Jolla, CA 92093. Tel: (619) 534-3940.

Mar 18–21. **3rd National Leukemia Society of America Symposium.** 17 Cat 1 Credits. Town and Country Hotel, San Diego. Info: Medical Programs Department, Leukemia Society of America, 733 Third Ave, New York, NY 10017. Tel: (212) 573-8484.

COLORADO

Mar 2–6. **Internal Fixation in Hand Surgery.** 32 Cat 1 Credits. Vail. Info: American Society for Surgery of the Hand, 3025 South Parker Rd, Suite 65, Aurora, CO 80014. Tel: (303) 755-4588.

Mar 7–14. **Decisions in Surgery.** 20 Cat 1 Credits. Snowmass Conference Center, Snowmass. Info: University of California, Extended Programs in Medical Education, Room U-569, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

Mar 22–28. **Practical Therapeutics for Internists and Family Practitioners.** 24 Cat 1 Credits. Keystone Resort,

NOW THE CHOICE IS YOURS.

- **Choose Summit Health Ltd., an established, successful organization committed to building the practices of its affiliated physicians.**
- **Choose a plan for success, provided by Summit to new or relocating physicians. It includes:**
  - **Guaranteed Income**
  - **Office Rent**
  - **Staff Salary Support**
  - **Marketing Assistance**
- **Choose a great location.**
  Private practice opportunities are available in the states and abroad. We have facilities in Arizona, California, Colorado, Iowa, Texas and internationally. Modern and fully accredited, our hospitals are utilized by both MDs and DOs.

  For immediate information, call collect **(213) 201-4000**, and ask for Physician Recruitment.

Please visit us at booth 41 at the annual meeting of the New York Medical Society.

Summit Health Ltd.
SUMMIT HEALTH Ltd.
1800 Avenue of the Stars, Suite 1200
Los Angeles, California 90067 • (213) 201-4000

Keystone. Info: University of Colorado School of Medicine, Office of Continuing Medical Education, 4200 East 9th Ave, Box C-295, Denver, CO 80262. Tel: (303) 394-5195.

FLORIDA

Mar 16–20. **The Physician in Management.** 31 Cat 1 Credits. Sheraton Sand Key, Clearwater Beach. Contact: Sherry Mason, American Academy of Medical Directors, 4830 W Kennedy Blvd, Suite 648, Tampa, FL 33609. Tel: (813) 873-2000.

HAWAII

Mar 7–14. **Topics in Internal Medicine for Primary Care Physicians.** 20 Cat 1 Credits. Molokai Sheraton, Molokai. Info: University of Colorado School of Medicine, Office of Continuing Medical Education, 4200 East 9th Ave, Box C-295, Denver, CO 80262. Info: (303) 394-5195.

Mar 9–13. **Critical Issues in Primary Care.** 20 Cat 1 Credits. Waiohai Hotel, Poipu Beach, Kauai. Contact: Valerie Murray, The Pacific Institute of Continuing Medical Education, PO Box 1059, Koloa, Kauai, Hawaii 96756. Tel: (808) 742-7471.

Mar 16–21. **Diagnostic Imaging 1987.** 24 Cat 1 Credits. Waiohai Hotel, Poipu Beach, Kauai. Info: Radiology Postgraduate Education, University of California, Third & Parnassus Ave, Room C-324, San Francisco, CA 94143. Tel: (415) 476-5731.

ILLINOIS

Mar 8–13. **Annual Meeting of the United States–Canadian Division of the International Academy of Pathology.** Palmer House, Chicago. Info: Dr Nathan Kaufman, Secretary-Treasurer, United States-Canadian Division of the International Academy of Pathology, Bldg C, Suite B, 3515 Wheeler Rd, Augusta, GA 30909. Tel: (404) 733-7550.

Mar 18–20. **Recent Advances in Gastroenterology and Hepatology.** 23 Cat 1 Credits. Drake Hotel, Chicago. Info: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175. Tel: 1-800-523-1546.

LOUISIANA

Mar 26–28. **Basic Microsurgery for the Gynecologist.** 20 Cat 1 Credits. Southern Baptist Hospital, New Orleans. Info: American College of Gynecologists Registrar, 600 Maryland Ave, SW, Suite 300 East, Washington, DC 20024-2588. Tel: 1-800-533-8811.

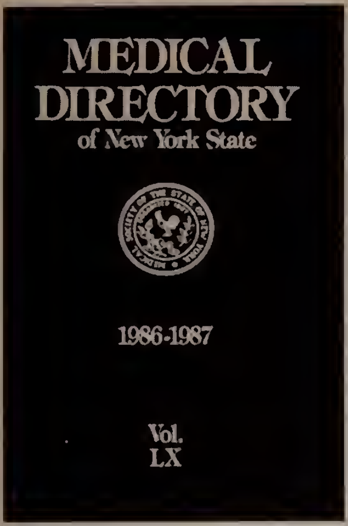
MARYLAND

Mar 6–12. **2nd International Conference on Anticarcinogenesis and Radiation Protection.** Gaithersburg. Info: Michael G. Simic, PhD, Conference Chairman, Room C216, Bldg 245, National Bureau of Standards, Gaithersburg, MD 20899. Tel: (301) 921-2374.

NEBRASKA

Mar 30–Apr 10. **Family Practice Review—A Comprehensive Update.** Oma-

(*continued on p 21A*)



ANNOUNCING THE 1986-87 Edition of the MEDICAL DIRECTORY of New York State

Published biennially, by the Medical Society of the State of New York, this important 920 page, reference source is now available.

The Directory lists over 27,000 New York State Physicians, both members of the Medical Society, and non-members who chose to be listed for a fee. It includes the following data.

SAMPLE LISTINGS

★**FARNSWORTH, Peter Birger,** 420 Lakeville Rd 11042. Tel 488-6100. MCGILL 59. D-P (PDC). Am Ac Ped, Dir Div Scientific Activities Med Soc SNY. Also North Salem (AMA)

★**FINEBERG, Henry Irwin,** 420 Lakeville Rd 11042. Tel 488-6100. YALE 27. Am Thor. Am Pub Hlth, Ac Med. Exec Vice Pres Emerit Med Soc SNY. Also Dix Hills.

★**LAWRENCE Jr, George J,** 420 Lakeville Rd 11042. Tel 488-6100. PENN 38. D-OG. SL. FACOG. FACS. Exec Vice Pres Emerit Med Soc SNY. Hon Chair O&G Flushing. Also Glen Cove (AMA)

★**O'CONNOR, Robert James,** 33 Valencia Ave 10301. Tel 447-7242. STATE UNIV BKLN. D-OG SL. FACOG. FICS. Am Abdominal Sur. Am Ac Comp Med. Attd O&G Staten Isl. Cons O&G USPHSH SI. Attd Gyn Sea View. Also Lake Success. (MEM-AMA)

☐

FLUSHING HOSPITAL AND MEDICAL CENTER

45th Ave. and Parsons Blvd. Flushing. 11353. Tel 6705000. Voluntary, General. 424 Beds. Maintains Registered School of Nursing. Exec. Dir. Philip A. Palese, Dep. Exec. Dir. Michael Marigliano, Asso. Dir. Gen. Serv. Morton Beckmen, Asso. Dir. Nursing Serv. Mrs. Mary G. Gamer, R.N., Asso. Dr. Fiscal Serv. Alen Currier, Dir. Corporate and Med. Stsff. Serv. Patricia DelGiorno.

Emeritus Staff

DENTISTRY—L. Schwartz
MEDICINE—J. P. Burghardt, E. J. Carey, B. A. Glass, J. Iraci, S. Iraci, H. Kalenderian, P. J. Lacovara, T. Nyilas, L. Richeimer, W. Seitz.
  ALLERGY—A. Notaro.
  ENDROCRINOLOGY—C. Delit.
  GASTROENTEROLOGY—A. Weiss.
  NEUROLOGY—S. Karlan
OBSTETRICS & GYNECOLOGY—E. Kiss, V. A. PArnell, M. Roland.

- Cities, towns and counties in which the physicians practice
- Hospital affiliations, Fellowships, and Board certifications
- Medical School and date of graduation
- Up to three office addresses and telephone numbers
- Separate list of hospitals with medical staffs identified.

Of course, the Directory is distributed free to members of the State Medical Society who will automatically receive the Directory when published. A special price of $40 per copy is offered to non-member physicians who chose to be listed, and to all others the price is $70 per copy. The Directory is the most comprehensive, up-to-date desk reference source on physicians in New York State, and is constantly utilized by physicians, libraries, and others in the health care field.

MEDICAL DIRECTORY ORDER FORM for non-members, or members requesting additional copies. (1986-87 Edition)

Name________________________________

Address______________________________

City/State/Zip_________________________

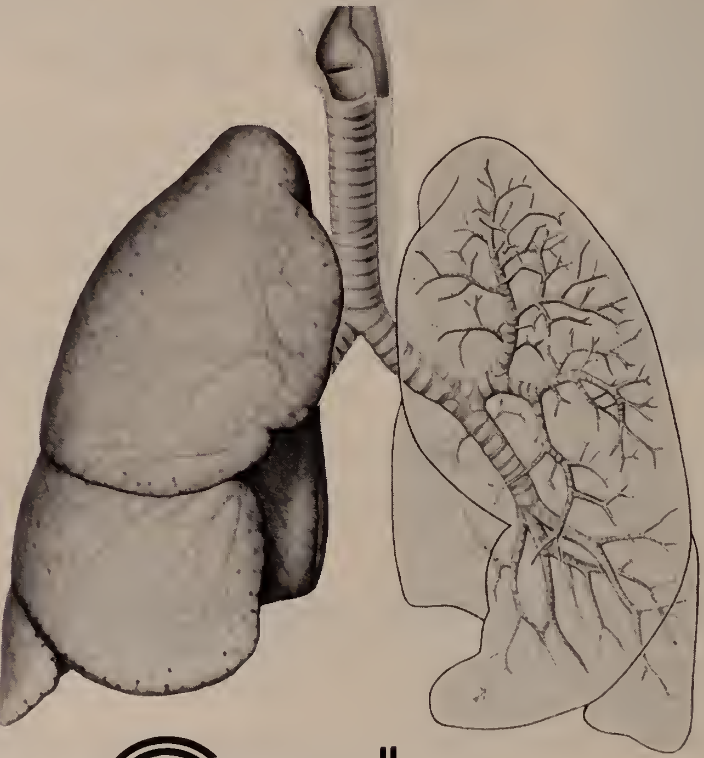
Please send me the following:

( ) copies @ $40 $__________
(*Limit three copies per listed physician*)
Plus applicable sales tax __________
Total enclosed $__________

( ) copies @ $70 $__________
Plus applicable sales tax __________
Total enclosed $__________

Make check payable to: Medical Society of the State of New York, and mail to: Accounting Dept. Medical Society of the State of New York, 420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042.

Consider the causative organisms...



Ceclor® cefaclor

250-mg Pulvules® t.i.d.

offers effectiveness against the major causes of bacterial bronchitis

Haemophilus influenzae, H influenzae, Streptococcus pneumoniae, Streptococcus pyogenes
(ampicillin-susceptible) (ampicillin-resistant)

Note: Ceclor® is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

Ceclor® (cefaclor)

Summary. Consult the package literature for prescribing information.

Indications: Lower respiratory infections, including pneumonia, caused by susceptible strains of *Streptococcus pneumoniae, Haemophilus influenzae,* and *S. pyogenes* (group A beta-hemolytic streptococci).

Contraindications: Known allergy to cephalosporins.

Warnings. CECLOR SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY. POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS.

Administer cautiously to allergic patients.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea. Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.

Precautions:
- Discontinue Ceclor in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of nonsusceptible organisms.
- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- In renal impairment, safe dosage of Ceclor may be lower than that usually recommended. Ceclor should be administered with caution in such patients.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk. Exercise caution in prescribing for these patients.

Adverse Reactions: (percentage of patients)

Therapy-related adverse reactions are uncommon. Those reported include:
- Gastrointestinal (mostly diarrhea): 2.5%.
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
- Hypersensitivity reactions (including morbilliform eruptions, pruritus, urticaria, erythema multiforme, serum-sickness-like reactions): 1.5%; usually subside within a few days after cessation of therapy. These reactions have been reported more frequently in children than in adults and have usually occurred during or following a second course of therapy with Ceclor. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.
- Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.
- Other: eosinophilia, 2%; genital pruritus or vaginitis, less than 1%.

Abnormalities in laboratory results of uncertain etiology
- Slight elevations in hepatic enzymes.
- Transient fluctuations in leukocyte count (especially in infants and children)
- Abnormal urinalysis; elevations in BUN or serum creatinine
- Positive direct Coombs' test
- False-positive tests for urinary glucose with Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (glucose enzymatic test strip, Lilly)

© 1986, ELI LILLY AND COMPANY [060485LR]

Lilly

Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285

Eli Lilly Industries, Inc.
Carolina, Puerto Rico 00630

600332

NEW YORK STATE
JOURNAL OF MEDICINE

February 1987 • Volume 87, Number 2

COMMENTARIES

Evaluating the impact of the 1986 New York State liability legislation

On July 8, 1986, Governor Cuomo signed into law the Medical Liability Reform Act of 1986, which had been passed by the legislature in late June.[1] This law was unique in that it changed the methodologies utilized by the New York State Department of Insurance in setting rates for medical malpractice insurance.

In the past, insurance companies submitted a proposed rate to the Superintendent of Insurance which was either approved or disapproved. For the first time, the 1986 Act authorizes the Superintendent to set rates. Before the bill was signed into law, the Superintendent announced rate increases of 14% retroactive for the premium year 1985–1986, 9% for 1986–1987, and 9% for 1987–1988. The 14% increase retroactive to July 1, 1985, and the 9% increase for the period beginning July 1, 1986, were imposed effective immediately. Together they constitute a 24% increase which is added to a previous increase of 32%—with an additional 9% due to be imposed on July 1, 1987.

There is much in the 42-section bill and the companion legislation enacted at the same time that bear on the practice of medicine in New York State.[1,2] Premium rate increases have been set prospectively for three years. Now, for the first time, the Superintendent of Insurance is empowered to set rates, rather than merely approve them retroactively. However, this authority applies only to the three policy years from July 1, 1985, to June 30, 1988.

The liability insurance rates in New York State are higher than those in most of the other states except Florida, and yet, these increases are characterized by some as inadequate to meet the rising costs of medical liability under the current tort system. Indeed, former New York Court of Appeals Judge Matthew Jason, hearing officer in the Insurance Department's rate hearings last summer, concluded that a 90% premium rate increase was required for just the 1985–1986 period.[3]

Governor Cuomo, in signing the 1986 law, stated that he believed that the tort changes included in it, and in the reform law signed the previous July, would ultimately stabilize the liability system. But he added that "if these bills prove to be only the penultimate steps toward solving the malpractice problem," he would be ready, based on the study authorized in Section 39 of the new law, to implement a more thorough and fundamental restructuring of the medical and dental liability system.[4]

Many of us are convinced that the two laws (1985 and 1986) will prove inadequate to solve the crisis. This is precisely why the Medical Society of the State of New York (MSSNY) is actively involved in the study that was authorized under Section 39.[1] This section requires that a study of alternatives to the tort system be undertaken by the Superintendent of Insurance and the Commissioner of Health, in consultation with the state medical society.

At the present time, the Department of Health has contracted with Harvard University's School of Public Health to conduct a study of untoward outcomes in three Albany-area hospitals—Albany Medical Center, St Peter's Hospital, and Glens Falls Hospital. The pilot phase of the study will attempt to identify untoward outcomes, regardless of negligence, from approximately 1,000 patient records in the three hospitals. It will seek to determine the costs of these untoward effects and their impact on patients, physicians, and hospitals, and to determine collateral sources of economic subsidization for insured vs uninsured patients. A report on this initial pilot phase will be submitted in March 1987. After preliminary data have been collected and the study protocols have been tested and evaluated, a more extensive statewide survey based on 20,000 patient records will be undertaken. It is anticipated that this second phase of the study will be completed by June 1988, when the controlled premium expires. Following successful completion of the second phase of the study, an attempt will be made to develop economic models as the basis for a new compensatory system that might be applied on the state and national levels.[5] Harvard University is concurrently conducting a similar study in Boston. The results of both studies will be compared, which will yield valuable data on exactly what costs are involved and whether or not some system other than the present tort system for compensating injuries would be more efficient.

In a sense this study represents a landmark, since nothing on this scale has ever been done before. When both phases are completed, there might be a real basis on which to judge which alternatives to the tort system would be feasible and affordable. Existing laws will not end the problem, thus alternatives must be found. From the medical profession's point of view, it is crucial to cooperate diligently in the gathering of needed data and then to move quickly toward a new liability system.

Setting premium rate increases at 14% for 1985–1986, at 9% for 1986–1987, and at 9% for 1987–1988 is arbitrary. The 14%, retroactive to July 1985, and the 9% for 1986–1987, combine for a 24% immediate increase in premiums. Holding down rates by arbitrary percentages makes the underlying insurance reserves even less adequate and makes the need for an alternative system even more urgent.

Free excess insurance coverage of $1 million/$3 million available through hospitals applies only through June 30, 1987, and it will not protect physicians' personal assets. The Medical Liability Mutual Insurance Company (MLMIC) has said that the excess liability insurance could increase the cost of primary liability insurance as the additional money available to claimants drives up the cost of settling claims. This is another reason why a more effective, more permanent solution is needed.

The requirement that plaintiffs' attorneys file a Certificate of Merit stating that they have consulted with at least one physician, and have concluded on the basis of that consultation that there are reasonable grounds to commence an action, sounds good. However, as written, there are too many exceptions. The MLMIC's analysis that this requirement is unlikely to reduce the number of frivolous suits to any large extent is accurate.

Reduction of awards to reflect income taxes that would have been paid had the defendant actually earned the money should have a favorable impact on claim payouts. However, awards for noneconomic losses, such as pain and suffering, would not be reduced in this manner. The MSSNY had sought a $250,000 limit on noneconomic losses.

The expanded arbitration features of the law will not affect most physicians in New York State. Prior to enactment of this section, defendants and plaintiffs were already able to agree to arbitrate one or more of the issues involved in malpractice actions. The new law states that the plaintiff in a case where liability is conceded may "elect" to consent to arbitration of damages within 20 days of a demand by a defendant that he do so. The MSSNY had urged the implementation of a requirement that plaintiffs in such cases *must* arbitrate. That would have made a major difference. The limited arbitration opportunities in the HMO setting will have little overall impact on most physicians, however.

The confidentiality of hospital incident reports and the granting of immunity to physicians who participate in this process, as well as for those physicians who participate in hospital peer review programs, represents a significant step forward. The MSSNY has for many years been seeking immunity from suits for physicians who participate in disciplinary cases, and the inclusion of this section is to be applauded. However, the MSSNY has repeatedly pointed out that the medical liability crisis and physician discipline are largely unrelated. While the MSSNY generally favors the sections of this law that seek to strengthen physician discipline, it is convinced they will have little or no effect in solving the liability crisis.

Two changes that may help considerably are the amendments to the Civil Practice Laws and Rules. The first adds a new code that limits the joint and several liability principles so that a defendant's liability for noneconomic damages is limited to that portion of his responsibility in cases where his liability is equal to or less than 50%. Up to now, a defendant with only minimal involvement in causing damage could be held liable for the full amount of the award in cases where the primary defendant was unable to pay. This new rule could help limit the so-called "deep pocket" approach. There are two drawbacks: First, the party asserting limited liability under this article will have the burden of proving by a preponderance of the evidence just what his equitable share should be. Second, this will apply only to "pain and suffering" and other noneconomic damages but not to damages for lost wages, medical bills, and other real dollar losses.

The second change in the Civil Practice Laws and Rules allows judges to reduce jury awards if they determine the award "deviates materially from what would be reasonable compensation." Previously, judges could reduce awards only if they were so high as to "shock the conscience." It is hoped that appellate courts will use this broader power to review and reduce excessive money judgments.

It is interesting to look at the 1986 tort law changes through the eyes of a trial lawyer. Writing in the September 11, 1986, edition of *The New York Law Journal*, Aaron J. Broder, a former president of the New York State Trial Lawyers Association, described the reforms as "an unmitigated disaster . . . In surveying what the Legislature has done, the cliche of a bull in a china shop comes to mind."[6]

Broder said the changes are "a humiliating capitulation to the insurance lobby by the Legislature, and if left as they now are by the Legislature and by the courts, will represent the sunset of personal injury litigation as we have known it in New York. Two hundred years of carefully developed legal doctrine have been trashed by the state legislature in two months. The result is a confusing, poorly drafted, wasteful, and heartless statute which is just about the worst piece of legislation we have seen in the area of tort law in this generation."[6]

All of this raises the questions: How could legislation with such sweeping impact on medical and other liability have so little impact on the cost of liability insurance? How is it possible that medical liability insurance premiums could actually go up by a large, but still grossly inadequate, percentage at the same time that the sun is setting on "personal injury litigation as we have known it . . ."?[6]

With regard to the search for alternatives, it is interesting and instructive to recall the landmark report of the McGill Commission in January 1976,[7] because that report did more than recommend reforms of the tort system. It said such reforms are of only "short term" importance since they "do not go to the heart of the matter, and will not, therefore, prevent unacceptable cost rises and an eventual breakdown of the system."[7(p3)]

With the notable exception of limits on noneconomic damages, the key McGill reforms have been enacted in New York State in the ten years since the report was submitted to Governor Carey, and they have not prevented unacceptable cost rises. Indeed, the reforms have not gone to the heart of the matter, and system breakdown is no longer a remote eventuality.

If the McGill Commission was correct, and history suggests strongly that its crystal ball was exceptional, we will achieve a "full and complete solution" of the medical liability crisis not through tort reforms but by a complete departure from the present system. This is the focus of the project mandated in Section 39 of the 1986 law.[1]

In summary, while there are several worthwhile elements in the 1986 law, as there were in the law enacted in 1985, the liability crisis in New York State has not been solved. The Medical Society of the State of New York has vigorously communicated this view to the legislature and to the governor and will continue to do so.

It is extremely important for the MSSNY to continue to participate in the study of alternative medical compensation systems mandated by the 1986 legislation. Hopefully something can be accomplished before the system entirely collapses, which will have a concomitant devastating effect on the availability of health care services in New York State.

DONALD F. FOY
Executive Vice President
Medical Society of the
State of New York
Lake Success, NY 11042

1. 1986 New York Laws, chapter 266.
2. 1986 New York Laws, chapters 220, 682.
3. Medical liability legislation signed; MSSNY to participate in search for a new system. *The News of New York* 1986; 41(14):2-4.
4. Governor's Memorandum of Approval, no. 16, 1986 New York Laws, chapter 266.
5. Medical Practice Study Group: *Hospital Record Screening Manual for Medical Practice Study*. Boston, Harvard School of Public Health/Harvard Law School, November 1, 1986.
6. Broder AJ: Trials, tactics, techniques: Legislative bombs. *New York Law J* 1986; 196(51):1-2.
7. *Report of the Special Advisory Panel on Medical Malpractice*. New York State, January 1976.

Ruptured diaphragm: A diagnostic dilemma

In this issue of the *Journal*, Sperber[1] presents a case of ruptured left hemidiaphragm with bowel herniation. The case illustrates the confusion and delay in medical management that commonly occur when the diagnosis of diaphragmatic rupture is not considered because the injury is indirect or remote from the time of presentation. The case further illustrates the difficulty of making a correct diagnosis when the results of various radiologic examinations are not correlated. Part of the problem in diagnosis stems from the difficulty in obtaining direct radiographic visualization of the diaphragm by any modality under pathologic conditions.

In the United States, 1.7 million people are injured annually in automobile accidents.[2] Rupture of the diaphragm is present in approximately 4.5% of accident victims who are subsequently hospitalized[3] and occurs on the left side in 90–95% of cases when the injury is due to blunt trauma.[4] Early correct diagnosis of rupture of the diaphragm has been reported in fewer than 50% of cases.[5] The mortality rate may be as high as 30% when rupture is associated with strangulation of the bowel.[6] The diagnosis of a ruptured diaphragm is more readily made when the injury is recent, left-sided, and large, and when herniated structures are readily identified. However, a high index of suspicion must be maintained and appropriate radiologic studies must be ordered.[7] The diagnosis is less likely to be made when the time of injury is remote, the tear is small or right-sided, the injury is indirect, and radiologic studies are not correlated. Fifty percent of patients with ruptured diaphragm due to blunt trauma have associated pelvic fractures.[6] Though the reverse is not true, a past history of pelvic fracture in a patient with unclear abdominal symptoms or findings should suggest the possibility of diaphragmatic rupture.

The normal diaphragm is well visualized on chest radiography. However, the diaphragmatic outline will be obscured when a small amount of fluid or atelectasis is present. Both of these are common findings in trauma patients with or without ruptured diaphragms, thereby reducing the value of chest radiography as a diagnostic tool. Other pathologic conditions mimicking findings of ruptured diaphragm have been addressed by Dr Sperber.[1]

The ease with which many organs and structures can be evaluated, even in very sick patients, has made the use of computed tomography (CT) increasingly common in cases of acute trauma as well as in complex medical problems. The normal diaphragm can be directly visualized on CT scan as a curvilinear, almost semicircular, structure extending bilaterally in a posterolateral direction from the descending aorta. Due to the normal domed shape of the diaphragm, any structure seen peripheral to it is intrathoracic. The finding on CT scan of bowel loops or fat peripheral to the diaphragm allows a diagnosis of herniation into the chest, even when the tear itself cannot be identified. In acute trauma, the presence of blood in the pleural space frequently obscures the outline of the diaphragm. This problem may be alleviated by introducing a chest tube with suction prior to CT. Focal or complete inversion of the diaphragm may occur with empyema and other intrathoracic disease. Inversion reverses the relative position of the abdomen and thorax in relation to the diaphragm on CT image. The normal diaphragmatic contour as seen on successive distal scans increases in size but decreases with inversion. When the diaphragm is not identified on CT scan, it is not possible to distinguish between diaphragmatic rupture and elevation. The contour is lost when the diaphragm is in contact with normal abdominal organs such as the liver, spleen, and stomach wall, but an intact diaphragm may be inferred when the diaphragmatic contour can be identified between organs and forms a smooth curve. The diaphragm is well outlined when adjacent to air, serous fluid, or fat. Herniated omentum and mesentery often contain a large amount of fat, which is helpful in outlining the contour of the diaphragm as well as in determining herniation. The organs herniating through a diaphragmatic rent, in order of frequency, are the omentum, the stomach, the colon, the spleen, the liver, and the small bowel.[8] Computed tomography is the only modality that can detect herniation when the omentum is the only structure to herniate. No CT diagnosis of a ruptured diaphragm has been reported prior to herniation but congenital diaphragmatic defects without herniation have been identified.

Herniation of the bowel can usually be detected on barium studies. The fundus of the stomach has an amputated appearance whereas the colon and small bowel show two limbs of a loop, approximated, with an area of constriction in each corresponding to the site of the tear. It is unusual to mistake the areas of constriction on barium enema for colon cancer, as in the case reported by Sperber,[1] since two areas of constriction can usually be recognized. With complete obstruction, however, only one site may be seen. When the rent is large there may be no deformity on barium studies.

Induction of pneumoperitoneum is used by some to detect a ruptured diaphragm. Air in the peritoneal cavity will migrate into the thoracic cavity due to the normal negative pleural pressure and produce a pneumothorax. Multiple injuries frequently prevent the use of erect films needed to detect a small penumothorax. A false negative examination may occur when the rent is occluded by herniating structures. A persistent congenital communication between the pleural and peritoneal cavity may give rise to a false positive interpretation. The danger of air embolism as well as of false negative and false positive examinations has rendered induced pneumoperitoneum an obsolete procedure at our hospital.

The posterior portion of the right hemidiaphragm is well visualized using ultrasonography. However, the left side is frequently obscured by bowel gas and the anterior portion is poorly visualized on either side due to lack of a sonographic window.

Partial herniation of the liver may also be detected by technecium liver spleen scan when an abnormal constriction is seen in the liver.

Because of the difficulty in visualizing the diaphragm, the diagnosis of rupture will most times be made only when it is directly considered and radiologic examinations are correlated. The possibility of a ruptured diaphragm must be actively considered in all cases in which the diaphragmatic outline cannot be seen, regardless of the patient's history, or there will inevitably be a detrimental delay in diagnosis.

ELISABETH HEIBERG, MD
Associate Professor
Department of Radiology
St Louis University
Medical Center
St Louis, MO 63104

1. Sperber RG: Diaphragmatic hernia mimicking pyopneumothorax and colonic mass. *NY State J Med* 1987; 87:122-123.
2. Carraro B: Traffic deaths decline slightly in 1985. *Traffic Safety* 1986; 84(3):20-24.
3. Epstein LI, Lempke RE: Rupture of the right hemidiaphragm due to blunt trauma. *J Trauma* 1968; 8(1):19-28.
4. Ramstrom S, Alsen S: Diaphragmatic rupture following abdominal injuries. *Acta Chir Scand* 1954; 107:304-308.
5. Grimes OF: Traumatic injuries of the diaphragm. Diaphragmatic hernia. *Am J Surg* 1974; 128:175-181.
6. Grage TB, MacLean LD, Campbell GS: Traumatic rupture of the diaphragm. A report of 26 cases. *Surgery* 1959; 46:669-681.
7. Ball T, McCrory R, Smith JO, et al: Traumatic diaphragmatic hernia: Errors in diagnosis. *AJR* 1982; 138:633-637.
8. Mattila S, Järvinen A, Matilla T, et al: Traumatic diaphragmatic hernia. *Acta Chir Scand* 1977; 143:313-318.

Tuberculosis of the knee

The article by Berson et al[1] on tuberculosis of the knee in this issue of the *Journal* reemphasizes the fact that tuberculosis is not a disease of the past, but must be a consideration in the differential diagnosis of any illness. In 1985, tuberculosis of the bone accounted for 2.4% (44 cases) of all cases of tuberculosis reported in New York City (Bureau of Tuberculosis, New York City Department of Health, unpublished data, 1986).

Skeletal tuberculosis develops as a consequence of a focus or foci of tuberculosis elsewhere in the body, primarily in the lungs or lymph glands, wherever the seat of the original primary infection may be. The bacteria reach the knee or other joints via the hematogenous route and implant in the bone or on the synovia. Once this process occurs, they may remain quiescent for many years, cause a contained inflammatory process, or extend directly into adjoining tissues and reenter the bloodstream. Most often, the bacilli remain quiescent for years. Although it may appear that only a single joint or bone is infected, other areas may also be involved, because tuberculosis is a general disease. The case described by Berson et al[1] illustrates these points.

Skeletal tuberculosis causes synovitis, arthritis, or adjoining osteomyelitis. It is not important to determine whether the organism first became implanted on the synovium or the capsule; synovitis is the earliest manifestation. Usually, as the disease progresses, granulation tissue replaces the synovium, with ensuing pannus formation which may erode and destroy cartilage and bone. With involvement of the bone marrow and cancellous bone, demineralization and sequestration occur, but joint surfaces are usually spared. Rarely, there is distant bony spread.

Joint tuberculosis is usually monoarticular. The spine is most frequently involved (50% of cases); the hip (25% of cases) and knee (14% of cases) are the next most common sites. Tuberculosis of the knee presents with pain, swelling, and limitation of movement. No x-ray changes are apparent early on, and the diagnosis must be made by biopsy and culture. Because of the relatively low incidence of tuberculosis, however, few clinicians consider it in the differential diagnosis. This low index of suspicion frequently results in a delay in diagnosis.

Local surgical treatment consists of complete debridement, depending on the extent of the disease. Because tuberculosis is a general disease, however, local treatment alone is insufficient. Chemotherapy must be employed to prevent the spread of disease and to eliminate any other existing foci. Until recently, a three-pronged treatment regimen employing isoniazid, ethambutol, and either streptomycin or rifampin was advocated. The ethambutol would be discontinued after three to six months but the isoniazid and rifampin therapy would be continued for a total of two years. Frequently, isoniazid was continued for a third year. However, since the bacillary load in cases of skeletal tuberculosis is small, two bacteriocidal drugs are now deemed sufficient. Today, the drugs of choice are isoniazid and rifampin. Ethambutol should only be added if the patient comes from a geographic area where primary resistance is suspected. If the subsequent cultures are fully sensitive to isoniazid and rifampin, ethambutol should be discontinued. Isoniazid and rifampin may also be discontinued if and when the cultures prove negative. If the biopsy results are compatible with a diagnosis of caseating granuloma, it is prudent to continue chemotherapy with isoniazid and rifampin for a period of nine months.[2]

ALJE VENNEMA, MD
Director
Bureau of Tuberculosis
New York City Department
of Health
New York, NY 10013

---

[1] Berson BL, Berkowitz DJ, Gilbert MS, et al: Tuberculosis of the knee presenting as internal derangement. *NY State J Med* 1987; 87:123–125.

[2] Treatment of tuberculosis and other mycobacterial diseases: Joint Statement of the American Thoracic Society and the Centers for Disease Control. *Am Rev Resp Dis* 1983; 127:790–796.

Arthur Donaldson Smith, MD

Physician, explorer, naturalist, and diplomat—Part I*

PASCAL JAMES IMPERATO, MD

On July 10, 1895, Arthur Donaldson Smith, MD, of Philadelphia, came in view of the northern end of a great salt lake in East Africa known as Lake Rudolf. He had left Berbera on the Somali coast exactly a year before at the head of a caravan of 82 Somalis[1(p9)] and had traversed over 2,000 miles of wild, unexplored country that today lies in Somalia, Ethiopia, and Kenya. He still had several hundreds of miles of unexplored territory to cross before reaching the coast. Smith wrote of seeing Lake Rudolf: "As I looked upon the bright sheet of water, it seemed to me like a roll of parchment awaiting me—a roll such as I have received on completion of a course of study." [1(p299)] He had reason to rejoice because his expedition was the first to reach the lake from the north and the first to arrive on its shores since it was discovered seven years before, in 1888, by the Austro-Hungarians, Count Samuel Teleki and Lieutenant Ludwig von Höhnel.[2] During the interim several others had set out for the lake but their attempts had failed. Why anyone, including Smith, would risk their lives and subject themselves to enormous hardships in order to fill in gaps on the map of Africa is partially explained by an entry Smith made in his diary, the day he saw the lake for the first time:

> . . . One thinks of the great world far away—how circumscribed it seems. Nature appears so immense out here that the greatness of civilized mankind dwindles by degrees in one's estimation the longer the separation from the artificial continues. But however the absolute greatness of that world of engines . . . may be, I long to see my friends in America and England, even if I do not envy them the regular lives they lead. [1(p290)]

Smith's yearnings for sport and adventure were leavened by his training as a physician and his skills as a naturalist. Because of this, his self-financed trip was no mere trophy hunt. Rather, it was one of the best organized scientific expeditions to go into this part of Africa and one that made major contributions to the fields of geography, ethnology, geology, paleontology, and natural history. Smith not only filled in the blank spaces of East Africa with the names of lakes, rivers, mountains, and villages, but also brought out of this area a treasure trove of scientific materials that eventually came to rest in the British Museum, the University of Pennsylvania Museum, and the Academy of Natural Sciences of Philadelphia.

Smith subsequently organized two other expeditions to East Africa and one to China, served as a US consul in Greece, Turkey, and Mexico, practiced medicine in Philadelphia, and then for the last several years of his career was a general practitioner in a small rural community in northern Pennsylvania. Clearly he was not cast from the usual mold.

EARLY LIFE

Arthur Donaldson Smith was born on April 27, 1866, in Andalusia, Pennsylvania,[3,4] a small community on the Delaware River in Bucks County, close to the city of Philadelphia. Determining his precise date and place of birth proved difficult because both secondary sources and information provided by Smith himself are inconsistent.[5,6] An assumption was made that Smith was Episcopalian and an inquiry made of the Episcopal Church of the Redeemer in Andalusia. This, it turned out, was an outstation of All Saints Episcopal Church in the Torresdale area of Philadelphia at the time of Smith's birth. A search of the baptismal records at All Saints Episcopal Church revealed that Smith was baptized there on September 10, 1866 (Fig 1). His mother and the rector served as sponsors and his date of birth was recorded as April 27, 1866.[3]

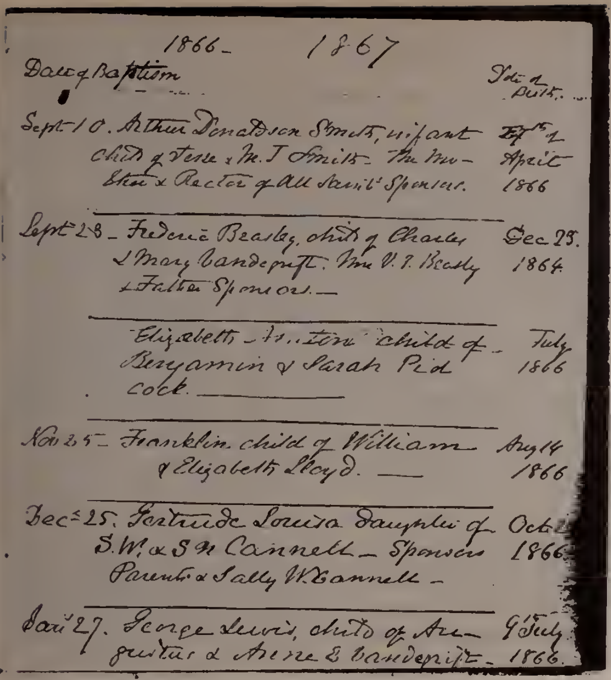
Smith was born into a patrician Philadelphia family, the youngest of three children. His older brother was Henry Cavalier Smith, who married Lucy Pancoast Miller, and his sister Agnes married Graham Spencer of Chester County, Pennsylvania.[7,8] His father, a wealthy Philadelphian, was Jesse Evans Smith, and his mother was Martha James Knight.[4,9,10] Smith's great-grandfather, Joest (Yost) Smith, Jr, was a member of the Revolutionary Army, serving in the Richland Company of Bucks County. His great-great-grandfather, Joest Smith, Sr, "died of the fever taken from soldiers living in his house for whom he was caring" during the encampment of the army at Valley Forge.[9]

Smith's family name was originally Schmidt, his ancestors having come from Germany. However, the name was later changed to Smith.[7] He grew up in his family's Phila-

---

* Parts II, III, and IV of this article will be published in the first half of 1987. Author's acknowledgments will appear at the end of part IV.

Address correspondence to Dr Imperato, Editor, *New York State Journal of Medicine*, 420 Lakeville Rd, Lake Success, NY 11042.



1866 – 1867

Date of Baptism
Date of Birth.

Sept 10. Arthur Donaldson Smith, infant child of Jesse & M. J Smith. The Mo- ther & Rector of All Saints' Sponsors.
1st of April 1866

Sept 28 – Frederic Beasley, child of Charles & Mary Vandegrift. Mrs V. T. Beasley & Father Sponsors. –
Dec 23. 1864

Elizabeth – [illegible] child of – Benjamin & Sarah Pid cock. –
July 1866

Nov 25 – Franklin child of William & Elizabeth Lloyd. –
Aug 14 1866

Dec 25. Gertrude Louisa daughter of S.W. & S.M. Cannell – Sponsors Parents & Sally W. Cannell –
Oct 1866

Jan 27. George Lewis, child of Au- gustus & Anne E Vandegrift –
9th July 1866.

FIGURE 1. Baptismal record of Arthur Donaldson Smith (courtesy of the All Saints Episcopal Church, Philadelphia, Penn).

delphia home and at "Wyndlawn," a summer home in Torresdale, in what is now northeastern Philadelphia, not far from Andalusia where he was born.[11] Smith and his sister Agnes developed a keen interest in natural history early in life.[7] He learned to fish, hunt, and ride, sports for which he maintained a great liking all his life.[7] While his brother Henry Cavalier became a fine horseman, Smith excelled at fishing and hunting.[7] But his interest in the outdoors went beyond those of a sportsman.[7] He gained empirical knowledge of fauna and flora and of their intricate interrelationships.

As an adult Smith dropped Arthur from his name and referred to himself as either A. Donaldson Smith or as Donaldson Smith. British writers referring to him have occasionally used Donaldson-Smith as his last name, inserting a hyphen between the two. This has created a research problem as references to him are sometimes listed under "D" for Donaldson-Smith. Under "Smith," reference sources variously list him as Arthur, Arthur D., A. Donaldson, A.D., or Donaldson. His brother also dropped the name Henry and used H. Cavalier instead.

UNDERGRADUATE EDUCATION

In 1881, at the age of fifteen years, Smith entered the University of Pennsylvania.[12] The university gave an entrance examination at that time in Greek, Latin, English, geography, history, and mathematics including algebra.[13] The requirements for the four-year AB degree included courses in Greek, Latin, English, mathematics, physics, German, inorganic and organic chemistry, logic, history, philosophy, astronomy, and social sciences.[13] Elective courses were available to juniors and seniors in French, German, Latin, Greek, pure mathematics (integral calculus or differential calculus) and American history.[13]

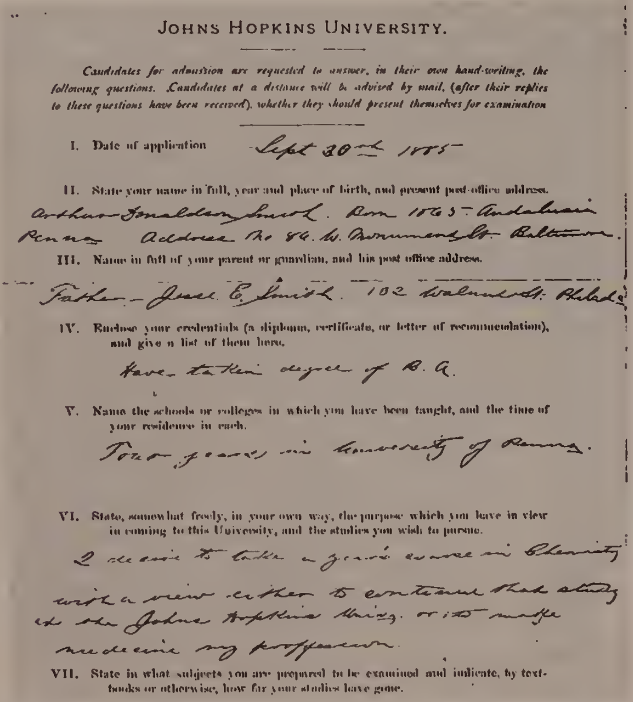
The medical school at Pennsylvania was among the best in the country, whose entrance requirements were on a par with those of Harvard.[13,14] However, Smith did not go there directly.

MEDICAL EDUCATION

On September 30, 1885, Smith applied to the graduate chemistry program at the Johns Hopkins University. The application form was quite summary (Fig 2). Of note is the fact that he gave 1865 as his year of birth, advancing his age a year from 19 to 20. He may have done this to facilitate admission, given his young age. This application provides the first documentation that Smith was contemplating the study of medicine. He stated: "I desire to take a year's course in chemistry with a view either to continue that study at the Johns Hopkins University or to make medicine my proffession [sic]." [15]

During his first semester, Smith was enrolled in three courses: general chemistry, taught by Ira Remsen, general biology, taught by Henry Newell Martin, and osteology, taught by James Playfair McMurrich. Laboratory work was an integral part of the courses in general chemistry and general biology.[16] During the second semester, Smith's course load was lighter, he taking general geology under Henry Newell Martin along with a biology laboratory and osteology under James Playfair McMurrich.[16]

Thus far, Smith had attained a higher level of university education than most physicians of the day. As Ludmerer has stated, "medical education was still generally regarded as a business and the number of medical schools continued to proliferate, growing to 100 in 1880, 133 in 1890, and 161 in 1900.[17(p62)] Proprietary schools flourished, providing students with superficial lecture courses, and admission requirements were lax as were those for graduation. In 1869, admission even at Harvard Medical School was open to anyone who could pay the fees.[17(p47)]



JOHNS HOPKINS UNIVERSITY.

Candidates for admission are requested to answer, in their own hand-writing, the following questions. Candidates at a distance will be advised by mail, (after their replies to these questions have been received), whether they should present themselves for examination

I. Date of application Sept 30th 1885

II. State your name in full, year and place of birth, and present post-office address.

Arthur Donaldson Smith. Born 1865 – Andalusia Penna. Address No 84 W. Monument St. Baltimore.

III. Name in full of your parent or guardian, and his post office address.

Father – Jesse E. Smith. 102 Walnut St. Philad

IV. Enclose your credentials (a diploma, certificate, or letter of recommendation), and give a list of them here.

Have taken degree of B. A.

V. Name the schools or colleges in which you have been taught, and the time of your residence in each.

Four years in University of Penna.

VI. State, somewhat freely, in your own way, the purpose which you have in view in coming to this University, and the studies you wish to pursue.

I desire to take a year's course in Chemistry with a view either to continue that study at the Johns Hopkins Univ. or to make medicine my proffession.

VII. State in what subjects you are prepared to be examined and indicate, by text-books or otherwise, how far your studies have gone.

FIGURE 2. Application to the graduate chemistry program, Johns Hopkins University, September 30, 1885 (courtesy of The Ferdinand Hamburger, Jr, Archives, The Johns Hopkins University).

Smith entered the study of medicine at a time and in schools where major educational reforms were underway. As Ludmerer notes, the better trained physicians among whom the influence of the French clinical school remained strong were skeptical of the utility of scientific knowledge in clinical practice. The young laboratory-trained physicians returning from Germany came into conflict with the former.[17(p49)] Fostering reforms was the attempt by American universities to exert influence over medical education. Ludmerer observes that all of these forces came together at four pivotal medical schools: Harvard, Pennsylvania, Michigan, and Johns Hopkins. They were in the vanguard of the reforms and made the "first genuine, lasting improvements in medical education in the United States." [17(p47)]

The Johns Hopkins medical school opened in 1893,[17(p63)] seven years after Smith completed his year of graduate chemistry study in 1886. Although Smith was not yet fully decided on studying medicine when he entered Johns Hopkins,[15] he was clearly more decided about it a year later when he enrolled at Harvard Medical School.[18]

The Harvard that Smith entered had undergone vast and constant reforms for almost 16 years. These reforms in governance, scheduling, curriculum content, finances, and entrance requirements had been implemented beginning in 1871 by Harvard University's then-new president, Charles W. Eliot, over the opposition of the medical school's faculty.[19] Eliot was himself a graduate of Harvard College and a chemist who had learned science in the laboratory and not merely in lectures or from textbooks. Eliot had not only studied chemistry at Harvard, but had also studied in Europe and had observed both French and German medical education.[17(p48)] The French scorned the laboratory and viewed it as of little importance to medical education or practice. The Germans held the opposite view, but it was the French school's attitude that prevailed among Harvard's faculty because they had trained in France.

By the time Smith applied to Harvard Medical School, Eliot's reforms, begun in 1871, had transformed the institution into one of the finest in the country. In 1877, a requirement was established that applicants without an undergraduate degree had to take an entrance examination.[17(p53)] In 1880, six years before Smith entered Harvard, an optional fourth year had been added which became mandatory by 1892. Eliot's nationally known reforms with their emphasis on laboratory experience and quality of undergraduate training had created a medical school for which Smith with his credentials was obviously well qualified. In applying to Harvard, Smith again advanced his age, this time by two years (Fig 3), making himself 22 instead of 20.[18] Again, this may have been done to reduce the risk of nonacceptance because of his youth. Alternatively, it may simply be a transcription error on the record. But the former possibility seems more likely given the fact that Smith had in his own handwriting advanced his age by a year on his application to Johns Hopkins in 1885.[15]

Smith remained at Harvard for two years and then in 1888 transferred to the medical school at the University of Pennsylvania. There is no information in the archives of these two universities concerning the reason for this transfer. He had one more year of study before obtaining his MD degree. The program at the medical school of the University of Pennsylvania to which he transferred rivaled Harvard in quality, for it too had undergone major reforms, beginning in 1877.

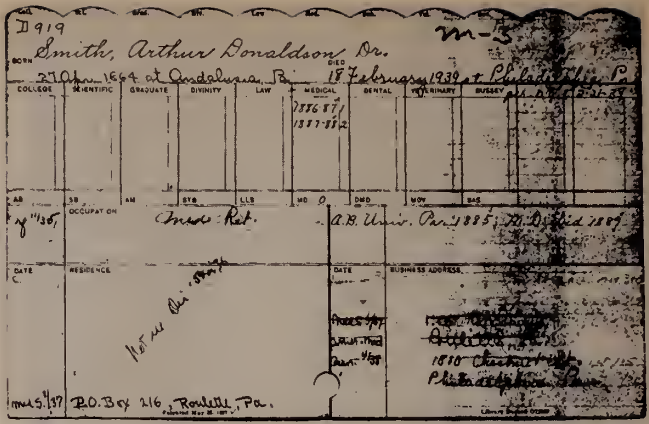

D 919
M-5
BORN Smith, Arthur Donaldson Dr.
27 Apr. 1864 at Andalusia, Pa. DIED 18 February 1939 at Philadelphia, Pa
COLLEGE SCIENTIFIC GRADUATE DIVINITY LAW MEDICAL DENTAL VETERINARY BUSSEY
1886-87 1
1887-88 2
AB SB AM STB LLB MD 0 DMD MDV BAS
OCCUPATION Med: Ret. A.B. Univ. Pa. 1885; M.D. ibid 1889
DATE RESIDENCE DATE BUSINESS ADDRESS
1810 Chestnut St.
Philadelphia, Pa.
P.O. Box 216, Roulette, Pa.

FIGURE 3. Registration record for Harvard Medical School, 1886–1888 (courtesy of the University Archives, Harvard University Library).

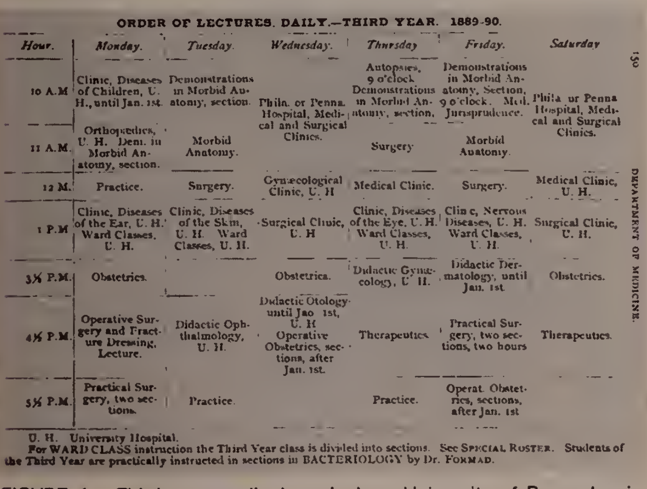
Pennsylvania had considered reforms similar to those implemented at Harvard in the early 1870s, but had procrastinated because of cost concerns.[20] The reforms at Harvard coupled to the stated curriculum design for the new medical school at Johns Hopkins prodded Pennsylvania's trustees into reforming the medical school.[17(p55)] The University of Pennsylvania Medical School that Smith entered in 1888 had much in common with Harvard Medical School. However, the faculty at Pennsylvania expressed concerns in 1887 about the school's ability to compete for high quality students whom they feared would go to Columbia or Harvard.[21(pp88-89)] Thus a Harvard transfer student such as Smith would have been most welcomed when he applied for advanced standing the following year.

The University of Pennsylvania had well-established criteria for transfer students to the School of Medicine when Smith applied. Students who had attended homeopathic or eclectic medical schools were not admitted.[14(pp142-143)] Those applying for advanced standing in the second year had to pass an examination in general chemistry, materia medica and pharmacy, and general pathology. Those such as Smith who wanted to be admitted to the third and final year had to pass an examination that covered the above named subjects plus anatomy and physiology. Arrangements were made for students admitted to advanced standing to make up courses required at Pennsylvania but not given at their previous medical school.[14(pp142-143)]

The first-year courses at Pennsylvania included anatomy, topographical anatomy, histology, materia medica and pharmacy, general chemistry including general physics, physiology, general pathology, hygiene, medical and surgical clinics, and bandaging. The latter was given as one hour of lecture and one hour of practice for half the year. Lectures occupied 18 hours per week and laboratory, clinical, and practical sessions, 19 hours.[14(pp144-145)] Of interest with reference to curricula in medical schools today was the introduction of clinical subjects in the first year.

The second-year curriculum consisted of 21 hours of lectures per week and 29 of laboratory, clinical, or practical sessions;[14(p145)] courses such as anatomy, topographical anatomy, medical chemistry, and physiology were continued as was general pathology to which morbid anatomy was added. Additional courses included physical diagnosis, therapeutics, theory and practice of medicine, surgery, obstetrics, medical and surgical clinics, and specialty clinics.[14(p145)] While Smith did not take any of these courses at Pennsylvania, he took similar ones at Harvard (Harvard Medical School Catalogue, 1886–1887, University Archives, Harvard University Library). His third-year curriculum included 18 hours of lectures and 23 hours of demonstrations and clinical and practical sessions.[14(pp145-146)] General pathology and morbid anatomy were included and two hours per week were devoted to autopsy sessions. Other subjects covered were therapeutics, the theory and practice of medicine, surgery, operative and minor surgery, operative obstetrics, and gynecology, besides instruction in both practical medicine and practical surgery, dermatology, otology, ophthalmology, general medical and surgical clinics at Philadelphia Hospital, medical jurisprudence, and toxicology and specialty clinics at both the university and Philadelphia hospitals[14(p146)] (Fig 4). Students who satisfactorily passed all examinations and met all other requirements were granted an MD degree. A fourth year was offered which was completely practical in nature. Students who opted to take it received a special certificate.[14(p146)] Smith did not take this year, but instead decided to go to Europe.

Smith's academic record during his year at Pennsylvania was quite good, he achieving an 84.5% average for the courses in which he received grades.[12] His examination averages were as follows: materia medica and pharmacy, passed; anatomy, 85; medical chemistry, 80; physiology, 75; pathology and morbid anatomy, 87; therapeutics, 74; practice of medicine, 81; surgery, 45; obstetrics, 95; and gynecology, 70. His only low mark was in surgery, for which he received a grade of 45. However, a careful examination of the grades in surgery for his class of 1889 reveals that in general they are quite low, many being in the 30s through the 50s. The passing grade in surgery appears to have been 40.[12]



ORDER OF LECTURES. DAILY.—THIRD YEAR. 1889-90.

Hour.	Monday.	Tuesday.	Wednesday.	Thursday	Friday.	Saturday
10 A.M	Clinic, Diseases of Children, U. H., until Jan. 1st.	Demonstrations in Morbid Anatomy, section.	Phila. or Penna. Hospital, Medical and Surgical Clinics.	Autopsies, 9 o'clock Demonstrations in Morbid Anatomy, section.	Demonstrations in Morbid Anatomy, Section, 9 o'clock. Med. Jurisprudence.	Phila. or Penna. Hospital, Medical and Surgical Clinics.
11 A.M.	Orthopedics, U. H. Dem. in Morbid Anatomy, section.	Morbid Anatomy.		Surgery	Morbid Anatomy.	
12 M.	Practice.	Surgery.	Gynæcological Clinic, U. H	Medical Clinic.	Surgery.	Medical Clinic, U. H.
1 P.M	Clinic, Diseases of the Ear, U. H. Ward Classes. U. H.	Clinic, Diseases of the Skin, U. H. Ward Classes, U. H.	Surgical Clinic, U. H	Clinic, Diseases of the Eye. U. H. Ward Classes, U. H.	Clinic, Nervous Diseases, U. H. Ward Classes, U. H.	Surgical Clinic, U. H.
3½ P.M.	Obstetrics.		Obstetrics.	Didactic Gynæcology, U. H.	Didactic Dermatology, until Jan. 1st	Obstetrics.
4½ P.M.	Operative Surgery and Fracture Dressing. Lecture.	Didactic Ophthalmology, U. H.	Didactic Otology until Jan 1st, U. H Operative Obstetrics, sections, after Jan. 1st.	Therapeutics	Practical Surgery, two sections, two hours	Therapeutics.
5½ P.M.	Practical Surgery, two sections.	Practice.		Practice.	Operat. Obstetrics, sections, after Jan. 1st	

U. H. University Hospital.

For WARD CLASS instruction the Third Year class is divided into sections. See SPECIAL ROSTER. Students of the Third Year are practically instructed in sections in BACTERIOLOGY by Dr. FORMAD.

150

DEPARTMENT OF MEDICINE.

FIGURE 4. Third-year medical curriculum, University of Pennsylvania School of Medicine, 1889–1890 (courtesy of the University of Pennsylvania Archives).

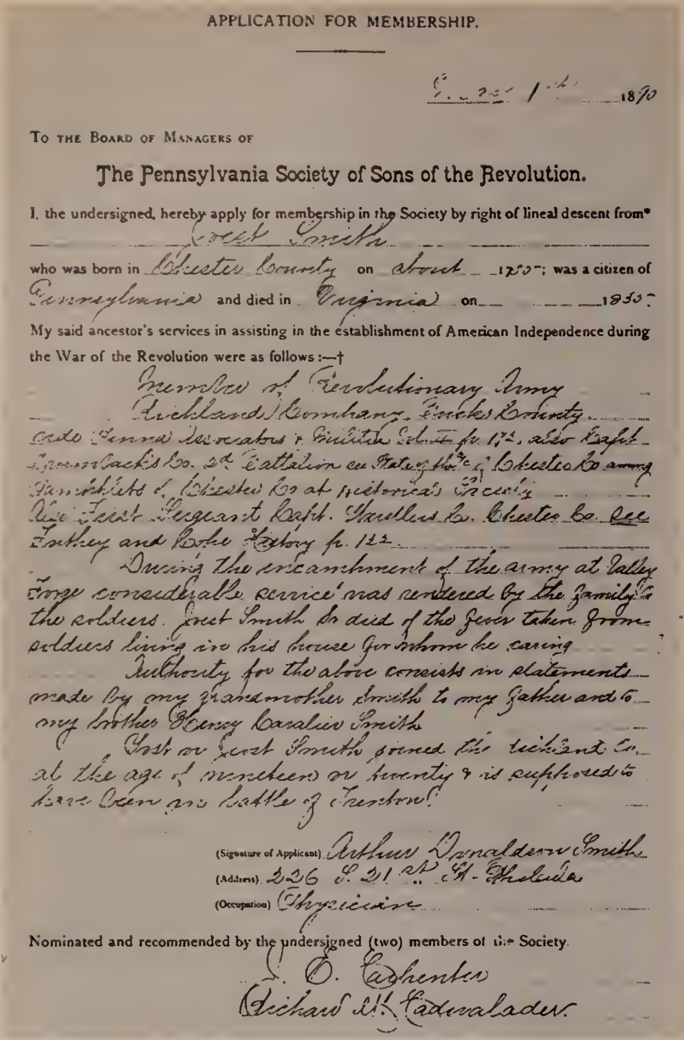
Grades are recorded for Smith for some courses that were given in the first two years and not in the third year when he attended. These include anatomy, medical chemistry, and physiology. The grades for Smith on the record may represent those he obtained in the transfer admission examinations, those from Harvard (it is recorded after his name, "1st and 2nd classes at Harvard Medical School" [12]), or those earned in course work he was required to take at Pennsylvania. The latter seems unlikely given that the full day for the third year was from 10 AM to 6:20 PM, five days a week and half a day on Saturday. Smith would not have been able to take these first- and second-year courses, even as an auditor, given scheduling conflicts.[14(p148)] These grades most likely represent those he obtained at Harvard.

Tuition at Pennsylvania was $150 for the year. In addition, there was a $5 matriculation fee and a $1 fee for material for operating.[14(p153)] During Smith's time at Pennsylvania instruction was given in the Medical Hall, the Laboratory Building, and the Hospital of the University, which in 1888, the year Smith started his third year of study, treated 8,889 cases.[14(p161)] The Medical Hall contained the Wistar and Horner Museum and a number of laboratories. The Stillé Medical Library, founded in 1879, had 6,000 volumes and 5,000 pamphlets. Clinical instruction was given not only at the University Hospital but also at the Pennsylvania Hospital, Philadelphia Hospital, Children's Hospital, and the German Hospital.[14(pp157-163)]

Smith received his MD degree from the University of Pennsylvania along with 123 classmates on May 1, 1889.[14(pp247-249)] A number of other students received their MD degrees a month later on June 5, 1889.[14(p249)] They may have been students who had to make up academic deficiencies. Of the 124 graduates, 46 (37%) were listed as possessing previous university degrees of some kind.[14(pp247-249)] Curiously, no degree is listed after Smith's name, even though he had an AB degree. This raises the possibility that a certain proportion of other graduates also may have had degrees.

After graduation, Smith went to the University of Heidelberg for a year of postgraduate study.[12] By that time both Germany and Austria had become meccas for American physicians seeking exposure to an educational philosophy whose essential tenet was that scientific knowledge and methods were essential to clinical medical practice. Taken for granted today, this represented a sharp departure from the then-existing philosophy in the United States, derived from the French school, that held that such knowledge was irrelevant to clinical practice.[22] The pilgrimage to German universities had begun in 1870 and faded with the outbreak of World War I,[23] even though some Americans continued to go over through the 1920s.[24] By the time Smith went in 1889, the migration of American medical school graduates was in full swing. Prestige and respect among both colleagues and patients accrued to those who returned from a stay at a German or Austrian university.

Smith's mother, Martha Knight Smith, died in March 1890 at the age of 63. It is not known whether or not Smith had returned from Heidelberg by then. However, he was in Philadelphia by May 1, 1890, since he submitted an



APPLICATION FOR MEMBERSHIP.

May 1st 1890

To the Board of Managers of

The Pennsylvania Society of Sons of the Revolution.

I, the undersigned, hereby apply for membership in the Society by right of lineal descent from* Jost Smith who was born in Chester County on about 1750; was a citizen of Pennsylvania and died in Virginia on 1850.

My said ancestor's services in assisting in the establishment of American Independence during the War of the Revolution were as follows:—†

Member of Revolutionary Army (Richland) Company. Bucks County [illegible] Penna [illegible] & Militia [illegible] p. 172. also Capt. [illegible]'s Co. 2d Battalion in [illegible] of Chester Co army [illegible] of Chester Co at historical Society [illegible] First Sergeant Capt. [illegible] Co. Chester Co. See [illegible] and [illegible] History p. 122.

During the encampment of the army at Valley Forge considerable service was rendered by the family to the soldiers. Jost Smith Sr died of the fever taken from soldiers living in his house for whom he caring

Authority for the above consists in statements made by my grandmother Smith to my father and to my brother Henry Cavalier Smith

Jost or Joist Smith joined the [illegible] Co. at the age of nineteen or twenty & is supposed to have been in battle of Trenton.

(Signature of Applicant) Arthur Donaldson Smith
(Address) 226 S. 21st St. Phila
(Occupation) Physician

Nominated and recommended by the undersigned (two) members of the Society.

[illegible] Carpenter
Richard M. Cadwalader

FIGURE 5. Arthur Donaldson Smith's application for membership in The Pennsylvania Society of Sons of Revolution, May 1, 1890 (courtesy of the Society).

application on that date in his own handwriting to the Pennsylvania Society of Sons of the Revolution (Fig 5) and an affidavit in support of his application the following day.[9] These forms were filed from his father's home at 226 South 21st Street, Philadelphia. Compared to most physicians of the day, Smith was superbly trained, in terms of both his premedical, medical, and postgraduate education. Under other circumstances, he might have had a long and successful medical practice career.

Early Career

In 1890, Smith was 24 years old. He was a handsome young man of modest height (about 5′7″) with blue eyes, brown hair, a distinctive moustache which he would later regularly wax, and striking white teeth that impressed people even in his late years[7] (Fig 6). He was soft-spoken, assertive, and patrician in his manners, but never pompous. He was extremely thoughtful and sensitive about other people's feelings, virtues which helped make him highly successful with patients. He was also a natty dresser, a habit he displayed most of his life.[7]

According to some of his obituary notices, Smith practiced medicine at this time.[25-28] Although such notices represent secondary sources, in Smith's case they are highly reliable because they were written from material supplied by his niece's husband, Charles A. Potter, Jr, with whom Smith was extremely close.[7,29] Potter married Margaret Spencer, the daughter of Smith's sister, Agnes Smith Spencer.[7] The relationship between Potter and Smith was described by Potter's son, H. Spencer Potter, in these words: "My father was not really a relation of Uncle Donnie, but the two were extremely close. Uncle Donnie was very fond of my father and treated him as he would have a son." [7] It was Charles and Margaret Potter who would look after Smith in his old age.

There is much similarity in these obituary notices and in some instances the phrasing is identical.[29] The *Philadelphia Record* said of this period of Smith's life, "An inheritance in 1892 enabled him to leave his medical practice and to pursue his love of adventure. He went on a hunting trip to Mexico and then in 1893 tried his hand at big game in Somaliland." [25] The *Philadelphia Evening Ledger* said: "When he inherited a fortune in 1892 Dr. Smith gave up medical practice and set out to satisfy his love of adventure. After a hunting trip to Mexico he sailed[26] in 1893 to hunt big game in Somaliland. *The New York Times* stated it this way: "When he inherited a fortune in 1892 he set out for Mexico on a hunting trip and followed it with a big game hunt in Somaliland." [27] The *Philadelphia Inquirer* used slightly more original wording but essentially said the same thing: "When he inherited a fortune in 1892, Dr. Smith took down the shingle he had hung up in Philadelphia and set out to satisfy his love of adventure. After a hunting trip to Mexico, he sailed in 1893 to hunt big game in Somaliland.[28] Three of these four notices[25,26,28] are quite explicit that he had a medical practice and the one that appeared in the *Philadelphia*

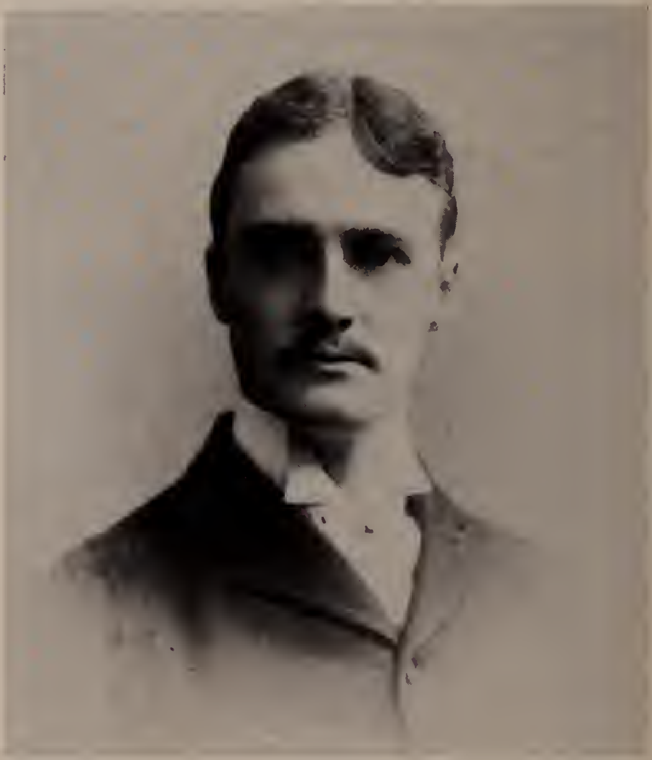


FIGURE 6. Arthur Donaldson Smith as a young man (courtesy of the University of Pennsylvania Archives).

Inquirer[28] is specific about Philadelphia's being the location of the practice.[30] Smith enjoyed performing surgery and was extremely accomplished at it.[7] This is confirmed by the fact that in 1895, while returning to the East African coast, he had to amputate the arm of Yusif, one of his most trusted Somali gunbearers, who had been attacked by a crocodile.[1(p358)] Smith was then on the banks of the Guaso Nyiro River in what is now Kenya. He wrote, "... the poor fellow's arm was nearly torn from him. I was obliged to amputate the arm close to the shoulder. When I was engaged in operating on Yusif's arm, a herd of elephants passed near the camp, but I did not go after them." This area of Kenya is still remote[31,32] and an upper extremity amputation of the kind Smith undertook would be a terrifying prospect for qualified surgeons today if they had to do it under the same circumstances.

In 1892 Smith was 26 years old, and toward the end of that year an event occurred that changed the course of his career. At 4:20 AM on Thursday, November 24, 1892, his father died at his home in Philadelphia,[33,34] where his funeral services took place at noon four days later on Monday, November 28, 1892.[34] The elder Smith, who was 68 years old, died without having made a will. Three days after his funeral, on December 1, 1892, his three children and heirs, Henry Cavalier Smith, Agnes M. Spencer, and Arthur Donaldson Smith, petitioned for and were granted Letters of Administration for his estate.[35]

Both Smith and his older brother Henry Cavalier were living in their father's home at the time.[35] Agnes Spencer, who had married Graham Spencer, a wealthy Philadelphian, was then residing in Kaolin, Pennsylvania, in Chester County, in a residence near the Delaware border where Smith was frequently a guest.[7] In their petition for Letters of Administration, the elder Smith's three children estimated the value of his estate at $200,000, of which half was in real estate holdings.[35] In compliance with one of the provisions set forth in the Letters of Administration, the estate was appraised. The appraisal was submitted on December 20, 1892, by William M. Sharpe and Anthony J. Sharpe. The estate's assets totaled $208,250, of which $26,742.47 was in cash; there was a $37,500 mortgage on the Philadelphia and Reading Railroad, a $10,000 mortgage on the Baltimore and Ohio Railroad, a $15,000 mortgage on the Lehigh Valley Company, and $140,000 in 1,400 shares of stock in the Charleston, South Carolina, M and M Company. Furniture was appraised at $500 as was farm stock, and sundry items of household and private property were valued at $900.[35] Arthur Donaldson Smith's share of this amount would have been close to $79,000 which in 1986 would be worth about $2,600,000. Thus Smith had, as his various obituaries stated, inherited a fortune. It was this inheritance that enabled him to temporarily give up the practice of medicine and embark on a remarkable career as an explorer. It seems unlikely that Smith intended to become an explorer when he stopped practicing. His inheritance enabled him to stop working and to devote himself full time to some of the pursuits of a vanishing gentry. In his case, these included hunting, fishing, traveling, and enjoying the rugged out-of-doors. This may have been the free spirited life he preferred compared to the one he had known growing up in a formal patrician environment.[7] However, he had a highly inquisitive and disciplined mind which, coupled to his medical and scientific training, pulled him into the pursuit of the unknown.

EARLY TRAVELS

It is probable that Smith had traveled widely on hunting and fishing trips even before his father's death. In the opening paragraph of his book, *Through Unknown African Countries*, he wrote, "Many a sporting trip to different parts of the world had taught me what to expect under most diverse conditions." [1(p1)] There is no record of where he went and when. Several of Smith's obituaries speak of a hunting trip he took to Mexico after his father died.[25,28] This most likely occurred in 1893, but no details about it have thus far been uncovered. He next went on a fishing and hunting trip to Norway,[1(p1)] with a friend named William Lord Smith, MD (no relation), of Massachusetts. The latter Smith was three years older than Arthur Donaldson Smith, had received his medical degree in 1892, and practiced in Boston, Massachusetts, until 1916.[36]

The two Smiths must have gone to Norway in early 1893 because Arthur Donaldson Smith returned to New York City from Liverpool on June 24, 1893, aboard the *S.S. Tauric*.[37] While they were in Norway, William Lord Smith told him that he was planning a sporting trip to Somaliland. In his book, Arthur Donaldson Smith wrote the following: "An exploring expedition offered me an opportunity for gratifying all my desires and ambitions." In the next paragraph he said: "... so I joined him with the idea that this preliminary journey would give me the requisite knowledge of the natives and beasts of burden that I intended taking with me when I made my exploring expedition." [1(p1)]

These statements imply that Smith was decided on an exploration expedition into East Africa while in Norway and viewed the hunting trip with his friend there as a preliminary field trial. However, in his presentation before the Royal Geographical Society on January 6, 1896, describing his expedition to Lake Rudolf, he said: "During a sporting trip to Somaliland over two years ago, I conceived the idea that I could carry out an expedition across the large extent of country lying between the Shebeli River and Lake Rudolf ...." [38] This statement implies that he did not decide on the larger expedition until after he arrived in Somaliland. It is probable that Smith gave some thought to the possibility of taking an expedition into the interior while in Norway, but that the final decision to do so was made after he arrived in Somaliland for the sporting trip.

Hunting trips for wealthy Europeans and Americans were extremely common in northern Somaliland in the 1890s,[39(p34)] because the area was teeming with big game. Later, as the wildlife was decimated through over-hunting, hunters moved on to what is now Kenya, which in the early part of the 20th century replaced Somaliland as the principal magnet in Africa for big game hunters.[40(pp137-138)]

On May 4, 1893, Percy Cox (1864–1937), who would later become famous as an administrator and diplomatist in the Middle East, was appointed assistant political resident at Zeila, a port in the British Somaliland Protectorate.[39(p30),41] Cox and Arthur Donaldson Smith later became close friends.

At that time the protectorate was attached to the government of Bombay for administrative reasons, but was immediately dependent on Aden. There was a resident at Berbera, and one at Aden, the two reporting to the political secretary of the Bombay presidency who in turn reported to the government of India.[39(p30)] In the summer of 1894, Cox was transferred to a higher administrative position as assistant political resident at Berbera.[39(p33)] This was relevant to Smith, who began his exploration expedition from Berbera on July 10, 1894.

Philip Graves in his biography of Cox said the following: "Travellers and sportsmen visiting or passing through Berbera provided a difficult financial problem. Some were agreeable and George Curzon, who as Viceroy of India was to assist Cox's advancement four years later and for whom Cox organized a successful shooting trip, Prince Esterhazy, Count Coudenhove, Dr. A. Donaldson-Smith and Lord Delamere left pleasant memories." [39(p34)]

Cox's immediate superior, the resident of Berbera, Lieutenant Colonel William Butler Ferris, said of the sportsmen in his 1895–1896 annual report:

> The number of sportsmen visiting Somaliland does not abate. They add much to the work and personal expenses of the Assistant Resident (Cox) and the want of a traveller's bungalow is more than ever felt . . . Some so-called sportsmen have amused themselves by killing everything they came across, large and small, male and female. [39(p34)]

Thus one of Cox's responsibilities was looking after the sportsmen and travellers, a responsibility that brought him into contact with Smith, perhaps in 1893 when he was at Zeila but certainly in 1894 when Smith set out for Lake Rudolf.

The British first began to exert political influence over northern Somaliland in 1885 after the Egyptians withdrew their garrisons from the three Somali ports of Berbera, Zeila, and Bulhar in 1884. The Egyptians needed their troops to deal with the uprising caused by the Mahdi in the Sudan. The British were not especially anxious to acquire these three ports and attempted in 1884 and 1885 to get Turkey, Egypt's suzerain power, to take them over. It was only after it became apparent that Turkey was unwilling that the British decided to occupy the ports. In addition, the British rapidly concluded treaties with tribal groups in the surrounding areas.[42] Britain's motive for acting was fear that the three Somali ports might fall into the hands of a rival European power. A hostile power in possession of this coast would control the Bab el Mandab, the strait between the Gulf of Aden and the Red Sea, and could potentially wreak havoc on the Suez Canal traffic that linked Britain to her Indian empire and her possessions in Asia and the Pacific. Since the three ports were on the Gulf of Aden along the route to India, the British occupied them. The British proclaimed the Somaliland Protectorate on July 20, 1887.[43,44]

This was a turbulent corner of Africa because of the competition between the colonial powers in the scramble for Africa. Britain's principal rivals were France, Italy, and Germany, with Russia doing whatever she might to impede British interests.[45] On the Horn of Africa the scramble focused on territories surrounding Ethiopia, an independent African power whose Emperor Menelik II had consolidated his power and who was proving adept not only at playing the European powers off against one another, but also in fostering his own plans of imperial annexation of adjacent territories.

In Somaliland the British, French, and Italians had to deal with effective local leaders who were frequently capable of challenging colonial authority. For example, in May 1895, when Smith was well into the interior near Lake Stephanie,[1(p248)] the Rer Hared clan of Somalis closed the trade routes in the British protectorate and began terrorizing the coastal tribes. Percy Cox, who was now a captain, was put in charge of an unauthorized military expedition against them by his immediate superior, Lieutenant Colonel Ferris. At the head of 52 trained Indian and Somali camelry and 1,500 irregulars who were unreliable, Cox defeated the Rer Hared in six weeks, detached their allies from them, and forced them to surrender. This quick war not only established Cox's reputation for ability and decisiveness, but also gave the protectorate peace for several years. Later in 1895, Cox was appointed assistant to the governor-general's agent in Baroda, India, where he would again meet Smith.[41]

On their hunting trip the two Smiths traveled as far south as Milmil, which is about 125 miles from Berbera. This was well within the British Somaliland Protectorate and an area that saw a fair number of European sportsmen. Smith wrote the following concerning this trip: "We had splendid sport, killing six lions, besides many elephants, rhinoceroses, and other big game. But what I valued most was that I was enabled to form my plans for my future expedition through the Galla countries to great advantage." [1(p2)] Smith goes on to say that he perceived that a trip from Berbera to Lake Rudolf would be a difficult and dangerous undertaking. "The preliminary details would require the most careful study, and no expense should be spared in preparing for every possible contingency." [1(p2)] He received a great deal of encouragement from Captain H.G.S Swayne, who had made many trips through Somaliland and had written much about its fauna. Swayne had gotten as far as the Webi Shebeli River on his last trip, some 125 miles beyond Milmil. But he was unable to go further because of the threats of the Galla and the small size of his armed escort of only 40 men. Swayne attributed the Galla hostility to the clashes they had previously had with Captain Vittorio Bottego and Prince Eugenio Ruspoli, two Italian explorers who had explored what are now the border lands between Ethiopia and Kenya. Bottego and Ruspoli had gone through some of the country that Smith proposed to explore, but they had not reached Smith's prize, Lake Rudolf.

Bottego and Ruspoli may have been justified in their use of force, as Smith would be later on with the Boran who unsuccessfully tried to pillage his caravan. But disparaging other explorers, particularly if they were of another nationality, and in the service of a rival colonial power, was an acceptable sport. The fact remained, however, that Ruspoli had gone up the Juba River and its tributaries, to 4° north latitude, and then to the northwest, and was within 150 miles of Lake Rudolf when he was killed by an elephant on December 4, 1893, at Burgi.[46] Bottego had gone as far north as 7° north latitude during his expedition of 1893.[47(p8)] It was just a matter of time before he got to Lake Rudolf.

PREPARATIONS FOR EXPLORATION

When the two Smiths returned to the coast from their hunt on February 1, 1894, Arthur Donaldson Smith began preparations for his expedition to Lake Rudolf. He left orders for a local merchant, Mohammed Hindi, to buy the best camels he could find.[1(p4)] He also engaged many of the Somalis who had been with him on the hunting trip, a wise move since he had already observed them under adverse field conditions. Smith planned to return in the summer to start his exploration expedition. His hunting trip had rightly taught him that a well-equipped expedition could pass through the Galla country provided it was appropriately armed and "especially if patience were exercised, and everything done to conciliate the natives." [1(p3)] Smith realized the need to provide against what he called "little acts of treachery," by taking an armed escort of 70 men. He made preliminary arrangements with Malcome Jones, the British resident at Bulhar, and with Charles McConkey, the agent of Brown, Shipley and Company at Aden, outfitters and freight forwarders.

Smith then returned to Britain where he set to work preparing himself for the geographic and natural history components of his trip. He entered into contact with the Royal Geographical Society whose map curator, John Coles, taught him the various methods for laying down positions. Since the 1850s the leaders of the Royal Geographical Society had become almost obsessed with precise measurements of latitude and longitude and tended to be dismissive of general geographic descriptions. Smith either knew this or was told it so it is not surprising that he equipped himself with surveying skills. The staff of the British Museum instructed him in the best methods for collecting and preserving natural history specimens. "I was determined to spare no expense to make my expedition as complete as possible, so I determined to engage the services of Mr. Edward Dodson, a young English taxidermist, with the idea of his being useful in helping me to collect specimens, as well as in skinning birds." [1(p4)] Smith goes on to say, "When my preparations were nearly completed, I agreed to the request of my friend, Mr. Fred Gillett, that he might accompany me, with twelve men and twenty camels, as he wished to shoot big game and I was desirous of company.[1(p4)] The Royal Geographical Society did not provide Smith with any financial support, but they did lend him several instruments, a six-inch theodolite (an instrument for measuring horizontal and vertical angles), an artificial horizon, boiling-point thermometers, aneroids (barometers in which the action of atmospheric pressure bends a metallic surface and so moves a pointer), and prismatic compasses. Some of Smith's obituaries state that he traveled under the auspices of the Royal Geographical Society, which to a certain degree is true, if one interprets auspices to mean "guidance."

Some secondary sources, namely the *National Cyclopedia of American Biography* and *Who Was Who in America*, state that he traveled with scientific staff provided by the British Museum.[5] This is somewhat misleading since the scientific staff consisted of only one person from the museum, Edward Dodson, the taxidermist, whose expenses and salary were paid for by Smith.

Having spent the spring of 1884 in London planning his trip to Lake Rudolf, Smith set sail for Africa from London on June 1, 1894. He says that "it was the universal opinion, both in London and Aden that it would be impossible to enter the Galla countries, let alone to reach Lake Rudolf, with less than 200 or 300 well-drilled followers. The expression of such opinion served, however, only to increase the zeal I felt in the enterprise." [1(p5)] If Smith succeeded he would be the first European to get to the lake since its discovery in 1888, the first to reach it from the north, and in the process he would traverse a large unexplored gap in the map of Africa. The lure of Lake Rudolf was a powerful one and Smith was not alone in wanting to get there.

Smith's accomplishments as an African explorer can only be fully understood if they are viewed against the backdrop of the larger canvas of events that unfolded both before and during the time he was in Africa. Specifically this requires some understanding of both the Lake Rudolf region and of the people and events that led to the lake's discovery.

THE PRELUDE TO DISCOVERY

Lake Rudolf,[48] which lies at 1,230 feet above sea level, is 155 miles long and 35 miles wide.[49,50(p3)] It is situated in northwestern Kenya, an East African nation that changed the lake's name to Lake Turkana in the 1970s after an ethnic group that lives along the lake's western shore. The principal affluent of this Rift Valley lake is the Omo River, which drains into it via a circuitous course cut through the highlands of western Ethiopia. Historically the lake was much larger than it is today and was supplied by several other river systems that today feed it at most through seepage. On the western shores these are the Kerio, Turkwell, and Suam rivers.[50(p2)] Ten thousand years ago the lake was approximately 200 feet higher than it is today, which means that it covered an enormous area stretching up into the highlands of Ethiopia.[51(p6)] At that time the lake must have been a source of the Nile, a fact attested to by the presence in the lake today of Nile perch, crocodiles, and hippopotami.[51(p6)] The lake shore is a treasure trove of fossils where Richard Leakey, the director of the National Museum of Kenya, has been conducting studies for several years.[51] The level of water in the lake has consistently fluctuated and in recent decades these fluctuations have been measured.[50(pp174-175)] It is a shallow lake compared to some others in East Africa and its waters are alkaline.

Lake Rudolf lies in inhospitable country surrounded by semi-desert, dry scrub, volcanic slag, and soda flats. The ethnic groups that live within its basin include the pastoral Turkana, Rendille, Gabra, Samburu, and Boran. To the north, around the rich agricultural delta of the Omo River, live farming groups among which are the Reshiat, Amar, and Nyanatom.[52(p82)] Despite its size, Lake Rudolf was the last of the great African lakes to be "discovered" by Europeans. Its discoverers, Count Samuel Teleki and Lieutenant Ludwig von Höhnel, arrived on its shores in 1888. However, news of its possible existence had reached Europe much earlier through the reports and writings of missionaries and other travelers, who had usually obtained their information from African traders who were and still are great travelers. But until Teleki and von Höhnel saw the lake it was only a whispered reality for the outside world.

The upper Lake Rudolf basin was first visited by two Portuguese diplomatic emissaries, Antonio Fernandes and Fequr Egzi's, in the late 16th century.[53] The record of their observations of the upper Omo River system was preserved in the 1646 descriptions of the Portuguese Jesuit, Manoel de Almeidsa.[52(pp29,155,157)] In 1843 the brothers Antoine and Arnaud d'Abbadie conducted explorations of the upper Omo.[54] These were followed by exploratory journeys by the Italian explorer Antonio Cecchi[55] and the Frenchman Paul Soleillet. The actual course of the Omo was not definitively determined until 1896 when a second Bottego expedition explored it. However, none of these explorers, except for the last, saw Lake Rudolf.

In 1860, a German missionary in the service of the Church Missionary Society, J. Ludwig Krapf, published his famous book,[56] in which he described his 18 years of experience in living and working in Eastern Africa between 1837 to 1855.[56] A lengthier version of the book had been published in two volumes in German in 1858 under the title, *Reisen in Ost-Afrika*. Krapf had spent six years in Abyssinia and most of the remainder of his time in what is now Kenya. He is best remembered for his discovery of Mount Kenya on December 3, 1849.

On one of the maps published in Krapf's book, entitled "Maps of East Africa" showing the routes of Dr Krapf between the years 1837 and 1855, is a large lake, called Lake Zamburu (Ufole?). This map is an adaptation of the "slug map" elaborated by Krapf's missionary-explorer colleagues, Johann Rebmann who discovered Mount Kilimanjaro on May 11, 1848, and J. J. Erhardt who had also undertaken expeditions into the interior. The latter is credited with drawing the "slug map." This map, which showed an enormous inland sea in central Africa called Lake Uniamesi, was first published in 1855 in a Württemberg missionary magazine in Germany called *Calwer Missionblatt*. It was then published in *Athenaeum* in 1855[57] and in the *Proceedings of the Royal Geographical Society* in 1856.[58] Publication in the *Athenaeum* caused a sensation and debate because the armchair geographers were quick to seize upon it as it was pivotal to the riddle of the source of the Nile.[56(intro p63)] However, this is an issue tangential to this narrative. Of interest is that this map showed a Lake Zamburu about where Lake Rudolf lies. Its placement is at 3° north latitude and 39° east longitude and it is depicted lying in a southwest to northeast direction. While the latitude placement is correct the longitude placement is off by about two degrees. Yet the lake being referred to was no doubt Lake Rudolf. Neither Krapf, Rebmann, or Erhardt had ever been in this region and their information was based on data collected from African traders.

As an aside, it is of interest that the original "slug map" was published in the 1858 German edition of Krapf's book but not in the 1860 English edition. In the latter, Lake Uniamesi, which has the form of a slug, appears only on the map showing Krapf's routes. The sketch map of East Africa that appears in the English edition was drawn by Ernest Georg Ravenstein (1839–1913) of the Royal Geographical Society and seems to incorporate the then-recent discoveries of Burton and Speke that Lake Tanganyika and Lake Nyasa were separate.[59] Yet this map reflected Krapf's own view of the interior lakes in 1855. He had always thought Lakes Tanganyika and Nyasa to be separate and had accepted Erhardt's interpretation of one huge lake in the interior with much reluctance.[56(intro p63)]

On the map drawn by Ravenstein, Lake Zamburu does not appear but Victoria Nyanza does in an undefined way (Speke had yet to fully explore it). The next mention of a lake in the area of Lake Rudolf was made by Leon des Avanchers, a Capuchin missionary from Savoy who gathered much geographic information about the interior while working on the Somali coast in Brava. His extensive findings were gleaned from caravan chiefs on the coast and in particular from Hadji Abul-el-Nour, the sheikh of Brava. Avanchers sent his findings in the form of a lengthy letter to Antoine d'Abbadie and they were published in 1859 in the *Bulletin de la Société de Géographie*.[60] Avanchers' communication was considered very important because the information in it covered the southern unexplored regions of Abyssinia and thus complemented the explorations to the north of d'Abbadie and others. An English translation of his French text concerning the lake reads as follows: "Lake Boo is the same as Lake Abbola noted in the basin of the Bahar-el-Abiad. Only its position is much more to the south, because in the Boran country there is no lake." [60(p163)]

It is possible that Avanchers' informants were confusing several lakes, including Rudolf, Stephanie, and one further to the north, Lake Abaya. Yet on his map accompanying the article, the placement of Lake El Boo, between 35° and 36° east longitude and 1° north latitude, puts it in the general vicinity of Lake Rudolf. In essence the placement is a little too far south and a little too far to the west. But it is close, considering that Avanchers had never visited the region. Thus there is a good possibility that Avanchers' informants were indeed talking about Lake Rudolf. While Avanchers could have had access to the "slug map," his own map bears little resemblance to it. The shape of his Lake Boo is quite different from Erhardt's Lake Zamburu, as is its configuration. Thus it seems likely that Avanchers was communicating information gathered from Africans and was uninfluenced by what European travelers to the south had heard. He also shows Lake Niasu (Nyasa) to the south as a distinct lake, unlike Erhardt who grouped all the central lakes together into one body of water. He places the Rendille just to the south and west of the lake, which is correct, and in his text[60(p164)] says that all of the country to the south of Lake El Boo is inhabited by the Wa-Kuafi (Samburu), which is also correct. Avanchers' informants also told him that a lake was located in Baharingo country (known today as Lake Baringo) and that hot springs existed in the area, as well as an active volcano (probably Mount Teleki at the southern end of Lake Rudolf which was active then). All of these descriptions were accurate. A major piece of misinformation was that Lake El Boo was a source of the Nile and that one could travel from it by boat to Nasser (an Egyptian fort set up in 1854 on the Sobot River in the Sudan). The Sobot flows into the Nile. Avanchers noted that several caravan leaders had drawn maps for him which bore a strong resemblance to those drawn by the protestant missionaries to "celle de M. Mac Queen jointe à l'ouvrage du Capitaine Harris" 60(p159),61,62 Overall, Avanchers had constructed a map containing much accurate information.

As intriguing as it was, the prospect of a large lake lying just below the Ethiopian highlands was insufficient to draw either explorers or the support of governments and institutions like the Royal Geographic Society to equip expeditions to go out after it. By 1860, attention was solidly riveted on solving the mystery of the source of the Nile, which lay well to the west. The Nile and its sources were not merely of geographic interest in Britain and the rest of Europe. As later Victorians saw it, the Nile prominently figured in their Mediterranean political interests, particularly in Egypt, and thus there were strong reasons not only for finding its sources but also for securing them and the river for Britain. This preoccupation overshadowed any possible interest in other unexplored regions of Africa as far as the British were concerned.

In 1860, John Hanning Speke, who had accompanied Richard F. Burton on an earlier expedition to Lake Tanganyika, set out with James Augustus Grant for central Africa in order to definitively solve the issue of the source of the Nile. They traveled to the northern end of the lake called Nyanza which Speke had named Victoria Nyanza on his earlier trip, and on July 28, 1862, Speke discovered the Nile issuing from the lake.[63] On February 13, 1863, they arrived at Gondokoro on the Nile in what is now the Sudan and there met a wealthy Englishman, Samuel Baker, and his wife, Florence, who were on their way up the river. Speke and Grant had been traveling for almost two and a half years, and had in their view solved the question of the Nile's source. They told Baker that there was another large lake to the west called Luta N'zige (dead locust) into which the Nile flowed and that the river then flowed out of it again. They had been unable to confirm this because of the obstacles placed in their path by Kamrasi, the king of Bunyoro, an area in northern Uganda.[63] Baker went on to confirm the suppositions of Speke and Grant, returning to England on October 14, 1865.[64,65(p155)] But Speke encountered much skepticism about his discovery because it conflicted with the preconceptions and ideas of some very influential explorers, including Livingstone, who sincerely believed that the source of the Nile lay further to the south. Fueling the controversy was Richard F. Burton and his supporters. However, this is an issue beyond the scope of this presentation. Suffice it to say that Burton and Speke were to debate one another at Bath, England on September 16, 1864, at a meeting of the British Association for the Advancement of Science, but that Speke accidentally shot himself while out shooting the day before.[66(pp70–77)]

Baker's findings did not satisfy the skeptics either, and subsequent exploratory efforts were undertaken by Stanley, who finally proved that the Lualaba River became the Congo and was not a source of the Nile as Livingstone had thought.[67] Thus the 1860s and 1870s in Britain were consumed with the issue of the Nile and its sources. But it can be said that Stanley's trip down the Lualaba finally settled the issue of the White Nile. The Blue Nile, which rises in the Ethiopian Highlands, was another matter, but by then its source was known.[68] The two rivers join one another at Khartoum, the capital of the Sudan, where the Blue Nile contributes far more water volume than the White Nile, which is ironic since in the 19th century so much attention was focused on the latter and hardly any on the former.

In 1870, Thomas Wakefield, a Methodist missionary working on the East African coast, published an extensive article on the native caravan routes into the interior.[69] Wakefield also published a detailed map with his article showing Lake Samburu lying between 38° and 39° east longitude and 1° and 2° north latitude. Although the lake's position is somewhat off the mark, its position relative to Lake Baringo to the southwest is accurate. With regard to other formations such as Mount Kenya, it is quite misplaced far to the east. Also, Lake Baringo is represented as being excessively large. Wakefield's information was gathered from a Swahili-speaking ivory trader, Sadi ben Ahedi, who claimed that he saw Lake Samburu. "He states that he saw the northern (?southern) end of the Samburu Lake, which appeared two days off, and describes it as being—length, from station at Ribe to Kipumbui (128 miles); breadth, from Ribo to Gasi (37 miles); bearing N.N.E., S.S.W."[69(p322)] Sadi's description is significant because it very accurately gives the actual measurements of the lake (155 miles long and 35 miles wide today). However, it repeats the inaccurate directions of the lake previously published. Whether Sadi actually saw the lake is an open question, but it is quite possible that he did because trading caravans had been going up to it for a very long time.

In 1873, another Methodist missionary, Charles New, a colleague of Wakefield, who also worked on the coast and who is best remembered for being the first to climb Kilimanjaro to the snowline, published his memoirs.[70] Included in these was a detailed map of trading caravan routes into the interior, including Lake Baringo. However, Lake Samburu is not mentioned. Alison Smith states in her introduction to the third edition of New's book that " . . . the information which he and Wakefield laboriously compiled from caravan accounts added up, when combined with the contributions of Krapf and others, to a very fair picture of the main features of the area covered by modern Kenya and the northern end of Tanzania." [70(intro p22)] Smith goes on to say that a fairly accurate map was compiled by a younger geographer, Keith Johnston, and published in the *Journal of the Royal Geographical Society* (LXI, 1874, pp 303–339).[71] Johnston very much wanted to lead an expedition across Masai country. However, the Expedition Committee of the Royal Geographical Society decided against sending him there because they feared for the safety of any expedition entering Masai country.

In 1879, Johnston, who had drawn Wakefield's map showing Lake Samburu, led the society's alternative expedition to Lake Nyasa, where he died of dysentery at Behobeo.[72(p5)] The Masai had by this time gained a reputation in Europe as fierce, blood-thirsty warriors. This was due not only to reports by Europeans who had traveled on the edges of their country but more to later descriptions circulated by coastal Swahili-speaking traders who wanted to protect their newly developed interior trade routes from European penetration.

The Masai were certainly a powerful force on the East

African steppes and plateaus by the middle of the 19th century. But about that time a series of widespread civil wars greatly decimated them in both numbers and power. Refugees from these wars sought haven in places like Taveta or else began raiding coastal peoples in order to survive. The latter action would have sustained belief in the continued power and ferocity of the Masai among Europeans and traders on the coast. Disease took its toll on the Masai as well. A serious cholera epidemic broke out in 1869 and smallpox in 1883–1884, and cattle diseases such as pleuropneumonia decimated their herds. Thus the reports of explorers from the 1860s were clearly dated by the 1880s. It was during the 1880s that Zanzibari-based Arab and Swahili traders began penetrating Masai country with considerable frequency.

By the mid-1870s Lake Samburu regularly appeared on maps produced by other explorers who had traveled through adjacent areas. An example of this is the map that accompanied the 1874 book of the Latvian botanist, Dr Georg Schweinfurth, who had spent three years traveling through the Upper Nile, the Bahr el Ghazal River, and the upper reaches of the Uele River in what is now northeastern Zaire.[73]

As an aside it is of interest that Sadi ben Ahedi, who provided Wakefield with much useful information on caravan routes and who traveled with New on his two expeditions to the Chagga country around Mount Kilimanjaro, was ill thought of by Joseph Thomson the explorer.[72] Thomson believed that Sadi had caused New's second expedition to fail by having been an accomplice to its plunder by Mandara the Chagga chief of Moshi.[72(p70)] New died of dysentery while returning to his mission station from this expedition in February 1875. Mandara told Thomson that Sadi had poisoned him, but Thomson doubted this because of the clear lack of a motive.[72(p4)] Mandara himself was not without a motive and confessed as much to Thomson. New suffered from severe dysentery before he died and whether or not his death was due to poisoning will never be known. Thomson was also of the opinion that Sadi, who was a guide for Baron von der Decken's expedition across Masailand, caused it to fail.[72(p4),74–76] Thomson knew none of this until he himself had hired Sadi as a guide for his famous trip across Masailand to Lake Victoria. During the course of that journey he found Sadi to be untrustworthy. But Sadi had his good points as well and Thomson says of him that he had "... a commanding and venerable presence." Thomson goes on to say that Sadi "... without exception had the most thorough knowledge of the Masai language of any man on the coast." [72(p70)] Sadi was quite a talker as well, a real attribute in that part of Africa. "I never knew anyone with such singular 'gift of gab'!" Thomson says.[72(p70)] Still Thomson thought that some of his difficulties were due to Sadi's treachery, although he musters no evidence to prove this nor any of the other serious allegations he makes against him.[72(p127)] This is relevant to the present discussion because it involves the integrity of the individual who provided Wakefield with precise information about Lake Samburu and who claimed to have seen it.[69] Even if Thomson's allegations against Sadi were true, one must remember that in that time and place what was perceived as treachery and duplicity by Europeans was often seen by the Africans as good common sense and protection of self-interest.

The fact that Sadi behaved treacherously as seen from a European viewpoint did not diminish his linguistic skills, his vast knowledge of the interior which he had visited on many occasions, and his skills as a leader and diplomat, abilities which when mustered worked wonders, as Thomson attests.[72(p70)]

A HASTY YOUNG SCOTSMAN

Coastal Swahili and Arab traders who dealt in both slaves and ivory had been extremely effective in keeping Europeans out of the vast, ivory-rich interior that lay beyond Mount Kilimanjaro, primarily by scaring them off with tall tales about the alleged ferocity of the Masai. By the late 1870s considerable interest in learning about this large unexplored tract of land between the mountain and Lake Victoria had grown, particularly in Britain. In 1881, the British Association for the Advancement of Science resolved at its annual meeting that the "snowy range of Eastern Equitorial Africa" should be explored as soon as possible.[77(p133)] The association agreed to contribute financially to the expedition if the Royal Geographical Society would sponsor it. The Expedition Committee of the Royal Geographical Society recommended that the society sponsor the trip at a meeting in June 1882.[77(p136)] The society found the ideal person to undertake the trip in the person of Joseph Thomson, a 24-year-old Scotsman who had already made two successful journeys into the interior of Africa, one to Lake Nyasa with Keith Johnston in 1879–1880, and one up the Rovuma River in 1881.[77] At the time there was in Britain no governmental concern about the threat of competitive German expeditions penetrating this area. However, influential men in Britain, with an interest in the future political and commercial value of this piece of Africa, were indeed concerned. For scientific exploration set the ground rules for later political negotiations over spheres of influence. If the Germans were successful in getting across Masailand, they would be in a strong position later on to annex the territory in the scramble for Africa that was shortly to follow. In fact when Thomson arrived in Zanzibar, he learned that Dr Gustav A. Fischer, a German naturalist, had already left the coast at the lead of a caravan headed across Masailand. Had it not been for internal organizational problems of Fischer's expedition, he would have beaten Thomson across the Masai steppes.[78] But at the time Thomson had no way of knowing that Fischer would not succeed.[72(p8)] The society had prepared Thomson as best it could with financial support and with technical instruction in map making from John Coles, the cartographer, who would later instruct Arthur Donaldson Smith. And enroute to East Africa he had the good fortune to meet Dr Georg Schweinfurth in Cairo, famous for his journey to the upper Nile. Schweinfurth, in effect, increased Thomson's anxieties about being beaten by the Germans because he told him of another German expedition under von Wissmann that had just gone through the Congo and East Africa.[77(p41)] The British may have been sleeping with regard to East Africa but the Germans certainly were not.

While on the coast, Thomson met with Thomas Wakefield, the Methodist missionary, who had carefully record-

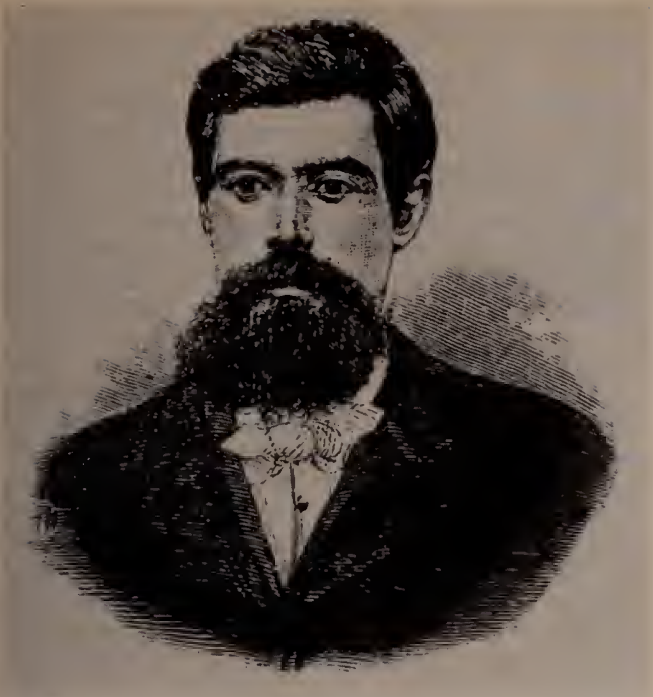


FIGURE 7. James Martin (Antonio Martini), who accompanied Joseph Thomson across Masailand (reproduced from *Through Masai Land*, London, Sampson, Low, Marston, Searle and Livington, 1887).

ed and published an extensive account of the caravan routes into the interior.[69,79(p184)] Thomson was also lucky in that in Zanzibar he met James Martin (Fig 7), a Maltese sail maker whose real name was Antonio Martini. Martin had landed in Mombasa some years before and had worked for the Church Missionary Society. Unfortunately, he was illiterate, and thus the journeys he made with Thomson as well as with others in subsequent years went unrecorded. He was clearly a man of exceptional abilities and according to some contemporary observers made Thomson's journey a success.[80(p68),81,82]

Thomson's expedition headed for the interior on March 6, 1883. His line of march took him to the mission station at Rabai where Krapf had served and he then headed for Taveta, a caravan staging point to the southeast of Mount Kilimanjaro. It was in Taveta that he hired Sadi ben Ahedi, the famous ivory trader. From there he went along the southern flank of the mountain into the country of the Chagga where he met Mandara, the Chagga chief of Moshi, whose importance and power had been artificially inflated through his cunning manipulation of previous European travelers. On leaving Mandara he proceeded west but after a short distance he learned that Fischer's caravan had had a bloody encounter with a group of Masai and that the latter were literally on the warpath. Thomson decided to retreat to Taveta, where he arrived on May 12, 1883.[72(p100)] He realized that his best chance of getting across Masailand was by joining forces with a large trading caravan. Meanwhile, he went down to Mombasa for additional supplies and reentered Taveta on July 2, 1883.[72(p111)] A stroke of luck in the form of a large trading caravan under Jumbe (Chief) Kimemeta (Fig 8) from Pangani heading for Masailand changed his fortunes.[72(p126),83] Kimemeta was an experienced ivory trader and traveler who, had he been literate in a European language, would have gone down in history as the "discoverer" of many East African landmarks, including Lake Rudolf, for which Europeans later received credit. But Kimemeta's interest was principally ivory and slaves. With the greater protection afforded by Kimemeta's caravan plus his knowledge of the country and its people, Thomson once again set off, around the northern flank of Kilimanjaro and on up to Ngongo Bagas, near modern-day Nairobi. Thomson found the Masai to be arrogant and intrusive, but he managed to deal with them diplomatically and by passing himself off as a *laibon* (medicine man) by popping out his false teeth and putting effervescent salts in water. The two caravans moved on north through the Rift Valley to Lake Naivasha and on up along the Nyandarua Mountains, which Thomson called the Aberdares in honor of the president of the Royal Geographical Society. He paid a hasty visit to the slopes of Mount Kenya but did not attempt to climb it as he had Kilimanjaro.[72(p222)] Then he rejoined the main part of the caravan that had gone on to Lake Baringo. From here Kimemeta went on north to trade for ivory among the Turkana while Thomson headed for Lake Baringo, and then set off in a rush to Lake Victoria, a relatively short distance away, which he reached on December 11, 1883.[72(pp292-296)] He then went up to visit the 14,178-foot-high mountain, Mount Elgon, whose name is Masai. And from there he rushed back to Baringo.

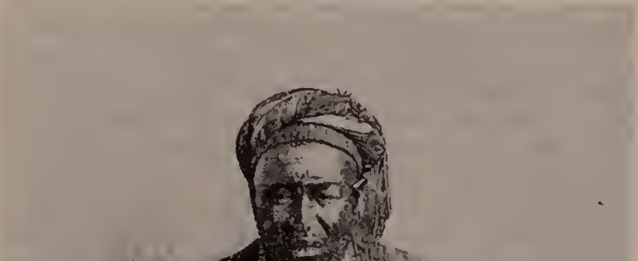

JUMBE KIMEMETA.

FIGURE 8. Jumbe Kimemeta, the famous ivory and slave trader who accompanied both Joseph Thomson and Count Samuel Teleki on their expeditions (reproduced from *Discovery of Lakes Rudolf and Stefanie*, London, Longmans, Green and Co, 1894).

For all intents and purposes, Thomson had achieved his objectives at this point. He had visited the snowy ranges of East Africa, had crossed Masailand to Lake Victoria, and had even attempted to climb Kilimanjaro. Yet had it not been for Martin at this point Thomson might have died, for shortly after reaching Baringo and until he regained the coast, he suffered from dysentery and was virtually incapacitated. He left Nyemps on February 22, 1884, and for most of the way to Ngongo Bagas was accompanied by Jumbe Kimemeta. There Thomson parted company and went down the coast via a parallel route through Kamba country, reaching the mission station at Ndara near the coast on May 21, 1884, and Rabai three days later.[72(pp339-340)] He had traveled over 3,000 miles in 14 months.

In some ways the real heroes of the Thomson journey are James Martin, who virtually took charge of the expedition when Thomson was ill, and Jumbe Kimemeta the trader, who had crossed most of this country before. Thomson was given a hero's welcome in Britain and his book, *Through Masai Land*, first published in 1885, was a sensation. Of note is that Jumbe Kimemeta's name does not appear in the index, although Thomson does give him credit for his invaluable assistance to his mission.[72(p338)] But over the years, scholars have come to realize that although Thomson accomplished a remarkable feat that had enormous implications for Britain's later colonial ambitions, he was hasty and careless in his observations, did things on the run, and lacked the ability to analyze and approach systematically the objects of his observation. In part this may have been due to his youth and to his fear that the German explorer, Fischer, would beat him to the prize. But these are characteristics that are evident in the accounts of his other journeys as well. To his credit, Thomson finally gave a precise idea of the size of Lake Baringo which had been greatly exaggerated in reports from traders and, relevant to this discussion, had some interesting things to say about Lake Samburu. He wrote:

> . . . This lake they described as from twenty to thirty miles broad; but its length they knew not, as they had never seen the northern end. They further spoke of it as lying between mountains several thousands of feet high, though the water does not quite reach their base, and there Wakwafi dwell. They say that the water is salt, and has surprising numbers of enormous *white* fish, with crocodiles and hippopotami. The natives have no canoes.[72(p314)]

On the map accompanying his book, Thomson places Lake Samburu at 37° east longitude with its southern end at about 3° north latitude. This represents an accurate placement of the lake today, but the true configuration is different from the one Thomson gave, which was essentially that which had been published by Wakefield and some others. His descriptions of Nile perch and the large number of crocodiles and hippopotami in the lake were all accurate. Thomson obtained this information while he was in Baringo from Wakwafi people who had come down from the area of the lake. These were probably Samburu or Masai people who would have known the environs of the lake quite well.

Thomson himself never returned to the high plateaus of East Africa but did undertake several subsequent expeditions to Nigeria (1885), the Atlas Mountains (1888), and Katanga (1890). He died in London on August 2, 1895, at the age of 37,[77(p296)] probably from systemic tuberculosis. In 1885, Thomson's book was published in German[84] and the following year in French. In Austria-Hungary it came to the attention of Crown Prince Rudolf, who had a keen interest in geographic exploration.[85] There on Thomson's map was a large lake that no European had ever seen, the last of the great African lakes left to be explored, an opportunity for Austria-Hungary to gain a place in African exploration and perhaps a stake in the colonial scramble for Africa as well.[86]

SPHERES OF INFLUENCE

Thomson's triumphal return in 1884 was one of the few bright spots for Britain in Africa that year, which was from the German perspective characterized by rapid and successful colonial expansion. The Germans annexed the Cameroons in July,[87(p366)] Southwest Africa in September,[87(p366)] and in October sent Gerhard Rohlfs, a noted explorer and outspoken advocate for German colonial expansion, as consul-general in Zanzibar. Zanzibar's independence had been assured in 1862 by a declaration signed by Britain and France, but the British exercised significant influence over the sultan and his policies. Zanzibar claimed sovereignty to much of the East African hinterland. Thus German attempts to seize any territory there brought the British into direct conflict with the Germans through the former's surrogate, namely Zanzibar. The British government of the day was indifferent to the acquisition of colonies in Africa in general. However, there were influential private men such as Sir William Mackinnon, who in 1888 obtained a charter for the Imperial British East Africa Company, and who were alarmed by Germany's successes. A similar German company, known as The Gesellschaft fur Deutsche Kolonisation (Society for German Colonization) had been founded by Dr Carl Peters. In 1884 Peters landed on the East African coast and, eluding agents of the sultan, successfully signed treaties with local chiefs in Usagara, just opposite Zanzibar on the mainland.[87(p369)] Peters was back in Berlin in February 1885 where at the time the Berlin Conference on the Niger and the Congo was coming to an end. This conference had been called in November 1884 to settle issues related to those areas and that affected the European powers concerned.[87(p366)]

The day after the delegates to the conference left Berlin, the kaiser placed under German protection the areas where Peters had signed treaties. Peters' success in bringing this about was not due so much to his political skills but rather to the fact that his proposals nicely fit into the scheme of German-British hostility in Europe. This had far-reaching effects because it successfully defied the Sultan of Zanzibar's rights and meant that Britain could no longer control German actions by using the sultan as a surrogate for its own interests.[88(p106)] In April 1885, Clemens Denhardt, who had been trading along the Tana River area since the 1870s with his older brother, Gustav, signed a treaty with the chief of Witu, an area located on the coast midway up the present-day Kenya coast. This action coupled to the Usagara protectorate effectively placed the British sphere of influence between two German ones, with Witu in the north and Usagara in the south. The Germans were in an ideal position to lay claim to the interior, circle the British sphere, and render it nothing more than a protruding coastal enclave.

By August the Germans had forced the sultan to recognize their claims and threatened him with the use of force if he did not.[88(p108)] Two months previously, in June, realizing their precarious hold in East Africa, the British requested that a commission with representatives from Britain, France, and Germany be set up to define the limits of the sultan's territories on the mainland. The work of this commission laid out the general issues but was unable to come to an agreement. Bismark was anxious to settle the Zanzibar issue and promised the British that if they made concessions on the East African question he would support them in their dispute with the French over Egypt.[88(p109)] The British had occupied Egypt in 1882.[89(p4)] The East African partition treaty that resulted in late 1886 recognized Witu as German and defined the German sphere of influence as extending inland to Lake Victoria between Rouvuma River and a line drawn to the lake from the coast at Umba.[87(p374)] The British sphere was north of this line, extending to the Tana River.[90(p10)] This line, which essentially constitutes the modern boundary between Kenya and Tanzania, effectively divided the two spheres of influence but did not define their western limits. And relevant to this discussion, the treaty set the following northern limits to the British sphere of influence:

> . . . and on the north by a line which, starting from the mouth of the Tana River follows the course of that River or its affluents to the point of intersection of the Equator and the 38th degree of east longitude, thence strikes direct to the point of intersection of the 1st degree of north latitude with the 37th degree of east longitude where the line terminates.[90(p10)]

What this meant was that the vast area of what is now northern Kenya and where Lake Rudolf is located was as yet unclaimed. With a German protectorate at Witu, on the coast, directly across from this area, there was little doubt among many Britons familiar with the issues that the Germans would sooner or later make a grab for it. Thus, as the Austro-Hungarian Count Samuel Teleki made preparations for his journey to discover Lake Samburu, in 1886, the area around the lake was still outside of anyone's sphere of influence. This may have provided Crown Prince Rudolf with some of the impetus for urging Teleki to undertake the journey.[86] In East Africa and Europe, however, a rapid series of complex events involving both the European powers and their agents and surrogates in Africa unfolded which effectively dimmed whatever vague colonial dreams Rudolf may have had.

In 1887, while Teleki was enroute to the lake, the Sultan of Zanzibar granted a land concession to his coast areas (the 1886 agreement granted the sultan full rights to the coastal strip, even that along German territory) to the British East Africa Association (later to be known as the Imperial British East Africa Company).[87(p378)] The association had been founded by a Scottish businessman, Sir William Mackinnon, who had had extensive business dealings with Zanzibar since the 1870s. He and the company were destined to play a very significant role in the development of what eventually became modern Kenya and Uganda. Both German and British agents in East Africa and the swirl of European big power politics led the British and the Germans to the bargaining table to settle the East African question in 1890. Their discussions led to the Heligoland Treaty of 1890 in which Germany ceded all interest in Witu and in the lands north of the British sphere of influence and accepted a British protectorate over Zanzibar. The British in turn promised to persuade the sultan to sell Germany his coastal territories adjacent to their sphere of influence and to cede the island of Heligoland, which at the time the Germans viewed as crucial to the development of their naval power.[87(pp383-384)] The Heligoland Treaty was signed on July 1, 1890, and shortly thereafter Carl Peters returned from a marathon trip through what was now the British sphere, having started out from Witu in 1889. Peters went up the Tana River from Witu and then across to Uganda, signing treaties along the way on behalf of Germany. When he arrived on the coast at Bagamoyo and learned that Bismark had signed away what he thought he had just acquired for Germany, he was so upset that he had to withdraw to a room to be alone for two hours.[88(p114),91] Bismark's willingness to sign the Heligoland Treaty had little to do with East Africa. He was fearful for Germany's position in a Europe in which France and Russia had grown uncomfortably close, and wanted to move closer to Britain as part of a strategy to offset any possible attack by France and Russia.[88(p114)]

In 1886, as Crown Prince Rudolf contemplated Teleki's trip, the Heligoland Treaty was four years off. The lands around Lake Rudolf were wide open for acquisition, a status that would be confirmed by the 1886 agreements between Britain and Germany.[90(p10)] As Teleki prepared to set out from Vienna, Arthur Donaldson Smith was completing his year of study in graduate chemistry at the Johns Hopkins University and getting ready to enter Harvard Medical School.[12] It is doubtful that at the time this would-be physician thought he would one day play a major role in this turbulent corner of Africa, or that his name would forever be associated with a lake that had yet to be discovered.

REFERENCES

1. Smith AD: *Through Unknown African Countries: The First Expedition from Somaliland to Lake Lamu*. London and New York, Edward Arnold, 1897, reprinted, New York, Greenwood Publishers, 1969.

2. Von Höhnel L: *Discovery of Lakes Rudolf and Stefanie: A Narrative of Count Samuel Teleki's Exploring and Hunting Expedition in Eastern Equitorial Africa in 1887 and 1888*, Bell N (D'Anvers) (trans). London, Longmans, Green and Co, 1894, vols 1, 2.

3. Registry of Baptism, All Saints Episcopal Church, Philadelphia, Penn, September 10, 1866.

4. Certificate of Death, Commonwealth of Pennsylvania, Department of Health, Bureau of Vital Statistics, Arthur Donaldson Smith, File No. 13166, filed February 21, 1939, obtained July 20, 1978.

5. *The National Cyclopedia of American Biography* (1906; 13:608) gives his correct date of birth, April 27, 1866, but incorrectly states that he was born in Philadelphia. *Who Was Who in America* (1943; 1:1137) incorrectly states that he was born on April 27, 1864, but correctly lists Andalusia as his birthplace. The *University of Pennsylvania Alumni Catalogue*, published early in this century, as well as the university's publication, *Matriculates of the College*, published in 1894, gives his place of birth as "Wyndlawn" (a family country home), Byberry, Philadelphia, Pennsylvania, and April 27, 1866, as his birthdate. His obituary notice in the *Philadelphia Inquirer*, "Dr. A.D. Smith dies; Famous Explorer, February 21, 1939," correctly states that he was born in Andalusia, but incorrectly states 1864 as the year of his birth. The earlier 1864 birthdate also appears on Smith's Harvard Medical School registration, 1886-1888. Smith himself gave an 1865 birthdate and Andalusia as his birthplace, on his September 30, 1885, application to Johns Hopkins University. This 1865 birthdate also appears under Smith's entry in the *Yearbook of the Explorers Club*, 1906, p 53.

None of the numerous obituary notices that appeared in newspapers correctly stated Smith's age, which was 72 years. Although he died three months before his

73rd birthday, most papers, including the *Philadelphia Record*, February 20, 1939, the *Philadelphia Evening Ledger*, February 21, 1939, *The New York Times*, February 21, 1939, the *Philadelphia Inquirer*, February 21, 1939, and the *St Petersburg Times*, February 22, 1939, incorrectly gave his age as 74 years. The *Philadelphia Evening Bulletin*, February 20, 1939, gave it as 73 years.

6. Statewide vital statistics in Pennsylvania are available from 1906 onwards. Vital statistics for Bucks County exist in the County Clerk's Office, Doylestown, for the period 1893–1905. In Philadelphia, the Vital Statistics Act took effect July 1, 1860. Birth and death registrations for Philadelphia County for the period July 1, 1860–June 30, 1915, are maintained in the City Archives and by the Vital Statistics Section of the Department of Health, New Castle, Penn, from July 1, 1915, onwards. Baptismal records are the only records of birth for Philadelphia prior to July 1, 1860, and for Bucks County prior to 1893 (personal communication, Philadelphia City Archives and County Clerk's Office, Doylestown, Bucks County, 1986).

7. Personal communication, H. Spencer Potter, PhD, Manhasset, NY, July 7, 1986.

8. *Social Register*, New York Social Register Association, August 1894, p 252.

9. Affidavit in support of application for membership and application for membership of Arthur Donaldson Smith in the Pennsylvania Society of Sons of the Revolution, May 1 and May 2, 1890, Archives of the General Society Sons of the Revolution, Ruxton, Md.

10. On Smith's death certificate his mother's name is incorrectly given as Margaret.

11. In 1902 Andalusia had a population of only 176. *Atlas of the World*, Chicago, New York, London, Berlin, Rand McNally and Co, 1901, p 21.

12. Student and alumni records of Arthur Donaldson Smith. University of Pennsylvania Archives, Philadelphia, Penn.

13. *University of Pennsylvania Catalogue and Announcement 1885–86*. Philadelphia, printed for the university, 1885 (University of Pennsylvania Archives).

14. *University of Pennsylvania Catalogue and Announcements 1889–90*, Philadelphia, printed for the university, 1890 (University of Pennsylvania Archives).

15. Application for admission, graduate chemistry, Arthur Donaldson Smith, September 30, 1885. The Ferdinand Hamburger, Jr, Archives, The Johns Hopkins University, Baltimore, Md.

16. These courses are listed for students enrolled in graduate chemistry in the *Johns Hopkins University Circular*, 1885–1886. Personal communication, James Stimpert, Assistant Archivist, The Ferdinand Hamburger, Jr, Archives, The Johns Hopkins University, Baltimore, Md, June 25, 1986.

17. Ludmerer KM: *Learning to Heal. The Development of American Medical Education*. New York, Basic Books, 1985.

18. Arthur Donaldson Smith, Registration Record for Harvard Medical School, 1886–1888. University Archives, Harvard University Library, Cambridge, Mass.

19. Ludmerer KM: Reform at Harvard Medical School, 1869–1900. *Bull Hist Med* 1981; 55:343–370.

20. Corner GW: *Two Centuries of Medicine: A History of the School of Medicine, University of Pennsylvania*, Philadelphia and Montreal, JB Lippincott Co, 1965.

21. *Annual Report of the Provost and Treasurer of the University of Pennsylvania*, Philadelphia, 1887.

22. Younger American physicians with postgraduate training in Europe often came into sharp conflict with their older colleagues who had received similar training in France.

23. Bonner JN: *American Doctors and German Universities: A Chapter in International Intellectual Relations 1870–1914*, Lincoln, University of Nebraska Press, 1963.

24. One of my uncles, P.J. Imperato, MD, a 1921 graduate of Cornell University Medical College, went to study at several German clinics in 1925. In his unpublished reminiscences he wrote: "I thoroughly enjoyed my studies at several clinics. Much as I had grumbled about having to learn German before it now turned out to be very helpful."

25. A.D. Smith Buried: African Explorer. *Philadelphia Record*, February 20, 1939.

26. Dr. A. D. Smith, 74; African Explorer. *Philadelphia Evening Ledger*, February 21, 1939.

27. Dr. Arthur D. Smith, Explorer of Africa. *New York Times*, February 21, 1939.

28. Dr. A. D. Smith Dies. Famous Explorer. *Philadelphia Inquirer*, February 21, 1939.

29. H. Spencer Potter clearly recalls his father, Charles A. Potter, Jr, saying that he had written the basic obituary notice for Smith. The essential information in the obituaries is no doubt highly accurate given Potter's close relationship with Smith and the fact that the latter lived with Potter and his wife, Margaret, for some time before his death. Personal communication, H. Spencer Potter, PhD, March 28, 1986.

30. The archives of the College of Physicians of Philadelphia and the American Medical Association do not contain any information on Smith for this period. The Pennsylvania Medical Society did not publish an annual directory of its members until 1920. There are no entries in it for Smith for the 1920s.

31. Imperato PJ, Imperato EM: Up north in Kenya. *Explorers J* 1979; 57:22–29.

32. Imperato PJ: Gateway to Kenya's north. *Off Hours*. 1983; 1:5–8.

33. Death certificate, Jesse Smith, No. 089222, City of Philadelphia, Department of Records, City Archives, November 24, 1892. Smith's age is given as 68 years, his cause of death as apoplexy, his physician's name as Dr Hatton, and his place of burial as Laurel Hill Cemetery.

34. Death Notice, Jesse Evans Smith, *Philadelphia Inquirer*, November 27, 1892. The notice reads in part, "Jesse Evans Smith in the 69th year of his age," meaning he was 68 years old.

35. Estate of Jesse E. Smith. Letters of Administration, granted December 1, A.D. 1892, No. 1505. Register of Wills and ex-officio Clerk of the Orphans' Court for the City and County of Philadelphia, in the Commonwealth of Pennsylvania.

36. William Lord Smith, MD, is listed at various addresses in Boston, Mass, in the first through fifth editions of the *American Medical Directory* (Chicago, American Medical Association). His address in the seventh edition, 1921, was Brockton, Mass, and in the eighth edition, 1923, he was listed as not being in practice. In the 11th edition, 1929, his address was listed as unknown.

37. *Social Register*, New York, Social Register Association, 1893, p 231. The *Morton Allan Directory of European Passengers Steamship Arrivals* (Baltimore, Genealogical Publishing Co, 1980, p 32), lists June 26, 1893, as the arrival date for the *S.S. Tauric*.

38. Smith AD: Expedition through Somaliland to Lake Rudolf, Part 1. *Geographical J* 1896; 7:120–137.

39. Graves P: *The Life of Sir Percy Cox*. London, Hutchinson and Co, 1941.

40. Trzebinski E: *The Kenya Pioneers*. New York, WW Norton Co, 1986.

41. Cox PZ, in Wickham Legg LG (ed): *The Dictionary of National Biography, 1931–1940*. London, Oxford University Press, 1949, pp 196–199.

42. *British and Foreign State Papers*, 1885–1886, London, William Ridgway, vol 77, pp 1263–1269.

43. *British and Foreign State Papers*, 1888–1889, London, Her Majesty's Stationery Office, vol 81, p 936.

44. French Somaliland was established in 1885 in the area around Obock and Djibouti and by 1889 Italy had firmly established control over an enormous area of the Horn of Africa from the Gulf of Aden to Kismayu on the Indian Ocean. This territory later became known as Italian Somaliland. Modern Somalia, which became independent on July 1, 1960, consists of the former British and Italian territories. French Somaliland, whose name was later changed to the French Territory of the Afars and Issas, became independent as Djibouti in 1977. See Touval S: *Somali Nationalism: International Politics and the Drive for Unity in the Horn of Africa*, Cambridge, Mass, Harvard University Press, 1963, and Hess RL: *Italian Colonialism in Somalia*, Chicago, University of Chicago Press, 1966.

45. Zaghi C: *I Russi in Etiopia. Il Protettorato Italiano sull'Etiopia*, vol 1. *Menelik e la battaglia di Adua*, vol 2. Napoli, Guida Editori, 1972.

46. Cufino L: *Riccordi della seconda spedizione Ruspoli, I diario del dott. Carlo Riva. Bull Soc Geog Ital* 1912; 49:33–47.

47. Bottego V: *Viaggi di Scoperta nel Cuore dell'Africa. Il Giuba Esplorato, Sotto gli Auspici della Societa Geografica Italiana*. Roma, Luescher & Co, 1895.

48. The lake's name was officially changed to Lake Turkana by the Kenya government in the 1970s. However, the name Rudolf will be retained in this text as this was its name during most of the period under discussion.

49. Hance WA: *The Geography of Modern Africa*. New York, Columbia University Press, 1964, p 395.

50. Butzer, KW: *Recent History of an Ethiopian Delta: The Omo River and the Level of Lake Rudolf*. Chicago, University of Chicago Department of Geography Research Paper No. 136, 1971.

51. Leakey RE, Lewin R: *People of the Lake: Mankind and Its Beginnings*. Garden City, NY, Anchor Press/Doubleday, 1978.

52. Cerulli E: *Peoples of South-West Ethiopia and Its Borderlands*. London, International African Institute, 1956.

53. Beckingham CF, Huntingford GWB: *Some Records of Ethiopia 1593–1646*. London, Hakluyt Society, 1954.

54. D'Abbadie A: *Douze Ans de Sejour dans la haute Ethiopie (Abyssinie)*, 1868, reprint ed. Citta Vaticana, Biblioteca Apostolica Vaticana, 1980.

55. Cecchi A: *Da Zeila alle Frontiere del Kaffa*, Roma, Societa Geographica Italiana, 1886–1887.

56. Krapf JL: *Travels, Researches, and Missionary Labours During an Eighteen Years' Residence in Africa*, 1860, reprint ed. London, Frank Cass and Co, 1968.

57. *Athenaeum*, 1855; 1457:1116.

58. Erhardt J: On an inland sea in Africa. *Proceedings of the Royal Geographical Society*, 1856; 1:8–13.

59. Burton RF: *The Lake Regions of Central Africa*. New York, Harper and Brothers, 1860.

60. Des Avanchers L: Esquisse Géographique des pays Oromo au Galla, des pays Somali, et de la Côte Orientale d'Afrique. *Bulletin de la Société de Géographie*, series 4, 1859; 17:153–170.

61. Avanchers is probably referring here to Krapf, Rebmann, and Erhardt.

62. James MacQueen was a noted armchair geographer in England who never actually traveled in Africa. He was partisan to Richard Burton in his dispute with John Hanning Speke over the true source of the Nile and mercilessly attacked Speke in the press in 1863. Speke had just published the results of his expedition during which he showed that Lake Victoria was the Nile's chief source. MacQueen had made his reputation by correctly deducing the course of the Niger River. He believed, unlike Speke, that the Nile issued from two major East African lakes. Even though he had never been to Africa, his opinion carried enormous weight. A former plantation manager in the West Indies, he was 85 years old at the time of the Speke-Burton controversy.

63. Speke JH: *Journal of the Discovery of the Source of the Nile*. London, Blackwood, 1863.

64. Baker S: *Albert N'yanza, Great Basin of the Nile and Explorations of the Nile Sources*, 2 vols. London, Macmillan, 1866.

65. Hall R: *Lovers on the Nile: The Incredible African Journeys of Sam and Florence Baker*. New York, Random House, 1980.

66. Moorehead A: *The White Nile*. New York, Harper and Row, 1960.

67. Stanley HM: *Through the Dark Continent*, 2 vols. London, Sampson and Low, 1878.

68. Moorehead A: *The Blue Nile*. New York, Harper and Row, 1962.

69. Wakefield T: Routes of native caravans from the coast of the interior of Eastern Africa, chiefly given by Sadi ben Ahedi, a native of a district near Gazi, in Udego, a little north of Zanzibar. *The Journal of the Royal Geographical Society*,

1870; XL:303–339.

70. New C: *Life, Wanderings and Labours in Eastern Africa: With an Account of the First Successful Ascent of the Equatorial Snow Mountain, Kilima Njaro. And Remarks Upon East African Slavery*, ed 3. London, Frank Cass, 1971.

71. The 1874 volume of the *Journal of the Royal Geographical Society* is actually volume XLIV and pages 303–339 fall into the index for this volume. Volume XLI (1871) does not contain any information relating to the subject. Smith may be referring to volume XL, 303–339, 1870, covering Wakefield's article and map. Johnston drew the map for the article based on information supplied by Wakefield. This is most probably the map that Smith is referring to.

72. Thomson J: *Through Masai Land: A Journey of Exploration Among the Snowclad Volcanic Mountains and Strange Tribes of Eastern Equitorial Africa.* London, Sampson, Low, Marston, Searle and Livington, 1887.

73. Schweinfurth G: *The Heart of Africa: Three Years' Travels and Adventures in the Unexplored Regions of Central Africa from 1868–1871*, Frewer EE (trans), 2 vols. New York, Harper and Brothers, 1874.

74. Baron Carl Claus von der Decken was a Hanoverian naturalist and traveler who made a quick trip to Mt Kilimanjaro in 1861 and climbed to 5,200 feet with Richard Thornton, a British geologist. In 1862 von der Decken and Otto Kersten, a young German scientist, climbed up to 14,200 feet of Kilimanjaro's 19,340 feet. Their plans to continue on across Masai country were foiled by Sadi ben Ahedi, their guide, according to Thomson. Von der Decken was killed in Somaliland in 1865. It was von der Decken who suggested to New that he attempt to scale Kilimanjaro.

75. Fosbrooke HA: Richard Thornton in East Africa. *Tanganyika Notes and Records*, 1962; 58/59:43–63.

76. Kersten O: *Baron K.K. Von der Decken's Reisen in Ostafrika*, 6 vols. Leipzig, 1869–1879.

77. Rotberg RI: *Joseph Thomson and the Exploration of Africa.* London, Chatto and Windus, 1971.

78. Fischer GA: *Das Masai-land.* Hamburg, 1885.

79. Wakefield ES: *Thomas Wakefield: Missionary and Geographical Pioneer in East Equitorial Africa.* London, The Religious Tract Society, 1904.

80. Jackson F: *Early Days in East Africa.* London, Edward Arnold & Co, 1930.

81. According to Jackson, Martin was born in Malta in 1857, and thus was a year older than Thomson. He sailed on an American ship from India to Zanzibar and then was sent to Freretown on Zanzibar, where the Church Missionary Society had its headquarters. This was done for his own safety as the ship had run aground and virtually all of the crew except Martin had been drunk. His testimony in the Naval Court put him into jeopardy with the rest of the crew. After his trip with Thomson, Martin served under General (later Sir) Lloyd William Mathews, as second in command of the Zanzibari army. He later accompanied a number of expeditions of the Imperial British East Africa Association (later Company), and in 1890 climbed Mt Elgon with Frederick Jackson, Ernest Gedge, and Dr Archibald D. Mackinnon. He became a district officer at Ravine station for the Uganda Protectorate, but was caught poaching ivory. He was then sent to Entebbe as a collector and after leaving the services of the Uganda Protectorate managed a rubber estate in Uganda. With the outbreak of World War I he served with the intelligence section of the British Army and also with the Belgians. He later lived in Dar es Salaam, the capital of modern Tanzania, and died in Portugal in 1924.

82. Walker R: "Little Martin," *Kenya Weekly News*, September 20, 1957; 24–25.

83. Jackson characterized Kimemeta as the "greatest and the best known of the caravan leaders whose spheres of operations were generally referred to as Masailand, but really extended from Kilimanjaro as far as Lake Rudolf . . . " His caravans were always large and well armed and he had a remarkable knowledge of local languages. He had had smallpox earlier in his life that had left his face badly scarred and was blind in one eye. Jackson thought that without Jumbe, Thomson could not have done what he did. Thomson, he maintains, succeeded because of Jumbe's backing. Jackson met him many years later at Ravine station when the old man was leading what proved to be his last safari. He was in ill health and almost blind. Jackson helped make his last safari a success by giving him material assistance. Jumbe died a year later at Pangani on the coast.

84. Thomson J: *Durch Masai-Land Forschungsreise in Ostafrika zu den Schneebergen u. wilden Stämmen zwischen dem Kilima-Ndjaro u. Victoria-Njansa un den 1883–1884.* Brockgaus, Leipzig, 1885.

85. Erdélyi L: *Teleki Samu Afrikaban: Az Afrika-Hutato Eredeti Fényképfélvetéleivel.* Bucharest, Editura Kriterion, 1977.

86. Kádár L: The man whose name the African Teleki Volcano bears. *Communications of the Geographical Institute of the University of Debrecen*, 1966–1967; 181–184.

87. Oliver R, Mathew G: *History of East Africa*, vol 1. Oxford, England, Oxford University Press, 1963.

88. Marsh Z, Kingsnorth GW: *An Introduction to the History of East Africa.* Cambridge, England, Cambridge University Press, 1963.

89. Collins RD: *Land Beyond the Rivers.* New Haven, Conn, Yale University Press, 1971.

90. Hill MF: *Permanent Way: The Story of the Kenya and Uganda Railway.* Nairobi, Kenya, East African Railways and Harbours, 1961.

91. Peters C: *New Light on Darkest Africa; Being a Narrative of the German Emin Pasha Expedition.* London, Ward, Lock & Co, 1891.

The exercise prescription

RICHARD B. BIRRER, MD, JOHN A. DALLER, MS

Within the last decade there has been a growing interest in physical activity and fitness by the consumer and by the medical profession. Long neglected as a therapeutic modality, exercise is now readily incorporated as a preventive measure and as a management strategy in a variety of illnesses. As with medication, the amount, frequency, duration, and mode of exercise can be prescribed and monitored. Although the long-term effects of regular fitness training on length of life are unknown, there is good evidence to support improvement in the quality of life.[1]

PHYSIOLOGY

There are two basic types of exercise: aerobic (dynamic or endurance) and anaerobic (static). At the biochemical level, the consumption of oxygen in the utilization of glucose during aerobic activities produces 18 times more adenosine triphosphate (ATP) molecules than during similar anaerobic or non-oxygen-utilizing metabolism. At the organ level, aerobic activities are translated into cardiopulmonary efficiency. The heart rate increases, but the major enhancement of cardiac output is due to an increase in stroke volume. Although the systolic blood pressure increases, the diastolic component and the mean arterial pressure remain the same or decrease. Peripheral vascular resistance also declines. With regular amounts of aerobic activities over prolonged periods of time, further metabolic changes occur. These include an enhancement of the fibrinolytic process, improvement of glucose metabolism, reduced levels of norepinephrine, epinephrine, cholesterol, and triglycerides with concomitant increases in the high density lipoprotein (HDL) fraction, improvement in immune function, and an endorphin-mediated improvement in psychological well-being.[2-4] There are six basic groups of aerobic exercises: running/jogging/walking, swimming, cycling, skating, skiing, and rowing.

Aerobic power is a function of heart rate. Between 70–85% of the maximum heart rate (MHR) is equivalent to 60–80% of an individual's aerobic capacity. A number of formulas can be used to calculate the training heart rate (THR). An easy example to remember is

$$(220 - \text{AGE}) \times 40 - 85\% = \text{THR} \pm 10$$
$$\text{where MHR} = 220 - \text{AGE}$$

For all age groups, the MHR, THR, and aerobic capacity for women are slightly lower than those for men. As people age, the MHR and aerobic capacity decline.[5,6] Higher percentages (70–85%) are used to calculate the THR when the patient is healthy and has been living an active lifestyle. Proportionately lower percentages (40–60%) are used for patients who have been ill or sedentary.

At the physiologic level, anaerobic activities primarily increase the systemic vascular resistance, resulting in an elevation of not only the systolic but also the diastolic and the mean arterial blood pressures. There is minimal effect on the stroke volume and, therefore, the cardiac output. An additional effect of an anaerobic program is the increase in muscle mass through fiber hypertrophy and hyperplasia. Examples of predominantly anaerobic training exercises include weight lifting in the form of body building, power lifting, and Olympic lifting.

In general, most exercises have both aerobic and anaerobic components. For example, many of the racquet sports are stop-and-go and, therefore, consist of cardiovascular as well as metabolic activities that increase muscle bulk. Activities that involve repetitive use of large muscle groups for 20 minutes or more on a regular basis predominantly have an aerobic effect, whereas exercises that involve specific muscle groups that are stressed maximally for a minimal number of repetitions favor anaerobic metabolic pathways. Examples of the latter include isotonic (ie, lifting a dead weight) and isometric (ie, Charles Atlas type exercises) training programs. Finally, the warm-up period for all exercises, regardless of basic type, should begin with an anaerobic component.

PREPARTICIPATION EVALUATION

The preparticipation evaluation should be specific for an activity or sport. The history should include a review of systems, particularly of those systems pertaining to the proposed activity, and a history of medications, hobbies, and recreational and occupational activities. The physical examination should focus on the system that will be directly affected by the training program. For example, individuals interested in running/jogging/walking or a sport involving the lower extremities, such as soccer, hockey, or hiking, should have their ankles, feet, knees, and hips examined. Similarly, swimmers, racquet sport players, and individuals interested in throwing activities should have their upper extremities evaluated. All candi-

---

From the Department of Family Practice, State University of New York, Health Science Center at Brooklyn.

Address correspondence to Dr Birrer, Box 67, SUNY Health Science Center at Brooklyn, 450 Clarkson Ave, Brooklyn, NY 11203.

dates should have their cardiopulmonary systems examined at rest and during exercise.

Table I lists levels of participation by sport type for a variety of medical conditions. Participation in contact sports is contraindicated for individuals who have a single organ—such as an eye, kidney, or inguinal testes—and in any sport for individuals with acute inflammatory/infectious or uncontrolled metabolic conditions. Physician discretion is often required in other situations. For instance, a modified aerobic program would be highly beneficial for individuals with controlled hypertension or angina pectoris. Similarly, individuals with well-controlled seizure disorders should be permitted to participate in aquatic and flying activities. On the other hand, the candidate should be warned that seemingly benign leisure sporting activities such as hang-gliding, flying, and diving are not without significant risk of serious injury.

TABLE I. Common Disqualifying Medical Conditions by Sports Category

Medical Condition	Sports Categories		
	Contact	Noncontact	Leisure
General			
Acute infections/ inflammations	X*	X	X
Physical immaturity	X	PD†	—
Uncontrolled metabolic states (ie, diabetes, thyroid)	X	X	X
Serious hemorrhagic/ anemic disease	X	X	X
Cancer	PD	PD	PD
Eyes and ears			
Single eye	X	—	—
Severe myopia (>20/200 in one eye)	X	—	—
Severe hearing loss	PD	—	—
Respiratory			
Uncontrolled asthma	X	X	—
Pulmonary insufficiency	PD	PD	—
Recurrent pneumothorax	X	—	—
Cardiovascular			
Uncontrolled hypertension	X	X	PD
Symptomatic arrhythmia	X	X	—
Mitral valve prolapse	PD	PD	—
Thromboembolic disease	X	X	—
Previous surgery	PD	PD	—
Valvular/cyanotic heart disease	X	X	PD
3° Heart block	X	X	X
Abdominal			
Hepato/splenomegaly	X	X	—
Jaundice	X	X	X
Genitourinary			
Single kidney/ testis (inguinal)	X	—	—
Inguinal/femoral hernia	X	X	—
Chronic kidney disease	X	X	—
Musculoskeletal			
Significant spondylolisthesis/spondylosis	X	—	—
Functional inadequacy	X	X	—
Neurological			
Multiple concussions	X	—	—
Serious head trauma	X	—	—
Uncontrolled seizures	X	X	X
Previous surgery	X	—	—

* X = Disqualifying condition
† PD = Physician discretion required

COMPONENTS OF THE PRESCRIPTION

To maximize the benefit obtained from any aerobic activity, it is essential to determine the appropriate amount and frequency of exercise. The patient-athlete should be encouraged to exercise three to five times per week; less than three times a week will not produce a significant aerobic capacity, and training sessions more frequent than five times a week will not produce an appreciably greater gain but will significantly increase the risk of overuse injury.[7] Each training session should run for about 20–40 minutes at the THR. Sessions shorter than 20 minutes will not produce a substantial improvement of aerobic capacity, and sessions longer than 60 minutes are associated with a remarkable increase of overuse injuries as well as insignificant improvement of the aerobic capacity.

The patient must be instructed in the determination of the THR by taking the pulse rate. A variety of anatomical sites may be chosen, such as the femoral, radial, temporal, and brachial arteries, or the apex heartbeat may be measured. The rate should be taken for only five to six seconds. Longer periods will result in a decline of the THR and, therefore, aerobic conditioning. Individuals with a sensitive carotid sinus or clinical carotid artery disease, or who use digitalis, should be advised not to use the carotid arteries as a site for pulse determination. If there is difficulty in teaching the pulse assessment method, or if confounding factors are present—such as usage of beta-blockers—the individual can be trained to use the rating of perceived exertion (RPE) method. A scale of one to ten is taught, in which one represents no exertion and ten is reserved for the most physically exhausting activity. It has been shown that the RPE accurately correlates with the pulse assessment method in determining aerobic conditioning.[9] Furthermore, after several months of aerobic training, most individuals can determine when they are in the THR range without assessing the pulse rate.

All training periods should be preceded by a warm-up and followed by a cool-down period. The warm-up period allows the body to shift from anaerobic to aerobic metabolism. Stretching, flexing, range of motion, and strengthening exercises that complement the individual's planned activity should be incorporated into the warm-up period. For example, athletes who are planning a run/jog/walk program should strengthen their anterior musculature (abdominal muscles) and stretch the posterior muscles of the back, thighs, and legs. Similarly, individuals active in aquatic or racquet sports need to stretch their shoulders and upper trunk before beginning their workout. A longer warm-up period should be allowed for older patients, particularly those who have been confined to bed due to illness or hospitalization. A number of useful chair and bed exercise programs have been developed for such individuals.[9] There have been no controlled studies that indicate fewer injuries among people who regularly stretch or improve their flexibility before and after a workout session.

In summary, the complete aerobic program should be approximately 60 minutes in length: a 5–15-minute warm-up, a 20–40-minute core of aerobic conditioning at the THR, followed by a 5–15-minute cool-down period. For the average 30–40-year-old individual who has no health risk factors and who has led a fairly active life style, aerobic fitness should be achieved in approximately six weeks. In general, for every decade over 40 years of age, particularly for those individuals who have been sedentary, it takes an additional month to achieve a similar level of aerobic fitness. Individuals who have been hospitalized or ill for prolonged periods of time should allow an additional three to six months to achieve the same fitness level, depending on their illness profile.

PRECAUTIONS

The patient must be advised that during the initial weeks of the training program minimal amounts of exertion will be needed to achieve the THR as well as to cause the discomfort associated with training, including nausea, breathlessness, anxiety, and sweating. During the ensuing weeks, the contribution that the heart rate makes to the cardiac output will fall, and the amount due to stroke volume will increase. The individual will note that it will become progressively easier to achieve and maintain training goals. Longer distances and more rapid speeds will become the rule rather than the exception.

Some common precautions for a variety of training programs include avoiding smoking, beverages that contain caffeine, environmental extremes, inadequate or unsafe training equipment, or constricting, heavy training suits. Large meals and caffeinated beverages should be avoided prior to heavy physical activity. Although some recent research indicates that caffeine may be an ergogenic agent, it is also arrythmogenic and, therefore, should be particularly avoided by older individuals.[10] Smoking has no place in a fitness program, and nicotine itself is an arrythmogenic agent. Heavy clothing or special sweat suits made from polypropylene, neoprene, or nylon can produce hyperthermia and dehydration, particularly in very young and older patients with borderline volume homeostasis. Lastly, the training environment should consist of color-coded, well-lighted areas that are equipped with functioning equipment and easily available instructions. Dim lighting with poor color separation and malfunctioning equipment are responsible for many accidents, particularly among the elderly, who have reduced neuromuscular coordination and proprioception.

TYPES OF ACTIVITIES

Table II describes occupational and recreational activities based on their aerobic energy expenditures. One metabolic equivalent (MET) is equivalent to 3.5 mL/kg/min of oxygen consumed. The patient's recreational, occupational, and hobby history should be incorporated into the prescription. It is, therefore, possible to design a predominantly anaerobic, mixed, or aerobic program based on the energy requirement per activity. Activities at or below the 4 MET level have minimal to no aerobic input, whereas those above this level are associated with progressively increasing amounts of cardiopulmonary conditioning. Nonetheless, substantive gains in physical capacity can be made by the chronically ill, hospitalized, or elderly patient who has been sedentary or bedridden by utilizing the more basic levels of conditioning. For example, sewing, knitting, and model shipbuilding are excellent activities for range of motion and flexibility exercises, particularly for individuals with arthritis. Bed exercises, light walking, cycling, shuffleboard, fishing, or light wood-working are good for neuromuscular coordination and strengthening. For many older individuals, fashionable aerobic activities such as jogging, cycling, or aerobic dancing are of limited interest. However, the prescription of a meaningful preretirement occupational activity such as construction, carpentry, masonry, or heavy machine repair, or an enjoyable hobby such as gardening, splitting of hard wood, or similar outdoor domestic activities would be of merit.

Table II can also be used to create a profile of the prospective patient-athlete. Based on the current activities of the candidate, it is possible to predict with a fairly high degree of accuracy his current physical capacity as well as to educate him in terms of his commitment to a long-term aerobic program. It is not uncommon, for instance, to have an interested housewife state that she is already in good physical shape because she is actively involved in domestic housework, or to have a middle-aged man state the same due to his janitorial work or activities on the machine assembly line. Both individuals could be shown that they do not achieve significant aerobic training effects based on their current activities. In addition, studies had indicated that sexual intercourse with a regular partner involved 2–4 METS of activity, whereas the values for intercourse with an unfamiliar partner were substantially higher (7–10 METS).[11,12] More recent research indicates that it is not possible to predict the energy expenditure for sexual intercourse due to the inherent variability of circumstances and partners.[13]

The prescription should state the type, amount, frequency, and duration of exercise and should include any special instructions. A subsequent appointment is advisable, since the prescription will change as conditioning progresses. The patient should be encouraged to sign the prescription and should be given a copy in order to encourage a more personal level of involvement.

SPECIAL CONSIDERATIONS

The Elderly. The overall decline in functional capacity associated with aging is due to genetic and environmental factors. In general, there is a 30% reduction in physical activity capacity by the age of 70.[4] The major changes occur in the cardiovascular system. There is a 50% decline in myocardial oxygen consumption.[15] There is also blunting of respiratory equilibrium as well as a 30% decline in muscular strength due to atrophy of muscle fibers.[16] There is a 30% reduction in flexibility due to changes in collagen and synovial fluid. Bone mass decreases significantly, particularly in women, with a concomitant rise in the fracture rate.[17,18] These changes are more dramatic in individuals who have been sedentary throughout their lives, but are minimized in individuals who have led an active life. Thus, chronological age should not be equated with physiological or functional age. For instance, an active 65-year-old can be as fit as a sedentary college student. Regular aerobic exercise can dramatically improve myocardial oxygen consumption, strength, flexibility, and bone mass. Neuromuscular coordination can also be im-

TABLE II. Approximate Energy Expenditure for Common Physical Activities

Energy	Recreation	Occupation
1.5–2 METS 4–7 mL O_2/min/kg 2–2.5 Kcal/min	standing card playing sewing/knitting flying, motorcycling walking (1–2 mph or 1.6–3.2 kph) model ship building bed exercise	shaving/dressing/showering desk work electric typing/writing auto driving dusting/light housework calculator machine operation watch repair light assembly work hammering
2–3 METS 7–11 mL O_2/min/kg 2.5–4 Kcal/min	golf (powercart) bowling, billiards darts, piano playing fishing (standing, sitting), croquet power boating, shuffleboard skeet shooting, light woodworking walking (2–3 mph or 3.2–4.8 kph) cycling (5 mph or 8 kph) shopping kayaking, rowing, canoeing (2.5 mph or 4 kph) horseback riding (walk) horseshoe pitching	car washing auto repair kneading dough manual typing ironing/tailoring bartending heavy lever work, dredge riding power lawn mower TV, radio repair crane operator upholstering using hand tools cleaning/scrubbing/waxing washing/polishing/sanding
3–4 METS 11–14 mL O_2/min/kg 4–5 Kcal/min	calisthenics (light) fly fishing archery badminton (doubles)/tennis (triples) sailing (small boat) ice boating horseback riding (trot) badminton (social doubles) softball (non-competitive) musician (energetic) golf (pulling bag cart) volleyball (6-man non-competitive) walking (3–3.5 mph or 5–5.8 kph) cycling (6 mph or 10 kph)	janitorial work brick laying/plastering twisting cables/pulling on wires raking leaves machine assembly hitching trailers cranking up dollies mopping/hanging wash window cleaning light plumbing welding (moderate) plowing, tractor trailer-truck driving light lawn mower pushing wheelbarrow (100 lbs or 220 kg) assembly line work (light or medium parts appear at an approx. rate of 500/day or more or lifting 45 lb part every 5 minutes or so) stocking shelves, packing or unpacking light or medium objects
4–5 METS 14–18 mL O_2/min/kg 5–6 Kcal/min	table tennis golf (carrying bag) dancing (social) tennis (doubles) badminton (singles) calisthenics (moderate) swimming (light) tetherball baseball (non-competitive) walking (3.5–4 mph or 5.8–6.4 kph) cycling (6.5–8 mph or 10.4–12.8 kph) kayaking, rowing, canoeing (3 mph or 4.8 kph)	heavy machine repair (farm, airplane, plumbing) using powersaw on hardwood pushing a power mower/cart/dolly gardening/hoeing baling hay carpentry (light) painting masonry paperhanging interior repair/remodeling stair climbing (slow) putting in a sidewalk carrying trays/dishes gas station mechanic military marching lifting & carrying 20–44 lbs. general heavy industrial labor walking from room to room pumping tire plowing with a horse

Table II continued

Energy	Recreation	Occupation
5-6 METS 18-21 mL O_2/min/kg 6-7 Kcal/min	hunting (small game) fishing (wading) softball (competitive) horseback riding (posting-trot) soccer (noncompetitive) walking (4 mph or 6.4 kph) cycling (8-8.5 mph or 12.8-13.6 kph) kayaking, rowing, canoeing (4 mph or 6.4 kph) ice/roller skating (9 mph-15 kph)	garden digging shoveling light earth stair climbing (moderate) exterior remodeling or construction
6-7 METS 21-25 mL O_2/min/kg 7-8 Kcal/min	backpacking (5 lbs) tennis (singles) scuba diving (warm water) downhill skiing (light) water volleyball ski touring (2.5 mph or 4 kph) calisthenics (heavy) water skiing badminton (competitive) walking (5 mph or 8 kph) cross country hiking dancing (rumba, square) swimming (moderate)	shoveling 10 lbs or 22 kg 10×/min snow shoveling hand lawn mowing splitting wood using pneumatic tools carry/lifting 45-64 lbs spading
7-8 METS 25-28 mL O_2/min/kg	horseback riding (gallop) snowshoeing (3 mph or 4.8 kph) sledding/tobogganing ice hockey touch/flag football paddle ball swimming (fast) mountain climbing basketball (nongame) walking (6 mph or 9.6 kph) cycling (12 mph or 19 kph) kayaking, rowing, canoeing (5 mph or 8 kph) jogging (5 mph or 8 kph) downhill skiing (vigorous)	stair climbing (fast) ditch digging hand saw/ax on hardwood carrying/lifting 65-84 lbs laying railroad track carrying 20 lbs up stairs
8-9 METS 28-32 mL O_2/min/kg 10-11 Kcal/min	light sparring (boxing, martial arts) fencing scuba diving (cold water) handball (social) endurance motorcycling basketball (vigorous) soccer (competitive) squash (social) running (5.5 mph or 9 kph) cycling (13 mph or 21 kph) ski touring (4 mph or 6.5 kph) in loose snow back packing (30 lbs)	carrying/lifting 85-100 lbs shoveling 14 lbs or 31 kg/10×/min. tending furnace using a pick using bar, sledgehammer climbing a ladder moving/pushing desks, file cabinets stock furniture; also pushing against heavy spring tension shoveling 16 lbs or 35 kg 10×/min
10+ METS 32+ mL O_2/min/kg 11+ Kcal/min	basketball (competitive) hunting (big game) heavy sparring (boxing, martial arts) ski touring (5+ mph or 8+ kph) running 6 mph = 10 METS 7 mph = 11.5 METS 8 mph = 13.5 METS 9 mph = 15 METS 10 mph = 17 METS cycling 14 mph = 10 METS 15 mph = 11.5 METS 16 mph = 12.5 METS swimming 850 yds/18-20 min = 10 METS (crawl) 950 yds/20-22 min = 11 METS	

Table II continued

Energy	Recreation	Occupation
10+ METS 32+ mL O_2/min/kg 11+ Kcal/min	swimming 1000 yds/20–22 min = 12 METS (*cont.*) gymnastics judo spaceball trampolining wrestling	

At rest, 1 MET = 3.5 mL O_2/kg/min
These values are for a 70 kg person
Some activities may have a large range of metabolic activity due to excitement, stress, anxiety, or impatience.

proved. In general, such programs need to be slowly progressive, to allow adequate warm-up and cool-down periods, and to utilize a broad spectrum of activities.

Arthritis and Obesity. The patient with arthritis that is in remission should be encouraged to participate in regular aerobic activities.[19] Aquatic activities are particularly beneficial, as there is no weight to bear. The amount of aerobic fitness will be determined by the amount of disability present. The warm-up and cool-down exercises are essential and should incorporate any hobbies or similar occupational activities that would improve range of motion and flexibility of the affected joints. During periods of acute flare-up of arthritis, the joints should be rested and the appropriate anti-inflammatory medications prescribed. Thereafter, progressive active and passive range of motion activities should be begun within the limits of pain and in conjunction with an active rehabilitation program. Those patients who are devoted runners/joggers/walkers can perform such activities in the shallow part of a swimming pool or in deeper water utilizing a neoprene suit. Rowing, cycling, and stationary cross-country ski machines can also be used to supplement aerobic training without overburdening the weight-bearing joints of the lower extremities. Similarly, the obese patient should be encouraged to take up training activities that do not overly burden the joints of the lower extremities.

Diabetes and Peripheral Vascular Disease. Regular aerobic exercise improves microcirculation and glucose metabolism. All patients with controlled diabetes or peripheral vascular disease (PVD) should be encouraged to exercise regularly. Insulin and oral sulfonylurea requirements are often reduced by 50% once a full aerobic training program has been started.[20] Hypoglycemic episodes are far less likely in the exercising diabetic individual.[21] Insulin injection sites must be modified in order to allow for differential absorption in exercising muscles. For instance, the abdominal panniculus, rather than the thigh, should be chosen for individuals who are running/jogging/walking. The feet of patients with diabetes or PVD should be examined regularly due to their enhanced vulnerability, particularly in running/jogging/walking. Once again, swimming is an excellent alternative, since it eliminates trauma to the feet. The feet must, however, be carefully dried to prevent maceration.

Chronic Obstructive Pulmonary Disease and Asthma. Regular aerobic conditioning has been shown to improve pulmonary function tests of individuals with chronic obstructive pulmonary disease (COPD) and asthma, particularly exercise-induced asthma (EIA).[22] Depending on the severity of the disease, a program can be tailored to involve progressive increases in the amount of aerobic training. As enhanced fitness levels are achieved, the amount of medication will often require reduction. Individuals with EIA usually fare well with inhalation of cromolyn sodium or metaproterenol before engaging in their planned activity.

Cardiovascular Disease. Although a maximal treadmill examination is recommended for individuals over the age of 40 years, this is often an unrealistic goal. All individuals, irrespective of age, who are found to be healthy based on their medical history and a physical examination, should be allowed to begin a progressive aerobic training program of their choice based on one of the age-based formulas. The prescription can be supplemented by a Master's step-test or the Kasch pulse recovery method in the practitioner's office.[23] Blood pressure readings and electrocardiographic monitoring enhances the validity of these tests. The results of these standard evaluations have been shown to correlate well with the standard treadmill examination.

On the other hand, individuals who have clinical evidence suggestive of cardiovascular disease should have a treadmill test. If one is screening for cardiovascular disease, the examination should be done with the patient off medication, whereas if cardiovascular disease has already been confirmed, the examination for the exercise prescription should be done with the appropriate medications being taken. Patients who have had a myocardial infarction should undergo a submaximal treadmill examination before discharge from the hospital in order to facilitate cardiac rehabilitation.[24] The test should be repeated at two and six months. Often, older patients (more than 65 years of age) will find the maximum treadmill examination a threatening experience due to a lack of neuromuscular coordination, concurrent disease, or significantly reduced functional capacity. A bicycle ergometer may be more acceptable, but patients with varicose veins, sparse gluteal pads, prostate disease, or varicoceles may find the ergometer uncomfortable. The physician should become familiar with the signs and symptoms of exercise intolerance (Table III) and should teach them to his patients. Inability to achieve projected fitness levels or the development of other signs or symptoms of exercise intolerance should prompt reevaluation.

TABLE III. Signs and Symptoms of Exercise Intolerance

- Dizziness/near syncope
- Angina
- Nausea
- Marked dyspnea
- Severe pain (eg, claudication)
- Staggering or persistent uncoordination
- Mental confusion
- Cyanosis/pallor
- Lack of skin flush following compression
- Loss of sustained vigor of palpable pulse
- Severe distress/exhaustion
- Significant chest pain
- Significant rise/fall in blood pressure
- Wheezing, rales, third heart sound
- Onset of arrhythmia/conduction disturbance
- Marked ST-T changes
- Patient's request to stop

COMPLIANCE

The mere writing of an activity prescription does not guarantee fitness. Educational level, income, and family and community resources have a significant impact on compliance.[25,26] The perceived susceptibility to disease and the severity of the disease are key factors in behavior modification. Safety, convenience, cultural influences, and efficacy are also important. For instance, the elderly or the indigent patient often does not have the resources to gain access to many training programs. Additionally, the possibility of an early myocardial infarction may not be a sufficient threat to motivate a healthy adolescent or young adult to a lifelong habit of exercise. Regular reinforcement through revisits, telephone calls, written reminders, and direct supervision is necessary to assure program compliance.

CONCLUSIONS

Primary care physicians should regularly incorporate the activity prescription in the comprehensive care of their patients. A clear understanding of basic exercise physiology and evaluation is essential. The type, intensity, frequency, and duration of the program can be quantified. Progress should be noted, problems identified, and the prescription modified accordingly. Behavior modification may be necessary to assure program success.

REFERENCES

1. Morris JN, Pollard R, Everitt MG, et al: Vigorous exercise in leisure time: Protection against coronary heart disease. *Lancet* 1980; 2:1207-1210.
2. Nadel ER: Physiological adaptations to aerobic training. *Am Sci* 1985; 73:334-343.
3. Vodak PA, Wood PD, Haskell WL, et al: HDL-cholesterol and other plasma lipid and lipoprotein concentrations in middle-aged male and female tennis players. *Metabolism* 1980; 29: 745-752.
4. Blumenthal JA, Williams RS, Wallace AG, et al: Psychological changes accompany aerobic exercise in healthy middle-aged adults. *Psychosom Med* 1982; 44:529-536.
5. Williams DH, Williams C: Cardiovascular and metabolic response of trained and untrained middle-aged men to a graded treadmill walking test. *Br J Sports Med* 1983; 17:110-116.
6. Drinkwater BL, Horvath SM, Wells CL: Aerobic power of females ages 10-68. *J Gerontology* 1975; 30:385-394.
7. Wenger NK: Exercise prescription. *Singapore Med J* 1981; 22:68-75.
8. Borg G: Psychological aspects of physical activity, in Larson LA (ed): *Fitness, Health and Work Capacity.* New York, Macmillan, 1974, p 141.
9. Sawaka MN, Glaser RM, Laubach LL, et al: Wheelchair exercise performance of the young, middle-aged, and elderly. *J Appl Physiol* 1981; 50:824-828.
10. Delbeke FT, Debackere M: Caffeine: Use and abuse in sports. *Int J Sports Med* 1984; 5:179-182.
11. Ueno M: The so-called coitian death. *Jpn J Legal Med* 1963; 17:333-340.
12. Datey KK: Sex and heart disease. *J Assoc Physicians India.* 1977; 25:647-652.
13. Bohlen JG, Held JP, Sanderson MO: Heart rate, rate-pressure product and oxygen uptake during four sexual activities. *Arch Intern Med* 1984; 144:1745-1748.
14. Smith EL, Gilligan C: Physical activity prescription for the older adult. *Phys Sportmed* 1983; 11:91-101.
15. Heath GW, Hagberg JM, Ehsani AE, et al: A physiological comparison of young and older endurance athletes. *J Appl Physiol* 1981; 51:634-640.
16. Murray MP, Gardner GM, Mollinger LA, et al: Strength of isometric and isokinetic contractions: Knee muscles of men aged 20-86. *Phys Ther* 1980; 412-419.
17. Smith El, Sempos CT, Purvis RW: Bone mass and strength decline with age, in Smith EL, Serfass RC (eds): *Exercise and Aging—The Scientific Basis.* New Jersey, Enslow Publishing Inc, 1981, pp 59-88.
18. Dalen N, Olsson KE: Bone mineral content and physical activity. *Acta Orthop Scand* 1974; 45:170-74.
19. MacKeen PC, Franklin BA, Nicholas WC, et al: Body composition, physical work capacity and physical activity habits at 18 month follow-up of middle-aged women participating in an exercise intervention program. *Int J Obesity* 1983; 7:61-71.
20. Devlin JT, Horton ES: An exercise regimen for diabetes. *Cardiovasc Med* 1985; 5:65-71.
21. Zinman B: Exercise in diabetes treatment. *Clin Diabetes* 1983; 1:18-21.
22. Stewart RI: Exercise in patients with chronic obstructive pulmonary disease. *S Afr Med J* 1985; 67:87-89.
23. Kasch F: A comparison of the exercise tolerance of post rheumatic and normal boys. *J Assoc Phys Ment Rehab* 1961; 15:35.
24. Blincoe WA, Lutz JF: The prognostic value of exercise testing. *Cardiovasc Rev Rep* 1985; 4:347-363.
25. Becker MH (ed): The health belief model and personal health behavior. *Health Education Monogram* 1975; 2:326-473.
26. Becker MH, Maimann LA: Sociobehavioral determinants of compliance with health and medical care recommendations. *Med Care* 1975; 13:10.

Treatment of acute sports injuries

JOHN BAUM, MD

In today's sports-minded environment, the primary care physician is likely to see more patients who are involved in sports activities. In past years, this group has primarily been limited to school-age children and young adults. There is usually a school physician on hand to take care of school children, and there are team physicians in the universities. However, primary care physicians may be called on to treat athletic injuries among people now engaged in sports. Examples of the activities that can produce injuries are running, cycling, weight training, and cross-country and downhill skiing.

While patients with severe injuries such as fractures will usually be referred to orthopedic surgeons, primary care physicians should be ready to care for patients with a variety of minor injuries.

EPIDEMIOLOGY OF SPORTS INJURIES

Since jogging and running require minimal equipment, they have become the favorite activities of a large segment of the new athletic population. As in all sports, there is a range of activity, from the around-the-block huffer and puffer to the marathoner. Within this range, a number of different injuries can occur with varying frequencies. Whereas it is relatively easy to determine how many fractures occur on the ski slopes, it is difficult to obtain accurate statistics on the frequency of minor problems such as sprains, strains, and muscle injuries.

ETIOLOGY AND DIAGNOSIS OF STRESS FRACTURE

Stress fractures are now well recognized as a problem among runners who log 50 to 70 miles per week. The injury is also seen in professionals, such as dancers, who stress their feet. The onset is subtle and insidious, with the patient first noticing foot or limb pain that appears during physical activity. As patients continue their usual rate of activity, the symptoms seem to appear earlier in the course of the specific physical exertion. Occasionally, there is local swelling at the site of the injury, but often there is no external feature that can be used for diagnostic purposes. Since individuals who develop this problem are often dedicated to their sport, it is not uncommon to see them trying to "tough it out," until they realize that they must seek a physician's advice.

A stress fracture can occur in almost any bone that has been exposed to repetitive stress, but in runners, it is often seen in the lower third of the tibia. It can also be present in any of the bones of the feet. It has even been reported in the iliac crest in runners. Stress fractures have been noted in persons who have undertaken an activity without proper training, as well as among persons who have pushed themselves to a presumably excessive limit.

On physical examination, one may find a localized area of bone pain; local soft tissue swelling may be apparent. Positive radiographic findings will show changes in subperiosteal new bone formation. This finding is usually not seen until three or more weeks after the condition is defined. What one sees at this point is the increased density associated with bone repair. With a bone scan, positive findings can be seen in about two weeks.

THERAPY FOR STRESS FRACTURES

The specific treatment of stress fractures is rest. It will alleviate the symptoms, which will eventually disappear as the fracture heals. Treatment beyond simple rest is unnecessary. Unfortunately, the use of ice and the anesthetic effect it produces can have the negative effect of providing enough temporary relief to allow a dedicated runner to resume his or her activity before there is sufficient healing.

An important point to consider is the role of the running shoe, which can be an unrecognized cause of problems. It has recently been demonstrated that the cushioning of a running shoe will wear down.[1] Almost all shoes have a half-life of about six months. Runners should be advised to buy at least two pairs of running shoes of different styles (straight last and curved last), preferably from different companies, and to rotate them for each run. Since the shoes will probably wear at different rates at different sites, the stress on different parts of the anatomy will shift, proving beneficial for the feet, lower limbs, and particularly the knees.

GENERAL THERAPY OF ACUTE SPORTS INJURIES

The standard therapy in acute traumatic injuries is best described by the acronym "RICE" (R—EST, I—CE, C—OMPRESSION, E—LEVATION).

"Rest" can be interpreted in a number of ways. With an ankle injury, for example, there is little that the individual

---

Dr Baum is Professor of Medicine and Pediatrics and of Preventive, Family, and Rehabilitation Medicine, University of Rochester School of Medicine and Dentistry, Rochester, NY.

Address correspondence to Dr Baum, Director, Arthritis and Clinical Immunology Unit, Monroe Community Hospital, 435 East Henrietta Rd, Rochester, NY 14603.

can do except to stay off the affected limb. This joint can be given rest by use of a cane or crutches to eliminate weight bearing. The immediate rest of an injured joint or muscle means the cessation of activity of the injured part to prevent further tissue damage. In addition, rest will cut down on further local bleeding and will help avoid the development of a space-occupying hematoma. Rest can also be part of other procedures that are utilized in the compression modality. Taping, splinting, and bracing will serve to accomplish this dual function.

As noted above, it is sometimes difficult to get dedicated athletes to rest long enough. This has been described as "negative addiction."[2] Because these individuals think they must have their daily exercise and truly believe they cannot live without it, they are particularly resistant to rest if they have had an injury. These individuals will run in spite of constant pain, taking analgesics or liberally icing the injured area. Such runners often consider their running the most important thing in their lives. Their primary care physician has an important role in not only prescribing the proper therapy but in convincing them to accept it.

CRYOTHERAPY

The use of ice, or cryotherapy, is a remarkably effective treatment of acute athletic injuries.[3] A variety of treatment forms exist, including ice packs, ice sticks, and vapocoolants (ethyl chloride spray). Chemical packs are a recent development and must be handled with care. The chemicals can occasionally leak and cause local chemical burns.[4] When an ice pack is used, the materials can be brought to a playing field in an ice chest, but the physician must be able to tell patients how to use ice at home. When an ice bag is used, the patient should be instructed to crush the ice cubes, so that the ice bag can be molded around the site of the injury. This provides an equal distribution of the cold so as to achieve a more rapid and complete cooling of the area. In fact, there should be an attempt to get the ice around the limb. Thus, two packs may be preferable.

Cold should be applied for about 20 minutes per hour for 24 hours after the injury. The amount of time that the ice is applied will vary with the amount of overlying fat. Since ice is most effective by direct application to the skin surface, there should not be any interposing materials. However, if compression is being used at the same time, the elastic bandage should be thoroughly soaked in cool water.[4] This will permit more rapid and thorough cooling of the underlying skin and muscle. It will take longer to cool down an injured area in an obese individual—up to 30 minutes. Conversely, in a very thin individual, ten minutes of application may be sufficient. Some physicians recommend that the ice be applied for three to five days, by which time the swelling of the injured area (knee and ankle joints in this case) should have resolved.[5]

Other methods of applying cold require some prior preparation. Ice massage is an effective way of delivering cold. A simple method is to freeze styrofoam cups of water.[3] When needed, the top of the cup can be peeled away and the cup itself used as an insulated grip. Although this technique is best used for a muscle injury in which there is a large area to cover, it can be used on joints or other localized sites.

Another chemical method for cooling an area of the body is the use of ethyl chloride spray. However, this spray should only be handled by someone experienced in its use. Prolonged use over a limited area can produce frostbite and tissue damage within minutes.

Cold has a primary role as a rapid anesthetic agent. The patient first feels the sensation of cold. This is followed by sensations of burning, pain, and finally numbness. At this stage, further application of cold can cause frostbite. This final stage may occur in about ten to 15 minutes. Beneficial physiologic changes include vasoconstriction, which reduces the amount of blood loss into the tissues. In addition, the cold reduces the metabolic activity of the tissue and, by reducing the need for oxygen, protects the tissue from further damage. The more quickly the ice is applied after an injury, the more effective it is. Thus, it is best to keep ice available for application during almost any type of athletic activity. Since the "ice-stick" is held by the individual applying it, it may be one of the safest ways to use cold. There is less danger when the condition of the skin can be observed and the response monitored.

Ice can also be used in subsequent therapy to restore function. Its analgesic properties are helpful when exercise of a previously injured muscle or joint is started. It must be used carefully in such cases, since there is some danger of overstrain or overuse under the protection of the cold-induced analgesia.

COMPRESSION

It has been stated that compression is the most effective measure to prevent swelling following an acute injury. The use, effectiveness, and rationale of compression have been well described by Wilkerson.[6] After trauma causing damage to tissue and vessels, a number of substances related to the inflammatory reaction are released. These substances cause vasodilation and increase the permeability of blood vessels. This allows for the egress of fluid and cells from the circulation into the surrounding tissue. The serum proteins that are now present in the interstitial tissue can hold large amounts of fluid because of the increase in osmotic pressure outside the vessels. The fluid, known as edema fluid, will restrict blood flow in the area by compressing the tissue and the vessels. In addition to separating tissue structures, the compression of vessels will reduce the local delivery of oxygen to the tissues. Pain is produced by local pressure of the fluid on nerve endings as well as by the distention of tissues. Eventually, healing of the tissue can be delayed because of the physical presence of large amounts of fluid that coagulates into a mass of fibrin, other proteins, and inflammatory cells.

This flow of fluid can be restricted and diminished by compression.[6] This maneuver increases pressure on the tissues to overcome the effect of osmotic pressure, prevents the egress of fluid from vessels, and decreases the amount of material (serum fluid and protein) that can accumulate in the interstitial tissue spaces. Basically the effect of compression is to reduce the ability of fluid to escape from damaged vessels into the surrounding tissue. Any fluid that has already escaped is spread throughout the area and is more accessible for return to the circulation by neighboring lymphatics, rather than by any direct effect of compression.

Compression can be applied by manual pressure with an ice bag, by bandaging, or by the application of tight fitting splints. There are two main types of material that are used to compress and support an injured joint: plain elastic and elastic adhesive bandages. Plain elastic bandages better preserve their elasticity and are significantly more elastic than adhesive bandages when initially tested.[7]

Maximum decrease in the effectiveness of compression of an injured leg occurs after walking for only 15 minutes. Therefore, resting the limb while using a compressive bandage will maintain the value of the compressive force.[7] If a patient is walking, the bandage should be reapplied at least once per day and preferably more often depending on the amount of usage of the limb.

ELEVATION

Elevation of an injured limb will also help to reduce swelling by reducing the flow of fluid from the vessels. Gravity is used to counteract intraluminal pressure in the vessels. Elevation cuts down on leakage from damaged vessels and from those that have dilated due to the secondary effect of inflammatory substances released after tissue damage. Elevation also serves the purpose of enforcing rest. If the trauma has been to the lower limb then elevation ensures immobilization.

CONCLUSIONS

Although it may appear that caring for acute sports injuries is simplistic, treatment is based on a sophisticated knowledge of the results of injury and tissue responses. Speed in applying these principles is essential. The rapid application of ice and compression can make a difference in terms of days needed for recuperation.

When one suspects that there is more severe injury—such as tendon tears or fractures—this therapy is truly first aid, and the patient should be removed immediately to a site where more appropriate and intensive care is available. Use of the basic treatment program—Rest, Ice, Compression, Elevation—will reduce the recuperation time even when more extensive injuries have occurred.

REFERENCES

1. Cook SD, Kester MA, Brunet ME, et al: Biomechanics of running shoe performance. *Clin Sports Med* 1985; 4:619-626.
2. Morgan WP: Negative addiction in runners. *Phys Sportsmed* 1979; 7:56-70.
3. Stamford B: Using cold for sports injuries. *Phys Sportsmed* 1985; 13:148.
4. Cidek JJ: Cryotherapy. *Clev Clin Quart* 1985; 52:193-201.
5. O'Brien WJ: Physical therapy in maintenance of athletic performance, in Appenzeller O, Atkinson R (eds): *Sports Medicine*. Baltimore, Urban and Swartzberg, 1983, pp 421-428.
6. Wilkerson GB: Treatment of ankle sprains with external compression and early mobilization. *Phys Sports Med* 1985; 13:97-106.
7. Viljakka T: Mechanics of knee and ankle bandages. *Acta Orthop Scand* 1986; 57:54-58.

FROM THE LIBRARY

ATHLETIC INJURIES

With the football season now at hand the problems of injury during practice and contest deserve consideration.

The known higher incidence of injury in intramural athletics over varsity sports is related to the inferior quality and quantity of physical training and conditioning. The effect of sudden, forceful body impact while in motion must be prepared for adequately by rigorous training. We are all aware of the Monday morning state of the touch football enthusiast, the result of once-a-week play.

When injury occurs, the first essential is a complete and thorough examination on the spot by one competently trained in the technic. Too often an initial diagnosis is missed because of a lack of swelling and other acute symptoms, and there is often the desire of the player to minimize his symptoms to get back in the game. An adequate examination is, for the most part, impossible on the playing field; it can only be carried out properly in the locker room where there is an opportunity to remove clothing and put the injured member through a range of motion, which will indicate the degree of injury. Those with head injuries should always be removed by stretcher and should be kept under close observation until a medical decision is made.

There is always pressure to return the injured player to competition, particularly if he is a valued member, and one must not be swayed if there has been noteworthy injury. This is especially true in injuries to the knee and shoulder.

It is important where there has been significant knee injury to take the time to re-educate and redevelop the quadriceps muscle group. It is well known that this muscle practically melts away after substantial injury and requires a thorough period of strengthening before it can be considered as strong as its fellow on the opposite extremity.

Likewise, the dislocated shoulder requires more respect than it usually receives. In spite of the lack of symptoms, this injury requires immobilization in a neutral position preventing abduction and external rotation for not less than five weeks. A subsequent period of five to six weeks is required to regain the functional range of motion. If this injury were given major attention there would be fewer chronic, recurrent dislocations of the shoulder.

Whether in high school or college competition, competent coaches and trainers backed by medical skill are an important safety ingredient that every athletic team should have.—P.M.W.

EDITORIAL
(*NY State J Med* 1961; 61:3236)

The child and adolescent in sports

THOMAS L. WICKIEWICZ, MD

The past twenty years have witnessed a mounting interest in physical fitness, vigorous recreation, and competitive sports. Most people enjoy and need competition, not only for personal satisfaction, but often to gain social acceptance. This is particularly true of adolescents.

Older children frequently have their best competitive experiences in sports activities. Today a wide selection of sports is available to both sexes. In addition, competitive sports conducted with organized teams or leagues on community recreation fields far outnumber those available in the school systems. It is estimated that more than 25 million boys and girls participate in competitive school sports, and nearly another 20 million engage in out-of-school sports.[1,2] Unfortunately, this latter group may not have the basic health safeguards that are important for the young athlete.

"Team physicians" cannot adequately provide the health protection and medical treatment that is required for these young athletes. This must come from primary care providers. Physicians have three major roles in sports medicine: one is to provide health services, the second is to counsel the individual with regard to the choice of sport as well as his or her fitness for that sport, and the third is to instruct the individual in proper conditioning techniques that he or she will need to engage in a particular sport.

PREPARTICIPATION SCREENING

An appropriate preparticipation examination of the young athlete requires both an evaluation of the individual's overall health and a consideration of his or her functional ability, growth, and maturation. The preparticipation examination should not simply be a procedure to separate the fit from the unfit. There is a wide variety of sports available to the young athlete and these often have different physical requirements. The demand on the participant for contact, endurance, or skill varies greatly from sport to sport. Thus, the physician can usually recommend some type of sports activity, even for the relatively unfit individual or for the physically impaired. Unfortunately, many coaches and group leaders are uninformed about children's maturation levels, health needs, emotions, and variations in their potential for physical performance, and thus the burden for appropriate counseling falls on the physician.

Good health is not the only factor to be considered in determining whether a child is fit to play a particular sport. The physician must insure that the individual is qualified for safe participation in that sport. Appropriate physical conditioning, which includes adequate endurance, overall strength, agility, and flexibility, is important. Depending on the time of year, acclimatization to heat and humidity may be essential for the young athlete's safety and performance.

Studies have shown that a routine preparticipation examination which focuses on general medical health has a very low yield, with up to as many as one third of the musculoskeletal injuries of children being missed by the primary care provider in the initial exam.[3] Rather, the emphasis should be on an adequate history, particularly of previous injuries related to sporting activity, and an appropriate sports-specific examination.[4] Ideally, the history should initially be reviewed at home by the young athlete and his or her parents and then reviewed by the primary care provider. A system that may work well would require an examination upon entry into school and sports participation. This would then be followed up with a yearly questionnaire which would update the athlete's injury history. With good continuity of care this system would work well for the younger athlete.

The examination should focus on the body systems primarily involved in the sport. For example, the muscles and bones of the lower extremity are important for soccer, track and field activities, and football. The upper extremity must be examined for swimming, racquet sports, and throwing activities (eg, baseball). An examination of the head, eyes, ears, nose, and throat is reserved for flying and diving sports.

An examination of the cardiovascular system should be performed on all candidates. The emphasis should be on the history, especially any history of heart disease in the family, fainting, or dizziness associated with physical activity. With the younger individual, the individual's level of activity can be compared to that of his or her peers. Any positive findings on the physical examination, such as an organic murmur or hypertension, should be evaluated by appropriately trained personnel.

With regard to the individual's fitness for a particular sport, a preseason evaluation utilizing various field tests is appropriate. A measure of the individual's endurance should be obtained. Various aerobic measures, such as a timed run or a run over a set distance, are good measures of this particular parameter. Measures of the individual's

---

Dr Wickiewicz is Assistant Professor of Clinical Surgery, Cornell University Medical College, New York, NY.

Address correspondence to Dr Wickiewicz, 535 E 70th St, New York, NY 10021.

strength and flexibility are also important. The examiner should understand the requirements of the particular sport that the athlete is interested in and should match those requirements with the measures of fitness listed above. The goal of the preparticipation examination should not be to disqualify, but rather to ensure that the participants are qualified for a specific sport.

Studies that have focused on the prevalence of injuries in the child and adolescent during sports participation[5,6] have shown that childhood is actually a very safe time to participate in sports. A common finding of these studies has been that the rate of injury increases during adolescence, with the peak injury age being at around 15 years in males and around 14 years in females. In general, football, basketball, wrestling, soccer, and track account for the majority of injuries in males, and gymnastics, soccer, and track account for the majority of those in females.[5] One must be careful not to infer that these are unsafe sports, however. Rather, these sports more than likely account for the majority of athlete-hours of participation.

TYPES OF SPORTS INJURIES

It is convenient to break down sports injuries into two types: macrotrauma and microtrauma. Macrotrauma is an injury to a specific tissue as a result of a singular event; for example, a broken wrist in a gymnast occurring as a result of a fall. Microtrauma consists of overuse injuries, where the repetitive stresses of the various sports lead to failure in either the bone or the muscle-tendon with subsequent injury.[7] An example is a stress fracture in a long-distance runner.

The rate of macrotrauma has been shown to be equal in males and females participating in similar sports.[5] It has also been shown that organized sports in general are either as safe as or, in some studies, safer than free play with regard to specific injuries. Females are more at risk of microtrauma than males, however, because of anatomic differences.

Microtrauma. Certain risk factors appear to be important in the development of an overuse injury. The most common has to do with training errors, including too frequent or too intense participation for the athlete at that level of training. This is especially common in the young population participating in sport-specific summer camps, where both peer pressure and inadequate instruction lead to overuse. Overuse injuries are almost never seen in a child at free play.

The second risk factor is a musculotendinous imbalance where inadequate strength or flexibility leads to problems. An example is the young thrower who, because of inadequate rotation at the shoulder, uses poor throwing mechanics which leads to an overuse syndrome elsewhere in the upper extremity.

A third risk factor is an anatomic malalignment, such as leg length inequality, tibia vara, or femoral anteversion. Young aspiring ballerinas with this latter anatomic variation are especially prone to problems because of a lack of adequate external rotation at the hip. Poor technique is used in performing a turn-out, thus placing excessive stress upon the knee and foot.

Improper playing surfaces may also contribute to overuse injuries. An example of this is when the high school track team moves indoors during the winter, and the young runners are suddenly forced to run on a different, and often excessively hard, surface.

Associated disease states such as tarsal coalitions or mild hip dysplasias, while they may be asymptomatic for the rigors of daily activities, will become predisposing factors when subjected to the stresses of sports training.

The final risk factor is the growth spurt. During the time of the second or adolescent growth spurt there is an imbalance between the skeletal system and the muscle-tendon unit, with a relative tightness of the muscle-tendon unit because of rapid bone growth.[8] This leads to a loss of flexibility and an enhancement of the risks for overuse injury. The second growth spurt often occurs at a time when the adolescent is exposed to intense, repetitive training and conditioning for his or her high school sports.

Macrotrauma. The majority of acute, traumatic injuries in the child and adolescent involve either sprains of the ligament complex or injuries of the bone. The major difference between the growing bone and the adult bone is the open growth plate. Depending on the individual's age and the mechanism of injury, bone injuries may result in complete fractures or, in younger individuals, in variations such as torus fractures or greenstick fractures. The highest incidence of epiphyseal separations occurs in the 12- to 13-year-old group, probably because this is a vulnerable time for the growth plate. This results from both a weakening of the perichondrial ring and the individual's becoming larger, thus generating more force in his or her sports activities.[9]

The final pattern of injury that can be seen is an epiphyseal avulsion. The individual with an open growth plate can in fact suffer a ligamentous injury, particularly around the knee. Ligament disruptions, especially those of the anterior cruciate ligament, can often be seen in the child and adolescent population.

SPECIFIC SPORTS INJURIES

Head and Neck. Head and neck injuries are fortunately uncommon in sports and this is true among young athletes as well.[10] Macrotrauma may result in concussive lesions. In general, any injury which results in a loss of consciousness, no matter how brief, should preclude that child from participation in sports activities for the remainder of the day. Should there be a prolonged loss of consciousness—that is, greater than a minute—a prolonged postconcussive syndrome, or a seizure related to any head injury, then a complete evaluation is mandatory. All children with any type of concussive injury should be evaluated by a physician.

Traction injuries of the brachial plexus, commonly referred to as "burners," are most often transient, although when first evaluated they can look very serious because of the associated numbness and paralysis. If the injury is transient and the child or adolescent regains full motion, strength, and sensation in the upper extremity, then he or she may be allowed to return to play. However, should any of these parameters not return, the child should be restrained from participation and fully evaluated. Late sequelae of this injury, with a prolonged period of recovery of muscle strength, does sometimes occur. Often the young athlete is evaluated and treated for a torn rotator cuff because of abnormal movement of the shoulder, when in fact there is a brachial plexopathy. These conditions should be resolved before the individual is allowed to return to sports participation.

Shoulder. Macrotrauma to the shoulder joint in the very young child may result in either epiphyseal injury or shoulder dislocation, both of which require appropriate orthopedic care. Rotator cuff tears are very uncommon in the adolescent but may occur postdislocation. Pain is an important indicator that this has occurred. An individual who has an inordinately high level of pain for several weeks following a dislocation may have an associated rotator cuff injury.

Microtrauma to the shoulder is more common than macrotrauma. "Little League shoulder"[11] or microfracture of the proximal humeral growth plate can occur from overuse. The characteristic x-ray appearance of this is a widening of the epiphysis, demineralization, and fragmentation of the head without evidence of avascular necrosis. Rest is the appropriate treatment, and the individual should not be allowed to throw until the syndrome has resolved.

Another condition is subluxation of the shoulder in individuals who are stressing the shoulder such as in throwing sports or in swimming. These individuals may primarily present with complaints of pain rather than instability and can sometimes pose a diagnostic dilemma. The position of the arm at the moment of greatest pain is important. In throwing, for example, the anterior structures of the shoulder are maximally stressed at the beginning of the acceleration phase, while at the moment of ball release the posterior structures of the shoulder are maximally stressed. Subluxation can sometimes be difficult to differentiate from impingement or supraspinatus bursitis, another overuse syndrome. The latter most commonly causes anterior shoulder discomfort with overhead activities and can be brought on by overuse. The swimmer, for example, because of the repetitive movement of his or her shoulder during the recovery phase, will often irritate the contents of the subacromial space of the shoulder. As in all overuse syndromes, initial treatment of the impingement syndrome consists of rest, ice applications, and occasional use of nonsteroidal medications. Appropriate orthopedic consultation is indicated if a patient is refractory to these types of treatments.

Elbow. Another problem of the upper extremity is that of "Little League elbow," which is really a constellation of findings caused by overuse.[7,11] During the act of throwing, the elbow is subjected to compressive forces on the lateral side and traction forces on the medial side. This can result in damage to the radial head in the form of an aseptic necrosis or in a traumatic osteochondritis of the capitellum. Similarly, due to the excessive traction forces, there is irritation of the medial epicondylian structures. Again, treatment consists of rest. Should any of the characteristic x-ray appearances become evident the individual should not be allowed to throw until mature in order to avoid the risk of permanent damage to the growing elbow.

Back. Back pain in a child or adolescent athlete is often due to a combination of overstress in a particular sport as well as the vulnerability of the low back during the growth spurt.[8] Because of enhanced growth anteriorly in the bodies of the vertebrae with posterior tethering by the lumbosacral fascia, during this time there is a tendency for hyperlordosis in the lumbar spine. A compensatory hip flexion contracture, tightness of the hamstring structures, and a dorsal kyphosis accompany this. This has the effect of increasing the stress on the posterior elements and the disc spaces of the spine. When this happens in combination with overuse (often in diving, gymnastics, or weight lifting), the individual may become symptomatic. Three problems commonly seen in the lumbar spines of young athletes are stress fractures of the pars interarticularis (spondylolysis), hyperlordodic mechanical low back pain syndromes, and disc herniation.[8] Conservative measures which initially emphasize a period of rest are appropriate for the second of these. Subsequently an appropriate training program is in order to strengthen the anterior or abdominal structures and adequately stretch out the posterior structures. One must be suspicious of a stress fracture in the individual with persistent low back pain. Appropriate x-ray studies, including oblique films of the lumbosacral spine and a bone scan if needed, will demonstrate a stress fracture of the pars interarticularis. If diagnosed early, one may anticipate healing of the stress fracture. An antilordodic brace may be very helpful for these individuals both to reduce their pain in the initial treatment period, and subsequently as a device to permit them to participate in sports.

Knee. The knee is the joint most commonly injured in sports participation. Macrotrauma may result either in separation of the epiphysis, dislocation of the patella, or disruption of a ligament. Serious ligament disruptions may occur despite the open growth plate. Any significant injury to the knee should be evaluated by an orthopedist. Overuse syndromes of the knee are quite common and often result from a malalignment of the patellofemoral mechanism. Individuals with serious malalignments may suffer from recurrent dislocations and/or subluxations of the patella. Anterior knee pain associated with stressful sports is more likely to develop in individuals with so-called malignant malalignment of increased femoral anteversion, infacing patellae, and compensatory external rotation of the tibia with the resultant high Q angle. Appropriate treatment includes adequate rest, quadriceps rehabilitation, bracing, and, when needed, modification of the individual's sports participation. Surgery may have a role in the treatment of serious malalignments. However, the majority of young athletes with anterior knee pain can be treated adequately with conservative methods. A period of conservative care of not less than six months to one year is indicated before surgery is contemplated.

Osgood-Schlatter's tenderness at the tibial tubercle, which is the attachment site of the infrapatella ligament, is also common among adolescents.[12] This is also often a result of overuse and responds well to appropriate conservative care.

Leg and Ankle. The lower leg is also prone to overuse syndromes such as shin splints and stress fractures.[11] Treatment should include not only rest and better flexibility training for the posterior leg musculature, but also appropriate orthotic balancing of the feet if hyperpronation is present.

With regard to the foot and ankle, the most common acute injury is a lateral ligament sprain of the ankle. Almost all of these can be treated in a conservative fashion, with an initial period of ice packs, compressive dressing, and elimination of weight bearing, followed by a progressive period of rehabilitation which stresses peroneal muscle strengthening. Prophylactic strapping during the initial phases of healing facilitates an early return to play. Achilles tendinitis in the older individual or apophysitis in the young individual is a common overuse syndrome made worse during the growth spurt, due to the combination of overuse and excessive tightness of the posterior structures. Treatment includes rest, ice massage, a heel lift for symptomatic relief, and an aggressive stretching program. In the individual with unexplained mid-foot pain associated with overuse, one must be aware of the possibility of stress fractures of the tarsal navicular, the diagnosis of which very often requires a bone scan. Immobilization and elimination of weight bearing are essential in the treatment of this. A large separation between the two fragments may sometimes require surgical treatment.

REFERENCES

1. Garrick JG, Requa RK: Injuries in high school sports. *Pediatrics* 1978; 61:465-469.
2. Martens R: Youth sport in the USA, in Weiss MR, Gould D (eds): *Sport for Children and Youths*, vol 10. Champaign, Ill, Human Kinetics Pubs, 1984, pp 27-33.
3. Goldberg B, Saraniti A, Witman P, et al: Pre-participation sports assessment—an objective evaluation. *Pediatrics* 1980; 66:736-745.
4. Harvey J: The preparticipation examination of the child athlete. *Clin Sports Med* 1982; 1:353-369.
5. DeHaven KE: Athletic injuries in adolescents. *Pediatr Ann* 1978; 7:704-714.
6. Linder CW, DuRant RH, Seklecki RM, et al: Preparticipation health screening of young athletes. Results of 1268 examinations. *Am J Sports Med* 1981; 9:187-193.
7. Micheli LJ: Pediatric and adolescent sports injuries: Recent trends, in Pandolf KB (ed): *Exercise and Sports Sciences Reviews*, vol 14. New York, Macmillan Publishing Co, 1986, pp 359-374.
8. Micheli LJ: Low back pain in the adolescent: Differential diagnosis. *Am J Sports Med* 1979; 7:362-364.
9. Singer K: Injuries and disorders of the epiphyses in young athletes, in Weiss MR, Gould D (eds): *Sport for Children and Youths*, vol 10. Champaign, Ill, Human Kinetic Pubs, 1984, pp 141-150.
10. Torg JS: *Athletic Injuries to the Head, Neck and Face*. Philadelphia, Lea & Febiger, 1982, pp 17-20.
11. Adams JE: Bone injuries in very young athletes. *Clinic Orthop* 1968; 58:129-140.
12. Harvey JS: Overuse syndromes in young athletes, in Weiss MR, Gould D (eds): *Sport for Children and Youths*, vol 10. Champaign, Ill, Human Kinetics Pubs, 1984, pp 151-163.

FROM THE LIBRARY

THE SCHOOL PHYSICIAN AND THE CARE OF THE ATHLETE

The policy of the school physician in respect to the care of athletes varies as to locality and allotted time, as to his own hobbies and interests, and as to the attitude of the coach with whom he is working. There is also a variance of opinion as to the allocation of duties to the physical educator, coach, or physician. In some instances one makes up for the shortcomings of the other to supply a fairly adequate care, but in a few instances perhaps both shirk that duty with a resulting sad fortune for the athlete. . .

As the physical examination is the basis of the entire health service program, so does it assume the position of vital importance in the care of athletes. In those instances where the family physician feels that a youth should not participate in athletics, his opinion is always to be respected and followed. In those instances, to the contrary, in which the examination at school reveals physical disability and the family physician feels the youth fit for sports, I feel we should remember that if that youth does participate to his detriment the responsibility for such usually falls upon the school physician.

There is a great variance of opinion as to the frequency with which physical examinations should be performed. In the Report of the Regent's Inquiry of the School Health Program, the opinion is expressed that "a series of three thorough physical examinations in the life of the school child (supplied by the school) should be adequate even for athletes if supplemented by awareness on the part of physical educators, teachers, and parents of the need of referral of children developing abnormalities between routine examinations." Perhaps some of you follow this system and feel that your personnel is keen enough in their observations to use this referral system satisfactorily with such a lapse between examinations. Rather than allow defects to reach the symptom-producing stage, I feel that by more frequent examination these may be found and corrected or proper therapy instituted before irreparable damage is done. . . .

ROBERT H. BROAD, MD
(*NY State J Med* 1940; 40:1603-1606)

Cardiac evaluation of school sports participants*

NORMAN B. SCHELL, MD, MARJORIE L. KING, MD

Most students who apply for athletic activities and team sports in junior and senior high schools have a normal cardiovascular system. Nevertheless, there is a potential for cardiac morbidity and mortality during and after participation in these events among a small number of students with cardiac risk factors. Since 1977, the School Health and Medical Aspects of Sports committees[1,2] (now the Committee on School Health and Sports Medicine) of the Medical Society of the State of New York (MSSNY) have been concerned with cardiac evaluation procedures for school sports participants throughout the state school system.

The State Education Department in New York requires "adequate health examinations before participation in strenuous activity and periodically throughout the season as necessary and to permit no pupil to participate in such activity without the approval of the school medical officer." [3] The following policies have been adopted in New York State to implement this regulation[4]:

1. Medical examinations may be scheduled at any time during the school year, and the results of the annual examination shall be valid for a period of 12 continuous months except as noted in (3) below.
2. Before participation in each sports season, the school nurse or nurse-teacher should review the pupil's attendance records and interview the pupil to update his or her health history.
3. Any pupil whose safe participation is questionable as a result of the health history interview, an injury during the current school year, or prolonged absence during the current year must be requalified by the physician prior to athletic participation.
4. The school physician has the final authority to determine the physical capability of a student to participate in the chosen sport.

The annual qualifying examination is usually performed by the school physician (or personal physician in some areas of the state), who reviews the past and interim histories of each applicant. In essence, the prospective athlete is screened for conditions that may predispose him or her to injury or death resultant from the exertion of the sports activity. If an abnormality or risk factor is detected and is confirmed by an examination performed out of the school, it is the school physician's responsibility either to allow or to exclude participation in specific categories of sports activities. These activities can be classified by their intensity levels and static versus dynamic demand levels.[5]

CARDIAC SCREENING

In 1985, the newly organized Committee on School Health and Sports Medicine of the MSSNY reviewed and updated the key physical findings of cardiac screening by the school physician, which were published in 1978[1] (Table 1). The primary abnormalities of concern in the cardiac examination are heart murmurs, arrhythmias, and elevated blood pressure.

In the presence of any of the five key physical findings (Table I), the prospective athlete should be referred to a pediatric cardiologist for further cardiac evaluation. After clinical examination, the cardiologist may recommend physiologic and/or radiologic tests to help determine the

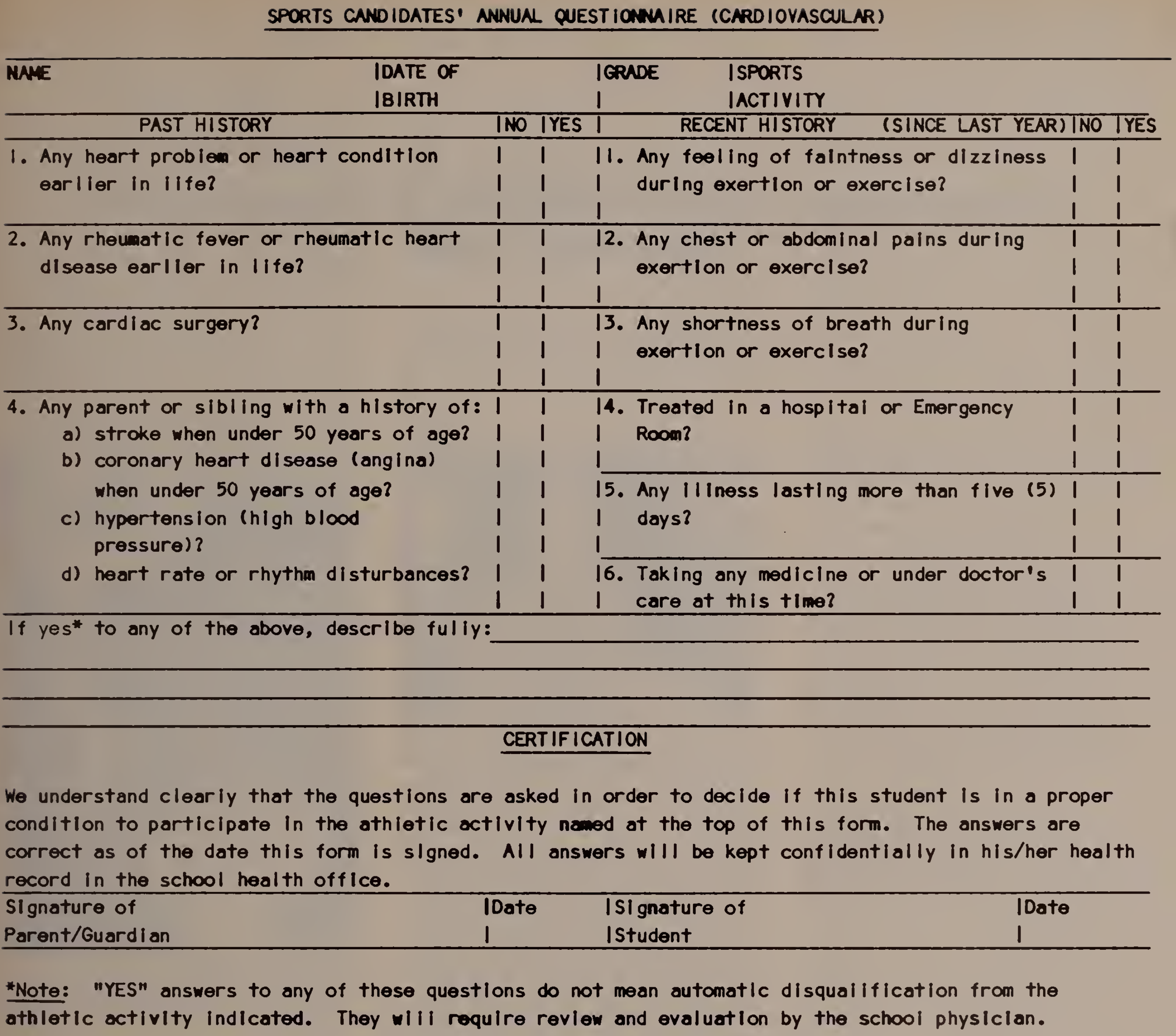
TABLE I. Cardiac Screening of Sports Applicants by School Physician

Key Physical Findings Requiring Further Evaluation
1. Heart rate over 120 per minute. This should be confirmed on a second occasion 15 minutes or more after resting.
2. Multiple extrasystoles or arrhythmias. Check after jumping or hopping 20 times to ascertain if they disappear.
3. Resting blood pressure of 130/86 mm Hg or higher for students under 12 years of age; 140/90 mm Hg or higher for those 13 years of age and older.[6] At least three separate occasions in a friendly environment are necessary for corroboration. Take the average of these three or more readings. a. A high reading on one occasion for an adolescent is not diagnostic of hypertension. b. For validity, be certain that the pressure cuff covers at least two thirds of the upper arm, from elbow to shoulder. (Adult cuff = 30 × 13 cm; pediatric cuff = 22 × 10 cm; obesity cuff = 39 × 15 cm).
4. All systolic murmurs grade 3/6 or louder at any location; all diastolic murmurs or mid-systolic clicks at any location; any continuous murmur, hum, or rub.
5. Absence of femoral or brachial pulses or large discrepancy existing between them.

From the Committee on School Health and Sports Medicine of the Medical Society of the State of New York, Lake Success, NY; the Nassau County Department of Health (Dr Schell), Mineola, NY; and the Department of Medicine, Helen Hayes Hospital (Dr King), West Haverstraw, NY.

Address correspondence to Dr Schell, Deputy Commissioner, Nassau County Department of Health, 240 Old Country Rd, Mineola, NY 11501.

* Guidelines approved by the Council of the Medical Society of the State of New York on December 19, 1985.



SPORTS CANDIDATES' ANNUAL QUESTIONNAIRE (CARDIOVASCULAR)

NAME	DATE OF BIRTH	GRADE	SPORTS ACTIVITY

PAST HISTORY	NO	YES	RECENT HISTORY (SINCE LAST YEAR)	NO	YES
1. Any heart problem or heart condition earlier in life?			11. Any feeling of faintness or dizziness during exertion or exercise?		
2. Any rheumatic fever or rheumatic heart disease earlier in life?			12. Any chest or abdominal pains during exertion or exercise?		
3. Any cardiac surgery?			13. Any shortness of breath during exertion or exercise?		
4. Any parent or sibling with a history of: a) stroke when under 50 years of age?			14. Treated in a hospital or Emergency Room?		
b) coronary heart disease (angina) when under 50 years of age?			15. Any illness lasting more than five (5) days?		
c) hypertension (high blood pressure)?					
d) heart rate or rhythm disturbances?			16. Taking any medicine or under doctor's care at this time?		

If yes* to any of the above, describe fully:____________________

CERTIFICATION

We understand clearly that the questions are asked in order to decide if this student is in a proper condition to participate in the athletic activity named at the top of this form. The answers are correct as of the date this form is signed. All answers will be kept confidentially in his/her health record in the school health office.

Signature of Parent/Guardian	Date	Signature of Student	Date

*Note: "YES" answers to any of these questions do not mean automatic disqualification from the athletic activity indicated. They will require review and evaluation by the school physician.

FIGURE 1. Sports Candidates' Questionnaire to be completed by prospective student participant and parent.

level of permissible sports participation. The cardiologist should then prescribe (in writing) the allowed and disallowed sports activities in school.

SPORTS CANDIDATES' QUESTIONNAIRE

In addition, the new committee, with the advice of a subcommittee composed of authorities in cardiology, also reviewed the updated Sports Candidates' Questionnaire, which was published in 1981.[2] As redesigned, the questions focus on key cardiovascular historical events—past and interim (Fig 1).

A positive answer to any of these ten questions should cause the school physician to advise a more thorough cardiac examination by a personal physician in the community. Previous illness with diseases such as rheumatic fever should also encourage the school physician to review recent electrocardiograms and chest films.

The MSSNY urges all school districts in the State of New York to adopt these guidelines by incorporating them into existing examinations for sports candidates.

Acknowledgment. The authors thank Filippo A. Balboni, MD, Director of Pediatric Cardiology, St Francis Hospital, Roslyn, NY, for his advice and assistance in the preparation of this manuscript.

REFERENCES

1. Schell NB: Cardiac evaluation of school sports participants. *NY State J Med* 1978; 78:942-943.
2. Schell NB: Sports candidates' questionnaire. *NY State J Med* 1981; 1548-1549.
3. Regulations of Commission of Education, New York State, 135.4(c)(7)(i)(h).
4. Memorandum of Assistant Commissioner for School Services, Education Department of New York State, 1978.
5. Mitchell JH, Blomqvist CG, Haskell WL, et al: Classification of sports. *JACC* 1985; 6:1198-1199.
6. The Report of the Second Task Force on Control of Blood Pressure in Children. *Pediatrics* 1987 (in press).

Diaphragmatic hernia mimicking pyopneumothorax and colonic mass

Robert G. Sperber, md

In cases of indirect trauma, diaphragmatic hernia is a condition not readily considered. Indeed, two previous studies have shown that this disorder is correctly diagnosed in fewer than 50% of cases, although the ambiguous nature of the patients' complaints often leads to the use of many diagnostic tests.[1,2] Unless clinical suspicion of such herniation is pursued, the patients' symptoms and initial radiographic studies will often support diagnoses of either colonic mass or intrathoracic disease.

Case Report

A 48-year-old man came to the emergency room after pushing a disabled car. He reported about five episodes of blood-tinged vomiting and intermittent lower abdominal pain. He had had gastric surgery for peptic ulcer disease in 1972.

The physical examination was normal except for a positive stool guaiac test. The chest radiograph disclosed no abnormalities, except for localized tenting of the left hemidiaphragm. Erect abdominal radiographs showed loops of bowel with air-fluid levels in the left upper abdomen.

One day after admission, a left pleural effusion developed with mediastinal shift. The patient was febrile (101°F) and had a pulse rate of 120 beats/min. The arterial Po_2 was 71 mm Hg. Thoracentesis was performed, and 2,050 cc of sanguinous fluid was removed.

During the next three days, the patient's condition remained stable, except for one episode of rectal bleeding, increasing distention of the bowel, and chest pain. Chest radiographs demonstrated recurrent left pleural effusion and apparent elevation of the left hemidiaphragm, with several air-fluid levels beneath it (Fig 1). A computed tomographic (CT) scan of the thorax demonstrated a large left pleural effusion ranging from 25-60 HU and containing air collections, consistent with pyopneumothorax or hemopneumothorax (Fig 2).

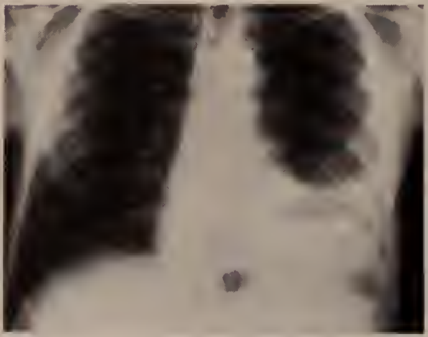


FIGURE 1. Posteroanterior radiograph of the chest showing apparent elevation of the left diaphragm with several prominent air-fluid levels beneath it.

On the sixth hospital day, proctosigmoidoscopy was performed; no mucosal lesions were discovered. A barium enema demonstrated a constricting lesion of the colon in the region of the splenic flexure (Fig 3). This finding led to the suspicion of a colonic mass, and surgery was performed. At surgery, there was found to be a diaphragmatic defect in the posterolateral aspect of the left hemidiaphragm measuring 5-6 cm. The entire transverse colon and part of the splenic flexure and greater omentum had protruded through this defect. The bowel was gangrenous and matted together. Additionally, 200 cc of sanguinous fluid was removed from the pleural cavity. There was no evidence of a lung abscess or empyema. Atelectasis was present at the base of the left lung. The strangulated bowel was resected, and the diaphragmatic tear was repaired. After surgery, a loculated left pleural effusion developed, then pneumothorax, which were treated successfully.

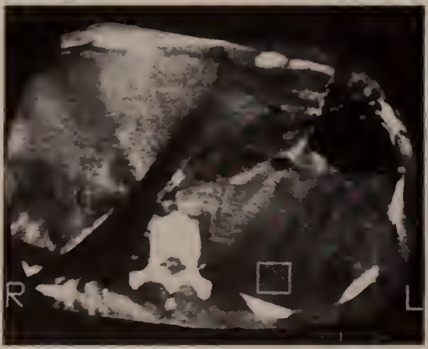


FIGURE 2. CT scan of the chest showing multiple collections of air in anterior left hemithorax with a nonhomogeneous pleural collection posteriorly. Left diaphragmatic crus (arrow) is noted, but the diaphragmatic contour is not visualized.

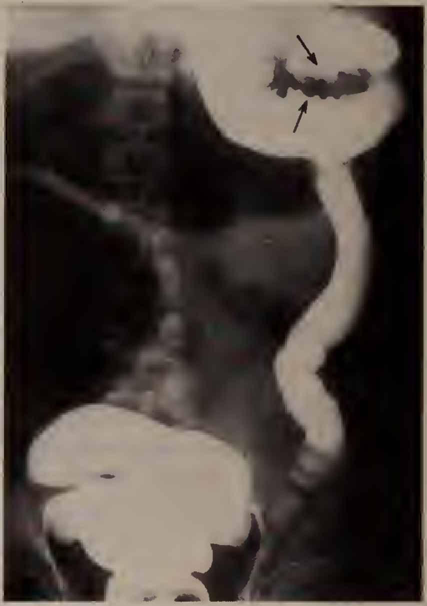


FIGURE 3. Barium enema demonstrating a 2-cm filling defect (arrow) in the splenic flexure of the colon as it passes through the left diaphragmatic defect.

Discussion

Diaphragmatic tears usually develop following crushing injuries. Various portions of the gut may protrude through such an opening and may become twisted or strangulated. Herniations may take place immediately after

---

From the Radiology Department, The Bronx-Lebanon Hospital Center, The Bronx, NY.

Address correspondence to Dr Sperber, 29 Edgewood Pl, Great Neck, NY 11024.

the injury or some time later. Ebert et al[3] mention that approximately 98% of ruptures occur on the left side.

This patient most probably had sustained a diaphragmatic rupture in the remote past which was exacerbated by his pushing a car. Since the trauma was indirect, it was disregarded. By the second hospital day, the bowel had protruded into the chest, producing hemothorax which was believed to be due to intrathoracic disease. The patient's vague abdominal symptoms of pain and blood-tinged vomiting were first thought to be due to pancreatitis or diverticulitis. In retrospect, these findings were probably related to diaphragmatic irritation and early herniation with subsequent bowel compression.[4]

Chest films demonstrated an abnormality at the base of the left lung, and the CT scan showed multiple air-fluid levels and an inhomogeneous left pleural collection which were misinterpreted as loculated pyopneumothoraces or hemopneumothoraces.

Interpretation was further complicated by a barium enema which demonstrated partial obstruction at the site where the colon passed through the diaphragm. This was misdiagnosed as a colonic tumor.

Radiologic definition of the level of the diaphragm can present a major diagnostic problem. In the presence of diaphragmatic herniation, plain films are generally abnormal, but the findings are often nonspecific.[5] The herniated bowel may flatten out beneath the lung and simulate an elevated diaphragm. When an air-filled bowel is present in the hemithorax along with pleural effusion, the appearance may mimic pleural tumors, lung abscesses, or cysts. Other diagnostic considerations include eventration, atelectasis, subphrenic collections, and phrenic nerve palsy.

The advantage of CT scans over conventional radiographs is the former's ability to give cross-sectional representation of the diaphragm and adjacent structures.[5] The normal hemidiaphragm may be either directly or indirectly identified on CT scans depending on the juxtaposition of abdominal fat, lung, and diaphragm.[6] Absence of a portion of the diaphragmatic contour in the area of a tear, and identification of omentum and contrast-filled loops of bowel lateral or posterior to the diaphragmatic contour may indicate herniation of abdominal contents into the chest.[5] However, when the diaphragmatic contour is obscured by pleural blood or pus, as in this patient, it is not possible to determine whether bowel loops are in the chest or the abdomen. Similarly, localization of an effusion or abscess to the subphrenic or pleural space is difficult when the position of the hemidiaphragm cannot be identified.[6]

Ultrasonography can be used to visualize the diaphragm and is helpful in determining the location of peridiaphragmatic fluid collections. Although the left side is more difficult to evaluate because of bowel gas, a portion of the left peridiaphragmatic region can be seen through the spleen.[7]

Only direct coronal CT scanning or peritoneography (with gas or iodinated contrast medium) enables visualization of the diaphragm comparable to that achieved with sonography.[7] Gallium or labeled-leukocyte scans are often utilized in the evaluation of infected peridiaphragmatic fluid collections. The nuclear liver/spleen scan is an important tool in the diagnosis of liver or spleen herniation across the diaphragm.[8]

This case illustrates the importance of rapid diagnosis to avoid further morbidity. Had there been some discussion of diaphragmatic involvement, the correlation of diagnostic studies might have led to an earlier diagnosis. If the abnormality at the base of the left lung had been considered with the partially obstructed colon and the obscured diaphragm seen on CT scan, the diagnosis of diaphragmatic hernia with incarceration might have been more readily made.

REFERENCES

1. Graivier L, Freeark RJ: Traumatic diaphragmatic hernia. *Arch Surg* 1963; 86:363–373.
2. Gourin A, Garzon AA: Diagnostic problems in traumatic diaphragmatic hernia. *J Trauma* 1974; 14:20–31.
3. Ebert PA, Gaertner RA, Zuidema GC: Traumatic diaphragmatic hernia. *Surg Gynec Obstet* 1967; 125:59–65.
4. Harvey A, Bordley J: Differential diagnosis, ed 2. Philadelphia, WB Saunders Co, 1970, p 334.
5. Heiberg E, Wolverson MK, Hurd RN, et al: CT recognition of traumatic rupture of the diaphragm. *AJR* 1980; 135:369–372.
6. Alexander ES, Proto A, Clark RA: CT differentiation of subphrenic abscess and pleural effusion. *AJR* 1983; 140:47–51.
7. Tarver RO, Goodwin JD, Potman CE: Symposium on nonpulmonary aspects in chest radiology. The diaphragm. *Radiol Clin North Am* 1984; 22:615–631.
8. Ball T, McCrory R, Smith JO, et al: Traumatic diaphragmatic hernia; errors in diagnosis. *AJR* 1982; 138:633–637.

See also pp 87–88.

Tuberculosis of the knee presenting as internal derangement

BURTON L. BERSON, MD; DOV J. BERKOWITZ, MD; MARVIN S. GILBERT, MD; MILTON C. ENGEL, MD;
HANS E. SCHAPIRA, MD

The recent orthopedic literature has shown skeletal and intra-articular tuberculosis to be a major diagnostic problem, with a confusing clinical picture that can delay diagnosis and management.[1] Skeletal involvement occurs in approximately 1% of the population with active tuberculosis.[2] We report a case of extrapulmonary tuberculosis in two sites—the knee and the kidney—that presented as mechanical derangement of the knee.

CASE REPORT

The patient was a 27-year-old, American-born, Oriental man who had lived in Seoul, Korea, between the ages of two and five years. His medical history was unremarkable, his family history was negative for tuberculosis, and his occupational history was noncontributory. The patient first complained of pain in the left knee in January

---

From the Departments of Orthopedics (Drs Berson, Berkowitz, Gilbert), Medicine (Dr Engel), and Urology (Dr Schapira), Mount Sinai School of Medicine and Mount Sinai Hospital, New York.

Address correspondence to Dr Berson, 1100 Park Ave, New York, NY 10028.

1982. There was no history of trauma or arthritis, and he participated in recreational athletics. He noted difficulty in going up and down stairs and getting in and out of chairs, and he noted pain and stiffness after prolonged sitting. He was unsuccessfully treated with nonsteroidal anti-inflammatory medication by his local physician.

In July 1982, because of persistent symptoms, he was referred for orthopedic evaluation. Further questioning prompted the patient to complain of intermittent swelling with questionable locking and clicking. There was no buckling of the left knee. The patient reported no temperature elevation, night sweats, or weight loss.

Physical examination of the left knee revealed minimal effusion, medial joint tenderness, patellofemoral crepitus, and limitation of full extension and flexion with a positive anterior drawer sign and significant atrophy of the quadriceps. Routine roentgenograms were unremarkable. An arthrogram revealed a tear of the body of the medial meniscus and a normal lateral meniscus, and only faintly outlined the cruciate ligaments. On this basis, the patient was scheduled for arthroscopic evaluation and surgery.

On August 8, 1982, the patient was admitted to Mount Sinai Hospital for a preoperative work-up. The hemoglobin count was 14.6 g/dL; hematocrit, 42.8%; white blood cell count, $5,000/\text{mm}^3$ with 60% segmented band forms, 35% lymphocytes, 4% monocytes, and 1% band forms. A subsequent erythrocyte sedimentation rate was 5 mm/h. Urinalysis showed a specific gravity of 1.015. There were zero to one red blood cells and many white blood cells on microscopic evaluation. A repeat urinalysis showed 14 to 15 white blood cells per high powered field. A routine culture of the urine revealed the urine to be sterile, with less than 1,000 bacteria/cc. The levels of SMA 6 and 12 were within normal limits.

Examination of the left knee under anesthesia revealed 2+ anterior laxity with a weakly positive pivot shift. At the time of arthroscopic examination, a significant amount of blood-tinged synovial fluid was aspirated. Grade II to III patellar and femoral chondromalacia and marked synovitis in the suprapatellar pouch were also noted. A transverse-horizontal type of complex tear of the medial meniscus posterior horn and body, as well as marked synovitis and fibrillation of the medial femoral condyle and tibial articular surface, were present in the medial compartment. In the intercondylar notch, marked synovitis with no functional anterior cruciate ligament was found. The lateral meniscus had a transverse tear of the posterior horn just in front of the popliteal tendon as well as synovitis of the lateral compartment. The synovial proliferation was especially prominent at the peripheral meniscosynovial junction. A limited synovectomy was performed using a motorized intra-articular shaver, along with partial medial and lateral meniscectomies.

The patient was discharged the following morning. The pathology report described fragments of synovium with chronic inflammation and multiple epitheliod cell granulomas with focal central necrosis (Fig 1). Stains for acid-fast bacilli and fungi were negative.

Ten days after surgery, the patient was reexamined and was found to have a slight effusion of the knee that was operated on. The stab wound incisions made at the time of surgery were healing well. Because of the abnormal pathology report, the patient was referred to an internist for further evaluation.

Physical examination disclosed no abnormalities except for the slight effusion of the left knee. The chest roentgenogram was normal except for minimal hilar calcifications. Urinalysis revealed persistent pyuria. The tuberculin skin test with 5 tuberculin units was positive and measured 17 mm of erythema and induration. Three urine specimens were submitted for *Mycobacterium tuberculosis* culture. The left knee joint was aspirated, and the fluid was submitted for culture of *M tuberculosis*. Because the index of suspicion was high for multiple sites of extrapulmonary tuberculosis—specifically the left knee joint and one or both kidneys—the patient was started on isoniazid, ethambutol, and rifampin, which was the recommended treatment at that time, especially for genitourinary tuberculosis. Subsequent culture reports showed both the synovial fluid and urine to be positive for *M tuberculosis*.

Urologic evaluation included an intravenous pyelogram, which showed nonvisualization of the right kidney. A renal scintigraphic study using Tc 99m revealed impaired right renal function and abnormal right renal parenchyma consistent with obstruction of the outflow tract. The left kidney and outflow tract were normal. Approximately four weeks after antituberculosis therapy was started, cystoscopy and a right retrograde pyelogram were performed; they revealed inflammatory changes in the bladder and prostatic urethra and a rigid-looking right ureteral orifice. Retrograde studies revealed multiple areas of stricture and beading of the right ureter, mainly in the lower third, with associated hydronephrosis of the right collecting system.

During the ensuing months, the patient's knee became asymptomatic and normal in appearance. There was a full range of motion, no swelling, no atrophy of the quadriceps, and 2+ anterior laxity. He had been on an isokinetic exercise program. Subsequent urine cultures for acid-fast bacilli were negative. The patient's therapy was discontinued after 18 months, and he has maintained good health without evidence of recurrent disease.

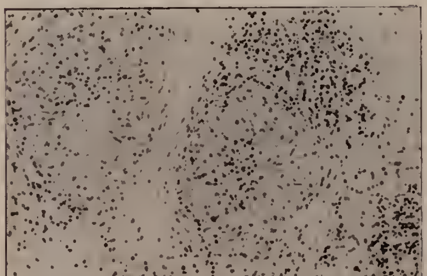


FIGURE 1. Synovial granuloma with epithelioid cells and focal central necrosis.

DISCUSSION

The prevalence of tuberculosis infection in the native Korean population in the 1950s was about 50%, with the incidence of tuberculosis disease being much lower. At least 50% of the children in Korea had a positive tuberculin test. This could have been due in part to the ravages of the Korean War (1950–1953) and postwar destitution. The patient described here, although American born, lived in Korea from 1956–1959, from age two to age five. There is a strong likelihood that he contracted pulmonary tuberculosis at that time. While the pulmonary infection healed spontaneously, the infections in the left knee and right kidney were a result of hematogenous dissemination of the *Mycobacterium tuberculosis* bacilli. The diagnosis was made only on the basis of the fortuitous partial synovectomy, which was performed to better visualize the meniscus.

The etiology of meniscal tears in this patient is of interest. There was no clear-cut history of trauma. Arthroscopy revealed an absent anterior cruciate ligament. In knees that lack the anterior cruciate ligament, the potential for subsequent meniscal damage is markedly increased. Warren and Marshall[3] reported a 60% incidence of meniscal damage in chronic anterior cruciate ligament insufficiency. Recent injection studies by Arnoczsky and Warren[4] demonstrated a capillary network that entered through the peripheral rim and penetrated 10–30% of the medial meniscus. Their study provides an anatomic basis for hematogenous dissemination of the mycobacterium to the meniscosynovial junction of the knee. Although not initially examined, the meniscal specimen was subsequently reviewed by both a pathologist and the authors and was found to be free of granulomas.

Of note in this patient was the marked synovitis. We may speculate that in the absence of trauma, the chronic tuberculous synovitis might be the cause of the anterior cruciate ligament deterioration and meniscal tears. The vascular invasion and hyperemia of the synovium surrounding the anterior cruciate ligament may have led to its gradual attrition. In a like manner, it is not inconceivable that enzymatic degeneration, which weakened the molecular structure of the menisci enough to allow tearing without significant trauma, may have been another consequence of the synovitis.

There have been recent reports of unrecognized tuberculous arthritis in the medical literature.[5-7] In most cases, the delay in diagnosis led to increased patient morbidity.[6] The most common error responsible for a delayed diagnosis seems to be a low index of suspicion, as was the case in this patient.

Tuberculosis must be remembered as a systemic disease. Complications associated with tuberculosis may occur soon after the onset of the disease or may be latent for months or years without producing clinical evidence of the disease.

Like syphilis, tuberculosis may remain dormant throughout the life of the individual. This diversity of the disease and its clinical manifestations makes tuberculosis one of the most interesting diseases that affects mankind, and one that must always be considered in the differential diagnosis.[8]

REFERENCES

1. Stanish W, Hyndman J, Forsythe M: Skeletal tuberculosis: The great imitator. *J Bone Joint Surg* [*Br*] 1977; 59:511.
2. Davidson PT, Horowitz I: Skeletal tuberculosis. A review with patient presentations and discussion. *Am J Med* 1970; 48:77-84.
3. Warren RF, Marshall JL: Injuries of the anterior cruciate and medial collateral ligaments of the knee. A retrospective analysis of clinical records—Part 1. *Clin Orthop* 1978; 136:191-197.
4. Arnoczky SP, Warren RF: Microvasculature of the human meniscus. *Am J Sports Med* 1982; 10:90-95.
5. Besser MI: Total knee replacement in unsuspected tuberculosis of the joint. *Br Med J* 1980; 280:1434.
6. Chow SP, Yau A: Tuberculosis of the knee—long term follow-up. *Int Orthop* 1980; 4:87-92.
7. Hecht RH, Meyers MH, Thornhill-Joynes M, et al: Reactivation of the tuberculosis following total joint replacement. A case report. *J Bone Joint Surg* [*Am*] 1983; 65:1015-1016.
8. Lincoln EM, Sewell EM: Tuberculosis in children. New York, McGraw-Hill Book Co, Inc, 1963.

See also pp 88–89.

Electrocardiographic anterior wall pseudoinfarction pattern caused by a metastatic brain tumor

RICK CONETTA, MD, BERNARD GITLER, MD

Electrocardiographic pseudoinfarction patterns are those which mimic myocardial ischemia or infarction in the absence of underlying coronary artery disease. They can occur in a variety of clinical settings.[1-4] Central nervous system diseases have also been associated with electrocardiographic abnormalities which simulate ischemia.[5-12] Concurrent coronary pathology need not be present.

We report the case of a patient who had neurologic symptoms caused by a metastatic cerebral lesion, and whose electrocardiographic reading suggested anterior wall myocardial infarction. Treatment of the neurologic problem led to reversal of the electrocardiographic abnormalities.

CASE REPORT

A 78-year-old female came to the emergency room after she noted right-sided facial twitching. One day previously she had experienced a self-limited episode of involuntary movements of the right hand. She had no syncope or generalized seizure activity, and there was no prior history of neurologic disease. Five years earlier she had undergone a modified right radical mastectomy for carcinoma. At that time regional lymph nodes were negative for metastatic disease.

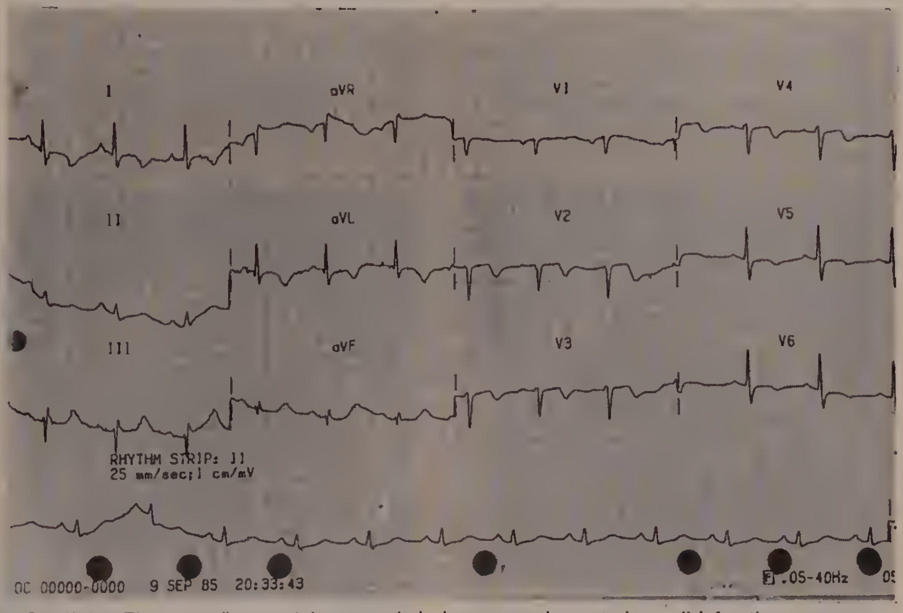


FIGURE 1. Electrocardiogram taken on admission suggesting anterior wall infarction.

In addition to the patient's right mastectomy scar, physical examination was remarkable for a mild palsy of the right central facial nerve and a mild right-sided hemiparesis. Results of a complete blood count and blood chemistries were normal. An electrocardiogram (Fig 1) demonstrated poor precordial R wave progression, slight

---

From the Department of Medicine (Dr Conetta) and the Division of Cardiology (Dr Gitler), New Rochelle Hospital Medical Center, New Rochelle, NY.

Address correspondence to Dr Conetta, Department of Medicine, New Rochelle Hospital Medical Center, 16 Guion Pl, New Rochelle, NY 10802.

ST segment elevation in leads V_2 through V_4, and inverted T waves in leads I, aV_L, and V_2 through V_6. These findings were new compared to her previous tracings and were felt to be consistent with anterior wall myocardial infarction. A chest roentgenogram showed a normal cardiac silhouette and a new small nodule in the apex of the left lung. Computerized axial tomography of the brain demonstrated a mass in the left frontal lobe with surrounding edema (Fig 2).

A diagnosis of recurrent carcinoma of the breast, metastatic to the brain and lung, was made. The patient was treated with radiation therapy to the brain, corticosteroids, and anticonvulsants. Within two weeks the neurologic symptoms disappeared. The electrocardiographic pattern suggestive of anterior wall infarction also resolved. A follow-up tracing 24 days after admission showed minimal nonspecific ST-T wave changes (Fig 3). Repeat computerized axial tomography of the brain, 31 days after the initial study, demonstrated almost complete resolution of the left frontal lesion (Fig 4).

Myocardial infarction was effectively ruled out by a finding of normal serial cardiac enzymes and a negative technetium pyrophosphate scan performed one day after admission. A nuclear ventricular function study revealed normal wall motion with a left ventricular ejection fraction of 59%. A two-dimensional and M-mode echocardiogram demonstrated mitral anular calcification and normal ventricular contractions. Percutaneous needle biopsy of the pulmonary nodule yielded malignant epithelial cells similar in appearance to those seen in the breast carcinoma specimens removed five years earlier, thus making it likely that the central lesion was of the same histology.

The patient was discharged with no neurologic symptoms after 31 days of hospitalization.

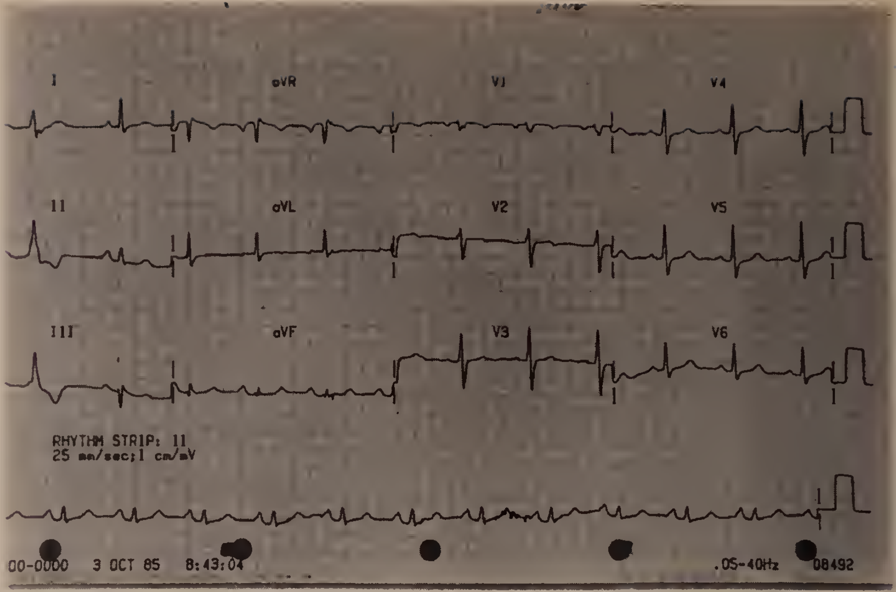


FIGURE 3. Electrocardiogram obtained 24 days after admission showing resolution of infarction pattern.

Discussion

Numerous pathologic conditions have been associated with electrocardiographic findings suggestive of myocardial ischemia and infarction in the absence of underlying coronary artery disease (these have been called pseudoinfarction patterns). Well-known examples include left ventricular hypertrophy, right ventricular hypertrophy, left bundle branch block, left anterior fascicular block, pericarditis, idiopathic hypertrophic subaortic stenosis, and Wolff-Parkinson-White syndrome.[1,2] Pericardial effusions[3] and hyperkalemia[4] have also been associated with pseudoinfarction patterns.

Electrocardiographic changes that mimic ischemia in relation to cerebral lesions have also been well described. In 1947, Byer et al[5] were the first to note electrocardiographic abnormalities associated with a subarachnoid hemorrhage. These consisted of large upright T waves and prolongation of the QT interval. In 1953 Levine[6] showed conclusively that electrocardiographic abnormalities could accompany a cerebral lesion in the absence of underlying cardiac disease. He reported the case of a 69-year-old female with a subarachnoid hemorrhage; an electrocardiogram showed diffuse T wave inversion. At autopsy no evidence of necrosis or any other cardiac abnormality could be found. Similar observations by others followed.[7,8] It became clear that, in addition to subarachnoid hemorrhage, a variety of other cerebral lesions were associated with electrocardiographic anomalies. These included intracerebral hemorrhage,[9] head trauma,[10] intracranial space-occupying lesions,[11,12] and infections.[12] Neurosurgical procedures have also resulted in electrocardiographic changes.[13]

Hugenholtz[11] was the first to note electrocardiographic alterations in relation to a cerebral tumor. These consisted of deeply inverted T waves in a patient with suspected cerebral metastases. In contrast to our report, the electrocardiographic changes noted by Hugenholtz were not typical for myocardial infarction and the presence of a

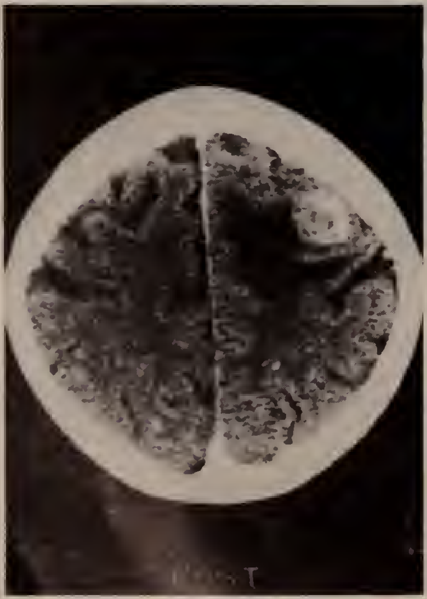


FIGURE 2. Computerized axial tomographic (CAT) scan of the brain taken on admission demonstrating left frontal mass with surrounding edema.

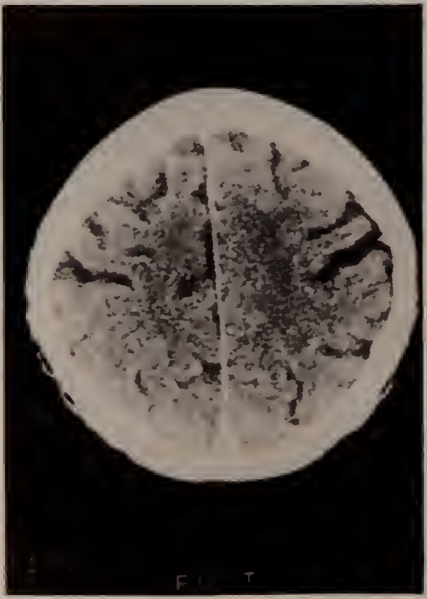


FIGURE 4. Repeat CAT scan of the brain performed 31 days after the initial study, demonstrating almost complete resolution of the left frontal lesion.

cerebral tumor was not definitively documented.

In 1976 Düren and Becker[14] reported the case of a patient who had an astrocytoma; an electrocardiogram was suggestive of anteroseptal infarction. These changes did not develop until seven days after surgical excision, however, so they cannot be related to the tumor with certainty.

Typical electrocardiographic abnormalities that have been related to central nervous system disorders are bradycardia, prolongation of the QT interval, ST segment elevation and depression, deeply inverted T waves, and U waves.[15-17] Large upright T waves[18] and notched T waves[19] have also been reported. None have been found to correlate specifically with any type of pathology.[15,16]

The pathophysiologic mechanism for these abnormalities remains speculative. Experimental studies have shown that manipulation of various central nervous system structures can result in electrocardiographic evidence suggestive of ischemia.[20,21] These phenomena may be mediated by sympathetic pathways resulting in alterations in cardiac autonomic tone. Yanowitz and associates[22] have shown that stimulation of the stellate ganglia in dogs can produce ST-T wave changes similar to those observed with central nervous system disease. Microscopic lesions of the myocardium may also play a role in the development of electrocardiographic anomalies. In 1968 Conner[23] found focal myocytolysis in the myocardium of 18 of 231 patients who died from intracranial disease. Thus, multiple factors may be responsible for the electrocardiographic phenomena in these patients.

The patient in this case presented with focal seizures and an electrocardiogram suggestive of anterior wall myocardial infarction. The initial differential diagnosis included cerebral embolization from a left ventricular mural thrombus versus a metastatic brain lesion (given the prior history of breast carcinoma). Computerized axial tomography demonstrated a cerebral lesion which was felt to be metastatic. Following radiation therapy and the use of corticosteroids, the cerebral mass diminished in size and the electrocardiographic abnormalities resolved over the same period of time. This temporal relationship supported a suspicion that the intracranial pathology was responsible for the infarction pattern. Further support for this impression was provided by the noninvasive cardiologic evaluation, which failed to demonstrate any evidence of myocardial infarction.

The possibility remains that the patient could have had a myocardial infarction prior to admission which escaped detection. The results of the noninvasive evaluation, including LDH cardiac enzymes, and the fact that resolution of the electrocardiogram occurred with radiation therapy, make this possibility seem remote.

Although the patient also had a metastatic pulmonary nodule, it was felt that it could not be responsible for the abnormal electrocardiogram since it was small in size and not adjacent to the heart.

To our knowledge, this is the first reported case of an electrocardiogram suggestive of anterior wall infarction which was clearly shown to be related to a metastatic cerebral lesion. This report also underscores the difficulty of using the surface electrocardiogram for cardiac diagnosis in patients with intracranial pathology.

Acknowledgments. The authors thank Mr Daniel R. Morse for his technical assistance.

REFERENCES

1. Goldberger AL: Recognition of ECG pseudoinfarct patterns. *Mod Concepts Cardiovasc Dis* 1980; 49:13–18.
2. Scheinman MM, Goldshlager N: ECG diagnostic pearls and pitfalls. 1. Acute myocardial infarction and pseudoinfarction. *Postgrad Med* 1981; 70:125–132.
3. Salem BI, Schnee M, Leatherman LL, et al: Electrocardiographic pseudo-infarction pattern: Appearance with a large posterior pericardial effusion after cardiac surgery. *Am J Cardiol* 1978; 42:681–685.
4. Klein LW, Meller J: Hyperkalemia-induced pseudoinfarction pattern. *Mt Sinai J Med* 1983; 50:428–431.
5. Byer E, Ashman R, Toth LA: Electrocardiograms with large, upright T waves and long Q-T intervals. *Am Heart J* 1947; 33:796–806.
6. Levine HD: Non-specificity of the electrocardiogram associated with coronary artery disease (Phei Chi lecture). *Am J Med* 1953; 15:344–355.
7. Cropp GJ, Manning GW: Electrocardiographic changes simulating myocardial ischemia and infarction associated with spontaneous intracranial hemorrhage. *Circulation* 1960; 22:25–38.
8. Menon IS: Electrocardiographic changes simulating myocardial infarction in cerebrovascular accident. *Lancet* 1964; 2:433–434.
9. Burch GE, Meyers R, Abildskov JA: A new electrocardiographic pattern observed in cerebrovascular accidents. *Circulation* 1954; 9:719–723.
10. Hersch C: Electrocardiographic changes in head injuries. *Circulation* 1961; 23:853–860.
11. Hugenholtz PG: Electrocardiographic abnormalities in cerebral disorders. Report of six cases and review of the literature. *Am Heart J* 1962; 63:451–461.
12. Hersch C: Electrocardiographic changes in subarachnoid hemorrhage, meningitis, and intracranial space-occupying lesions. *Brit Heart J* 1964; 26:785–793.
13. Finkelstein D, Nigaglioni A: Electrocardiographic alterations after neurosurgical procedures. *Am Heart J* 1961; 62:772–784.
14. Düren DR, Becker AE: Focal myocytolysis mimicking the electrocardiographic pattern of transmural anteroseptal myocardial infarction. *Chest* 1976; 69:506–511.
15. Abildskov JA, Millar K, Burgess MJ, et al: The electrocardiogram and the central nervous system. *Progr Cardiovasc Dis* 1970; 13:210–216.
16. Umali F, Gould L, Gomprecht RF: Electrocardiographic changes in intracranial lesions. *Angiology* 1971; 22:616–621.
17. Chia BL, Ghosh MB, Tay HH: Electrocardiographic changes in cerebral diseases. *Singapore Med J* 1971; 12:341–345.
18. Burch GE, Philips JH: The large upright T wave as electrocardiographic manifestation of intracranial disease. *Southern Med J* 1968; 61:331–336.
19. Millar K, Abildskov JA: Notched T waves in young persons with central nervous system lesions. *Circulation* 1968; 27:597–603.
20. Porter RW, Kamikawa K, Greenhoot JH: Persistent electrocardiographic abnormalities experimentally induced by stimulation of the brain. *Am Heart J* 1962; 64:815–819.
21. Melville KI, Blum B, Shister HE, et al: Cardiac ischemic changes and arrhythmias induced by hypothalmic stimulation. *Amer J Cardiol* 1963; 12:781–791.
22. Yanowitz F, Preston JB, Abildskov JA: Functional distribution of right and left stellate innervation to the ventricles: Production of neurogenic electrocardiographic changes by unilateral alteration of sympathetic tome. *Circ Res* 1966; 18:416–428.
23. Connor RC: Heart damage associated with intracranial lesions. *Brit Med J* 1968; 3:29–31.

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication. All letters are personally acknowledged by the Editor.*

Physicians and access to health care

TO THE EDITOR: Access to health care is one of the crucial yardsticks by which physicians are being judged in this time of turbulence. As some hospitals veer in their terminology from "patient" to "consumer" and from "caring" to "managing the product," physicians must be careful to keep their concern for the patient uppermost in their minds.

The plight of the elderly, the impoverished, and the severely ill patient is going to darken the medical landscape as health care is pruned in the name of fiscal responsibility. One must clearly distinguish between medical and business ethics. Caring and profit will not always go hand in hand. There are choices and consequences and physicians must ally themselves with their patients if they are to withstand the onslaught of those corporate and governmental thrusts that seek to make physicians the scapegoats for their cost-cutting philosophy.

If the poor among us are ignored and only the affluent can receive quality care, if the elderly and the severely ill are viewed as excessive consumers of health care resources, physicians will be recorded as the uncaring agents of society. The hour is late and the bell tolls. Physicians must join with their patients to assure "medical untouchables" that they have not lost their way. Physicians must show that patients are paramount in their eyes regardless of their income and medical condition. This is a sacred oath and physicians must honor it lest they become fallen idols.

RANDALL D. BLOOMFIELD, MD
Department of Obstetrics
and Gynecology
SUNY Health Science Center
at Brooklyn
450 Clarkson Ave
Brooklyn, NY 11203

The jury is a crowd

TO THE EDITOR: The recent "stun gun" case in Queens, New York, unveiled potential problems that may occur in the common jury room. After the jury's decision was rendered, one juror alleged that her vote had been made as a result of compulsion. This emphasizes the possible influence of crowd psychology under such circumstances and underscores the need for a reassessment of this phase of the judicial process.

The present system of trial by jury protects the individuality of each juror as best as possible while he or she listens to the presentation of evidence by attorneys for both plaintiff and defendant. The presiding judge oversees the conduct of the adversarial process and carefully instructs members of the jury regarding how to weigh the quality and quantity of evidence and their responsibility to decide facts based on the evidence presented. Most important, the members of the jury are repeatedly admonished not to discuss the case with anyone, not even other jurors, during the presentation of evidence. Finally, the judge apprises the jurors of the nature of the issues which they have to decide. They, the jurors, are to establish the facts. The trial is over. Each juror has been exposed to due process, has heard the evidence presented, and has been educated about his or her responsibilities by the judge.

The system now takes the members of the jury, who had functioned as individuals up to this time, and herds them into a common jury room. The jury becomes a psychological crowd that possesses its own characteristics, quite different from those of an individual. In effect, the system in which the jury decides facts now says that if you want to know facts you must ask a crowd. This mistake is avoidable if we maintain the individuality of each juror and have each juror brought to his or her own room separate from all the other jurors. Here he or she could be presented with all the evidence, testimony, exhibits, etc. The juror could decide independently what the evidence supports, without the influence of a leader or peer pressure to conform. It is the individual juror's decision based upon the evidence as he or she interprets it. The individual vote is delivered to the judge who counts it with the others in the presence of both attorneys. It is at this time that the degree of consensus is known.

In the present system, little attempt is made to protect the individuality of each juror. The inherent group psychodynamics are such as to make the jurors identify with one another and thereby attain a consensus, a group feeling, before a vote. This is the mistake—and quite different from the circumstance wherein each juror is isolated and votes alone, with the only constraint being that of the interpretation of evidence presented during the trial.

Is the opinion of each juror voting independently to be feared? If this results in more "hung juries," so be it. That is justice. This would be an indication that there was insufficient evidence to permit each individual juror to vote so that a consensus emerged.

The present system creates a jury that is in effect a psychological crowd. The implications of this for all jury decisions, including those concerned with medical malpractice, are great. A review of this situation may reveal some of the reasons for today's increasing discontent with the decisions of juries assembled in a common jury room.

ALBERT W. COOK, MD
The Long Island College Hospital
340 Henry St
Brooklyn, NY 11201

Relation of stroke to cocaine abuse

TO THE EDITOR: The frequency of cocaine abuse continues to escalate in American society. It is estimated that 30 million Americans have used cocaine and that some 5 million use it regularly.[1] A review of the current literature on cocaine revealed seven case reports of cerebrovascular accidents in cocaine users.[2-5] Cases of stroke with a temporal relation to cocaine use were reported in five individuals (mean age, 28 years; range, 22–43 years). We report the occurrence of stroke in an older, diabetic man, a complication of cocaine use not previously described in this population.

Case Report. A 63-year-old diabetic, normotensive man was seen in the emergency room after the sudden onset of severe pain in the back of his head radiating down the neck. The severe head and neck pain had commenced eight to ten hours before the patient sought treatment, and approximately one and a half to two hours after cocaine use. The patient appeared lethargic and somnolent, and initially was deemed to be intoxicated. He was given thiamine, glucose, and naloxone hydrochloride without clinical response. The patient became comatose and responded to deep painful stimuli only.

Physical examination revealed a well developed, obese man appearing to be his stated age. His blood pressure was 160/90 mm Hg in the supine position. His heart rate was 92/min; respiratory rate, 20/min; and temperature, 36.6°C. The pupils measured 3 mm bilaterally. Laboratory studies revealed a white blood cell count of 15,700/mm^3, hemoglobin level of 42.3 g/dL, and a glucose level of 305 mg %. The blood alcohol level was zero. Arterial blood gas studies on room air showed a pH of 7.39; PCO_2 of 43 mm Hg; and PO_2 of 73 mm Hg. The chest was clear with decreased breath sounds at the base. A chest radiograph was normal. Cardiovascular examination revealed no abnormalities; no murmur or gallop was heard. An electrocardiogram indicated normal sinus rhythm with nonspecific ST-T changes and a prolonged QT_c interval. Neurologic examination revealed a comatose individual who responded to painful stimuli only. There was pain on neck flexion and a right extensor plantar response.

A history obtained from the patient's brother indicated that the patient had used approximately 1 g of cocaine intranasally 10 to 12 hours prior to admission to the hospital, but it was unclear if he had used cocaine again on the day of admission. He had been using cocaine intranasally for several years with a frequency of one to two times a week. Six months prior to admission he had increased his usage to two to three times a week. The patient had had diabetes for 15 years which was treated through diet; an oral agent was prescribed which he did not take. He had no prior history of headaches.

After the patient became comatose, a computed tomographic (CT) scan of the head was performed. The CT scan showed a subarachnoid hemorrhage, small intraventricular hemorrhage, and marked hydrocephalus (Figs 1, 2). Cerebral angiography was not performed.

A decompression shunt was introduced, and the patient was treated with steroids and mannitol. He remained comatose and died eight days after admission.

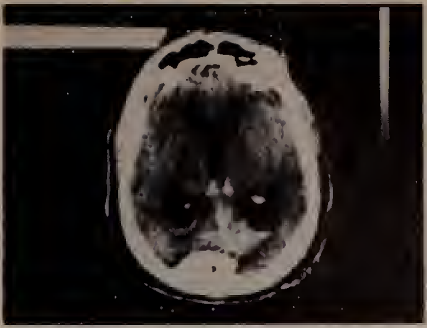


FIGURE 1. Noncontrast CT scan showing increased density in the occipital horn of the lateral ventricles. Blood can be seen in the superior cerebellar system.

Discussion. It is difficult to establish the exact temporal relationship between cocaine use and subarachnoid hemorrhage in the patient described here, as the family was inconclusive about the patient's drug history. This patient, like five others previously described, manifested a subarachnoid hemorrhage temporally related to cocaine usage. In the previous five cases, subarachnoid hemorrhage occurred within several minutes of intranasal ingestion of cocaine. Two of the patients had anterior communicating artery aneurysms and another had a right posterior communicating artery aneurysm. In the fourth case, bleeding was probably from an arteriovenous malformation. The fifth patient probably had an occlusion of the anterior branch of the left middle cerebral artery.[2] This occurred between one and two hours after intramuscular cocaine use. Two additional cases of stroke possibly related to cocaine use have been reported. Of these, the first patient was evasive about drug use; therefore it was difficult to establish the exact temporal relationship between cocaine usage and the intracerebral hemorrhage. The second patient had used cocaine one day prior to the cerebral infarct.[3]

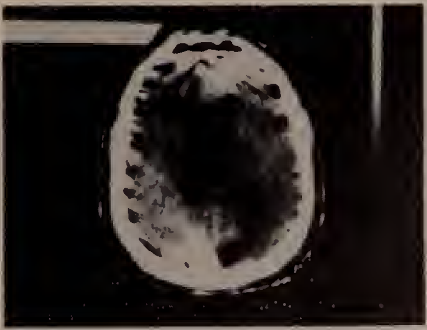


FIGURE 2. Noncontrast CT scan showing markedly dilated lateral ventricles; posterior interhemispheric subarachnoid bleeding is present.

The mechanism of the cerebrovascular accidents probably represents adrenergic stimulation and a sudden surge in blood pressure. Sudden surges in blood pressure in normotensive persons may be the precipitant for spontaneous bleeding.[4] Tachycardia and elevation of blood pressure may occur within minutes of intranasal cocaine usage. Any person with an arteriovenous malformation or aneurysm may be at risk.[4]

The incidence of subarachnoid hemorrhage related to cocaine usage is unknown. Physicians caring for patients with headaches that follow the use of cocaine should be aware of the possibility of intracranial hemorrhage.[4,6] The previously reported subarachnoid hemorrhages all occurred in young people but the physician must also be alert to severe headaches in normotensive older individuals who are recreational users of cocaine. A greater risk may be assumed in older patients with vascular disease, especially in the presence of diabetes.

Acknowledgment. The authors thank Carolyn Morrison for her assistance in the preparation of this manuscript.

LOUIS L. CREGLER, MD
Assistant Chief of Medicine

HERBERT MARK, MD
Chief of Medicine
Department of Medicine
Veterans Administration
Medical Center
Bronx, NY 10468

1. Abelson HI, Miller JD: A decade of trends in cocaine use in the household population. *Natl Inst Drug Abuse Res Monogr Ser* 1985; 61:35-49.
2. Brust JC, Richter RW: Stroke associated with cocaine abuse? *NY State J Med* 1977; 77:1473-1475.
3. Schwartz KA, Cohen JA: Subarachnoid hemorrhage precipitated by cocaine snorting [letter]. *Arch Neurol* 1984; 41:705.
4. Lichtenfeld PJ, Rubin DB, Feldman RS: Subarachnoid hemorrhage precipitated by cocaine snorting. *Arch Neurol* 1984; 41:223-224.
5. Caplan LR, Hier DB, Banks G: Current concepts of cerebrovascular disease—stroke: Stroke and drug abuse. *Stroke* 1982; 13:869-872.
6. Cregler LL, Mark H: Cardiovascular dangers of cocaine abuse [editorial]. *Am J Cardiol* 1986; 57:1185-1186.

AN APPRECIATION

Our referees

The cornerstone of a peer-reviewed journal is its refereeing process. In 1986 the individuals listed below served as referees for manuscripts submitted to the Journal. *Their continuing contribution is acknowledged with deep appreciation. We invite other individuals who would like to become involved in the editorial process to write to the* Journal.

Steven B. Abramsen, MD, New York, NY
John C. Adkins, MD, Pittsburgh, PA
Martin A. Adson, MD, Rochester, MN
Stewart G. Albert, MD, St Louis, MO
Elliot Alpert, MD, Houston, TX
Dana K. Andersen, MD, Brooklyn, NY
George J. Annas, JD, MPH, Boston, MA
Linda K. Ansbacher, MD, Columbia, MO
Donald Armstrong, MD, New York, NY
Barry Gilbert Arnason, MD, Chicago, IL
Keith W. Ashcraft, MD, Kansas City, MO
Robert Austrian, MD, Philadelphia, PA
Michael S. Baggish, MD, Syracuse, NY
Henry D. Balfour, Jr, MD, Minneapolis, MN
William F. Balistreri, MD, Cincinnati, OH
Gene V. Ball, MD, Birmingham, AL
Emil Bardana, Jr, MD, Portland, OR
Ellen Jo Baron, PHD, Manhasset, NY
George E. Barratt, MD, Yuma, AZ
Martin I. Baskin, MD, New York, NY
Stephen Baumgart, MD, Philadelphia, PA
Ronald Bayer, PHD, Hastings-on-Hudson, NY
Peter M. Becker, MD, Durham, NC
Lowell E. Bellin, MD, New York, NY
Joseph H. Bellina, MD, New Orleans, LA
Roberto Belmar, MD, Bronx, NY
James P. Bennett, Jr, MD, Charlottesville, VA
Ralph C. Benson, Jr, MD, Rochester, MN
Joseph R. Berger, MD, Miami, FL
Paul E. Berger, MD, Long Beach, CA
Kenneth J. Bergmann, MD, Ridgefield, CT
Michael E. Berlow, MD, Albany, NY
Bram H. Bernstein, MD, Los Angeles, CA
Robert F. Betts, MD, Rochester, NY
Henry J. Binder, MD, New Haven, CT
Randall Bloomfield, MD, Brooklyn, NY
Naomi Bluestone, MD, Great Neck, NY
Clifford R. Boeckman, MD, Akron, OH
Eugene L. Bodian, MD, Greak Neck, NY
Raymond Charles Bonnabeau, MD, St Paul, MN
John M. Bowman, MD, Winnipeg, Manitoba, Canada
John Boyce, MD, Brooklyn, NY
Robert D. Brandstetter, MD, New Rochelle, NY
Emmanuel L. Bravo, MD, Cleveland, OH
Robert G. Brayton, MD, New York, NY
Lloyd Hay Bremer, MD, Centerville, MA
Arthur E. Broadus, MD, New Haven, CT
Charles W. Broders, MD, Temple, TX
Charles G. Brown, MD, Columbus, OH
John C. Brust, Jr, MD, New York, NY
George T. Budd, MD, Cleveland, OH
Richard J. Calame, MD, Brooklyn, NY
Donald B. Calne, MD, Bethesda, MD
John R. Campbell, JD, Portland, OR
Paul P. Carbone, MD, Madison, WI
Philip R. Caropreso, MD, Mason City, IA
C. James Carrico, MD, Seattle, WA
Herbert B. Carter, MD, New York, NY
Saml Choi, MD, New York, NY
Leonard A. Cobb, MD, Seattle, WA
Carl Cohen, MD, Brooklyn, NY
Hartley Cohen, MD, Los Angeles, CA
Seymour R. Cohen, MD, Los Angeles, CA
David M. Cook, MD, Portland, OR
Edward M. Cordasco, MD, Cleveland, OH
Lawrence Corey, MD, Seattle, WA
Stanley W. Coulthard, MD, Tucson, AZ
David Crippen, MD, Indianapolis, IN
John A. Crocco, MD, New York, NY
James W. Curran, MD, Atlanta, GA
Louis C. Cutolo, MD, Brooklyn, NY
Michael Cynamon, MD, Syracuse, NY
David A. Danford, MD, Bellaire, TX
Angelo M. Taveira DaSilva, MD, Washington, DC
Leonard Davis, MD, New York, NY
Jack A. DeHovitz, MD, Brooklyn, NY
Robert A. Derzon, San Francisco, CA
Frank DeStefano, MD, Atlanta, GA
David E. Dines, MD, Rochester, MN
George W. Drach, MD, Tucson, AZ
Daniel B. Drachman, MD, Baltimore, MD
Roger C. Duvoisin, MD, New Brunswick, NJ
Anne Dyson, MD, Beloit, WI
Alvin N. Eden, MD, Forest Hills, NY
John N. Edson, MD, Brooklyn, NY
Kathryn M. C. Edwards, MD, Nashville, TN
Paul M. Ehrlich, MD, New York, NY
John M. Eisenberg, MD, Philadelphia, PA
Toomas Eisler, MD, Walla Walla, WA
Sven Erling Ekholm, MD, Rochester, NY
Patricia H. Ellison, MD, Englewood, CO
James Endicott, Jr, MD, Tampa, FL
Franz Enzinger, MD, Washington, DC
Gerald Erenberg, MD, Cleveland, OH
Yener S. Erozan, MD, Baltimore, MD
Andrew L. Evans, MD, Springfield, MO
Harry L. Evans, MD, Houston, TX
Stanley Falin, MD, New York, NY
Robert J. Fass, MD, Columbus, OH
Anthony S. Fauci, MD, Bethesda, MD
Jack M. Fein, MD, Poughkeepsie, NY
Elaine B. Feldman, MD, Augusta, GA
Joseph Feldman, DRPH, Brooklyn, NY
Yehudi Felman, MD, Brooklyn, NY
Birdwell Finlayson, MD, PHD, Gainesville, FL
Pamela Fischer, MD, Baltimore, MD
Richard N. Fogoros, MD, Pittsburgh, PA
John R. Ford, MD, San Diego, CA
Arthur Fox, MD, New York, NY
Donald P. Francis, MD, Atlanta, GA
Andrew J. Franzone, MD, New York, NY
Joseph H. Friedman, MD, Providence, RI
David Fromm, MD, Syracuse, NY
Susan A. Fuhrman, MD, Minneapolis, MN
Osvaldo Fulco, MD, East Meadow, NY
Holly S. Gallion, MD, Lexington, KY
Bruce M. Gans, MD, Boston, MA
Charles D. Gerson, MD, New York, NY
Louis A. Gilula, MD, St Louis, MO
Ciril Godec, MD, Brooklyn, NY
Gerald S. Golden, MD, Memphis, TN
Myles Gombert, MD, Brooklyn, NY
Alexander Gotta, MD, Brooklyn, NY
Sherwood L. Gorbach, MD, Boston, MA
Frederick J. Goulding, MD, Charleston, SC
Harry S. Greenberg, MD, Ann Arbor, MI
Richard L. Guerrant, MD, Charlottesville, VA
Marc J. Gurwith, MD, Philadelphia, PA
Peter M. Guzy, MD, Los Angeles, CA
Stephen Hadler, MD, Atlanta, GA
Steven M. Haffner, MD, San Antonio, TX
John S. Haller, Jr, Denver, CO
James Hardy, MD, Jackson, MS
Dwight E. Harken, MD, Cambridge, MA
Jay K. Harness, MD, Ann Arbor, MI
Elisabeth Heiberg, MD, St Louis, MO
Ming K. Heng, MD, Sepulveda, CA
Victor Herbert, MD, Bronx, NY
Alan Hertz, MD, Brooklyn, NY
John C. Hoefs, MD, Irvine, CA
Robert C. Hoeldtke, MD, Philadelphia, PA
George Richardson Huggins, MD, Philadelphia, PA
Harvey D. Humes, MD, Ann Arbor, MI
Robert S. Hunter, MD, Cincinnati, OH
Anthony M. Imparato, MD, New York, NY
Alfred P. Ingegno, MD, Brooklyn, NY
Ida Ismailer, MD, Forest Hills, NY
Harold L. Israel, MD, Philadelphia, PA
Michael B. Jacobs, MD, Stanford, CA
Bernard M. Jaffe, MD, Brooklyn, NY
Fidelio A. Jimenez, MD, Brooklyn, NY
Carol G. Johnson Johns, MD, Baltimore, MD
Larry Junck, MD, Ann Arbor, MI
C. Ronald Kahn, MD, Boston, MA
James G. Kane, MD, Washington, DC
Richard E. Kanner, MD, Salt Lake City, UT
Allen P. Kaplan, MD, St James, NY
Elaine M. Kaptein, MD, Los Angeles, CA
Constantine P. Karakousis, Buffalo, NY
Edward H. Kass, MD, Boston, MA
Florence Kavaler, MD, Brooklyn, NY
Martin Kay, MD, Flushing, NY

F. A. Khan, MD, Jamaica, NY
Alan P. Knutsen, MD, St Louis, MO
Morris Kotler, MD, Philadelphia, PA
Charles Kramer, New York, NY
Guenter J. Krejs, MD, Dallas, TX
Yosef Krespi, MD, Brooklyn, NY
Peter E. Krumpe, MD, Martinez, CA
Kusum L. Kumar, MD, Portland, OR
Patricia Kuszler, MD, Meriden, CT
Robert A. Kyle, MD, Rochester, MN
Robert Landesman, MD, New York, NY
Herbert G. Langford, MD, Jackson, MS
Carl V. Leier, MD, Columbus, OH
Henry M. Lerner, MD, Newton, MA
Barry W. Levine, MD, Boston, MA
David A. Lewis, MD, Iowa City, IA
Peter A. Lewitt, MD, Detroit, MI
S. Arthur Localio, MD, New York, NY
Frank A. Loda, Jr, MD, Chapel Hill, NC
James Lomax, MD, Brooklyn, NY
Kenneth M. Ludmerer, MD, St Louis, MO
Tom F. Lue, MD, San Francisco, CA
Daryl L. Luke, MD, St Louis, MO
D. Joanne Lynn, MD, Clifton, VA
Douglas McCallum, MD, Denver, CO
Kathleen M. McCarten, MD, Framingham, MA
George H. McCracken, Jr, MD, Dallas, TX
John H. McGovern, MD, New York, NY
Kelly Tilson McKee, Jr, MD, Frederick, MD
Richard Macchia, MD, Brooklyn, NY
Lewis R. Malinak, MD, Houston, TX
Renee Malouf, MD, New York, NY
Francis E. Marchlinski, MD, Philadelphia, PA
Jose A. Marti, MD, Brooklyn, NY
Stephen Marx, MD, Bethesda, MD
Byron J. Masterson, MD, Louisville, KY
John S. Marr, MD, New York, NY
Alfred W. Meikle, MD, Salt Lake City, UT
Wilfred Minkin, MD, White Plains, NY
Vijay K. Mittal, MD, Detroit, MI
Rogelio Moncada, MD, Maywood, IL
Ernest E. Moore, MD, Denver, CO
George E. Moore, MD, Denver CO
David M. Morens, MD, Honolulu, Hawaii
James Morrissey, JD, Albany, NY
Myron Moskowitz, MD, Cincinnati, OH
Donald Moy, JD, Lake Success, NY
Edward C. Muecke, MD, New York, NY
Peter S. Mueller, MD, Princeton, NJ
David P. Munson, MD, Sioux Falls, SD
Anthony C. Mustalish, MD, New York, NY
David P. Nardich, MD, Baltimore, MD
Kenwyn G. Nelson, MD, Tyler, TX
Thomas Nims, MD, Columbus, OH
Barry N. Nocks, MD, Boston, MA
Lawrence W. Norton, MD, Denver CO
Andrew C. Novick, MD, Cleveland, OH
John G. Nutt, MD, Portland, OR
Paul Allen Offit, MD, Palo Alto, CA
Stanley E. Order, MD, Baltimore, MD
Michael J. Osborn, MD, Rochester, MN
Charles N. Oster, MD, Nairobi, Kenya
Kent B. Pandolf, PHD, Natick, MA
Joseph P. Patin, MD, Baton Rouge, LA
Howard A. Pearson, MD, New Haven, CT
Karl H. Perzin, MD, New York, NY
Mark E. Pessa, MD, Gainesville, FL
Eric W. Peterson, MD, Ottawa, Ontario, Canada
Jack Pickleman, MD, Maywood, IL
Paul J. Placek, PHD, Hyattsville, MD
Carl Pochedly, MD, Austin, TX
Ronald J. Polinsky, MD, Rockville, MD
Margaret M. Portwood, MD, Sacramento, CA
Charles S. Powell, MD, Buffalo, NY
John H. C. Ranson, MD, New York, NY
Lee Reichman, MD, Newark, NJ
Ronald Rieder, MD, Brooklyn, NY
James Rippe, MD, Worcester, MA
S. David Rochoff, MD, Washington, DC
Harlan D. Root, MD, San Antonio, TX
Leslie I. Rose, MD, Philadelphia, PA
Carol Lynn Rosen, MD, New York, NY
Hana Rostain, MD, New York, NY
David A. Rothenberger, MD, St Paul, MN
Stanley J. Rothman, MD, Valhalla, NY
Lawrence Z. Rubenstein, MD, Los Angeles, CA
Alan M. Rushovich, MD, Evanston, IL
Thomas M. Saba, PHD, Albany, NY
Thomas D. Sabin, MD, Boston, MA
Stephen A. Sacks, MD, Los Angeles, CA
Julius Sagel, MD, Charleston, SC
John S. Salaki, MD, Newark, NJ
John C. Scatarige, MD, Norfolk, VA
Andrew I. Schafer, MD, Boston, MA
Daniel V. Schidlow, MD, Philadelphia, PA
Joseph Schluger, MD, Brooklyn, NY
Peter L. Schnall, MD, New York, NY
Marshall T. Schreeder, MD, Huntsville, AL
Philip Schulman, MD, Manhasset, NY
Michael J. Schultz, MD, Baltimore, MD
Steven Schultz, MD, New York, NY
Kenneth A. Schwartz, MD, New York, NY
Ralph M. Schwartz, MD, New York, NY
Robert R. Shaw, MD, Dallas, TX
Joseph L. Sheets, MD, Lansing, MI
Sheldon G. Sheps, MD, Rochester, MN
Stanley S. Siegelman, MD, Baltimore, MD
Mark Siegler, MD, Chicago, IL
Archie Silver, MD, Tampa, FL
Jeffrey Silvers, MD, San Leandro, CA
Franklin H. Sim, MD, Rochester, MN
Marvin H. Sleisinger, MD, San Francisco, CA
Raymond A. Smego, MD, Towson, MD
Henry W. Smith III, MD, Atlanta, GA
Joseph J. Smith, MD, Bronx, NY
Carl Soderstrom, MD, Baltimore, MD
Martha Sonnenberg, MD, Chicago, IL
Richard F. Spark, MD, Westwood, MA
Allen Spiegel, PHD, Brooklyn, NY
Randy E. Sprague, MD, St Louis, MD
Mark R. Starling, MD, Ann Arbor, MI
Richard A. Stein, MD, Brooklyn, NY
William M. Steinberg, MD, Washington, DC
Philip Steiner, MD, Brooklyn, NY
Dennis L. Stevens, MD, Boise, ID
Ronald D. Stewart, MD, Pittsburgh, PA
Richard Stillman, MD, Brooklyn, NY
Alex M. Stone, MD, New Hyde Park, NY
Diane E. Stover, MD, New York, NY
Michael S. Stulbarg, MD, San Francisco, CA
Ofelia C. Tablan, MD, Detroit, MI
Michael Tenner, MD, Valhalla, NY
Patrick O. Tennican, MD, Spokane, WA
Arthur N. Tessler, MD, New York, NY
Bjorn Thorbjarnarson, MD, New York, NY
Charles D. Tischler, MD, Newark, NJ
W. W. Tourtellotte, MD, PHD, Los Angeles, CA
William E. Truog, MD, Seattle, WA
Rodrigo Urizar, MD, Albany, NY
Jerome A. Urban, MD, New York, NY
Alje Vennema, MD, New York, NY
Frank R. Venezio, MD, Maywood, IL
Charles N. Verheyden, MD, Temple, TX
Lee W. Vliet, MD, Norfolk, VA
David Hughes Walker, MD, Chapel Hill, NC
Sylvan Wallenstein, MD, New York, NY
David A. Waller, MD, Dallas, TX
Shaw P. Wan, MD, Rollo, MO
Rudolph A. Watson, MD, Minneapolis, MN
Barbara M. Weissman, MD, Cleveland, OH
G. Rainey Williams, MD, Oklahoma City, OK
Terry P. Yarbrough, MD, Portsmouth, VA
Charles A. Young, MD, Mt Clemens, MI
Jimmy Boyd Zachary, MD, Baltimore, MD
Harold S. Zarkowsky, MD, St Louis, MO
John L. Ziegler, MD, San Francisco, CA
Richard J. Zigrossi, MD, Pittsburgh, PA
Stanley Zinberg, MD, New York, NY
Adrian W. Zorgniotti, MD, New York, NY

EXERCISE AND SPORTS SCIENCES REVIEWS

Volume 14. Edited by Kent B. Pandolf, PHD. 475 pp, illustrated. New York, Macmillan Publishing Co, 1986. $39.95 (hardcover)

Edited by Kent Pandolf, a Fellow of the American College of Sports Medicine and the current Director of the Ergonomics Division of the US Army Research Institute of Environmental Medicine in Natich, Massachusetts, volume 14 of *Exercise and Sports Sciences Reviews* represents another fine addition to an outstanding series. The series has been published annually since 1973 and is devoted to current research in exercise science. Twenty recognized authorities were chosen for this edition, and they address specific topics in the clinical, biomechanical, and rehabilitative aspects of sports and exercise. The target audience includes students, educators, practitioners, and exercise investigators.

The book's contents consist of 14 chapters that can be categorized as follows: physiology, biochemistry, rehabilitation, biomechanics, clinical practice, and growth and development. The latter chapter discusses the controversial topics of body composition in children and adolescents. A multicomponent approach to body composition is presented with respect to accurate estimations of body fat and chemical immaturity. The clinical chapter provides a succinct review of injury trends in the pediatric population. A brief overview of treatment for common conditions is provided. The two biomechanics chapters discuss sport shoe design and long jump, respectively. Both include discussions of analytic laboratory methods as well as clinical applications of such techniques.

The topic of rehabilitation is presented in a clinical (exercise training following cardiovascular surgery) and basic science format (isokinetic training). The advantages and disadvantages of each are discussed. The relevance of protein in endurance exercise and its clinical and physiological correlates (protein supplements, strength) are handled in that same chapter. The purpose, technique, and results of fiber typing and potential physiological classification are discussed in a second presentation on biochemistry. This is a rapidly changing field with significant impact on clinical sports science.

The remaining six chapters deal with selected physiological issues of static exercise, spinal circuitry and motor reflexes, upper body exercise, sleep deprivation and exercise, cold acclimatization, and high-altitude cardiovascular adaptation to exercise. With the exception of the neurophysiology chapter (which brought back neuroscience nightmares to this reviewer), all the physiology chapters have short sections on clinical applicability and relevance. This perspective negates what would otherwise be an imbalance in the overall distribution of the chapters.

This volume outperforms its immediate predecessor in this reviewer's opinion in that it successfully blends basic science and clinical material in an understandable format. It is recommended for the practitioner and investigator in the sports sciences alike.

RICHARD B. BIRRER, MD
Consulting Editor

SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

LIGAMENT INJURIES AND THEIR TREATMENT

Edited by D. H. R. Jenkins, MB, CHB, CHM, FRCS. 346 pp, illustrated. Rockville, Md, Aspen Publications, 1985. $40.00 (hardcover)

David H. R. Jenkins has endeavored to provide the orthopedic community with a "view of ligament problems from both sides of the Atlantic." To this end, 19 contributors, seven from the United States and 12 from Europe, have provided material divided into three sections. Part One deals with the biomechanical aspects of ligaments, Part Two with the treatment of ligament injuries, and Part Three with the use of substitute graft material. While the title implies a broad discussion of ligament injuries, the majority of the material is related to the knee joint. Parts One and Three together comprise the smaller portion of the book and unfortunately add little to generally available knowledge. In addition, several unfounded statements are presented as fact in Part One, eg, "This is in line with the findings that the anteromedial fibers [of the anterior cruciate ligament] are taut in flexion as shown in Figure 1.15." In reality, exactly the opposite is true.

On page 38, the statement appears that "cruciate ligaments are essential for full athletic activity but limited function is possible in a patient who has sustained a complete rupture of both ligaments and in whom treatment has restored good muscle function." This statement is controversial at best but is presented without benefit of references.

Part Two comprises the bulk of the book. Ligament injuries of the elbow, ankle, and knee are presented in terms of anatomy, mechanisms of injury, and treatment. Unfortunately for the attending orthopedic surgeon or resident, the descriptions of operative procedures are insufficient and many often-used procedures are mentioned only in passing. Illustrations of the procedures are often not available and, when shown, are not detailed enough. To the unwary, the many available options are not clearly presented. Postoperative care, so important in returning the athlete to maximum performance, is not adequately treated. The chapter by J. H. Miller on elbow lesion, however, was an extremely comprehensive review of this often forgotten joint. Illustrations are well done and complement the text well.

Thus, we have a book with excellent intentions. Unfortunately, given the impressive list of contributors it does not live up to its potential. It can, however, be useful for the practicing orthopedist with an interest in ligament injuries as well as for the orthopedic resident. The chapter on elbow injuries alone may well be worth the price of the book. It must, however, be carefully supplemented with other available texts and current journal material. Libraries may find it a useful addition to their section on ligament and athletic injuries.

SHEPARD H. SPLAIN, DO
The Brookdale Hospital
Medical Center
Brooklyn, NY 11212

THE PHYSICIAN AND THE HOPELESSLY ILL PATIENT. LEGAL, MEDICAL AND ETHICAL GUIDELINES

By The Society for the Right to Die. 92 pp. New York, Society for the Right to Die, 1985. $5.00 (softcover)

This book is an excellent introduction to an area that confounds physicians, lawyers, ethicists, philosophers, and theologians alike. It deals with such subjects as when treatment is too much treatment, and when treatment should be withheld from a patient, either at the patient's or at the family's request.

The book is organized into three major sections. The first section consists of an article published in 1984 in the *New England Journal of Medicine* entitled, "The Physician's Responsibility Toward Hopelessly Ill Patients." Written by a group of eminent physicians, it is an excellent, although perhaps too brief, treatment of a complicated subject. This section emphasizes that the wishes of competent patients must be respected, even if those wishes are contrary to a physician's privately or professionally held beliefs. While a physician may consider the prolonging of life a primary value, the hopelessly ill patient may value "death with dignity" more. This section challenges physicians to clarify their own values and how they may inappropriately affect treatment decisions for a patient. Lastly, it urges physicians to communicate fully with their patients about the nature of their illnesses, and discusses proposed treatment modalities.

The second section of the book is presented in a question-and-answer format and discusses those commonly asked questions regarding treatment of the hopelessly ill. This section reviews such areas as the legal basis for refusing treatment, civil liability for disregarding a patient's wishes and giving life-sustaining treatment, living wills, and the concept of "substituted judgment."

The first two sections of the text must be supplemented by the laws of the state in which the physician practices. Anticipating this need, the last section of the book contains a state-by-state table that, for each state, describes statutes and cases, if any, that may be helpful in guiding the physician treating the hopelessly ill patient.

The Society for the Right to Die has done much to clarify the rights of hopelessly ill patients to determine their own medical course, and this book is a further step in that direction. The entire area, however, is extremely complex and volatile, subject to swift statutory, regulatory, or judicial change. The book, therefore, should be viewed as an introduction to the rights of the hopelessly ill, and physicians should be aware that changes in state and federal laws will eventually make some of the information dated.

JAMES MORRISSEY, JD
Public Utility Law Project
Albany, NY 12207

HANDBOOK OF 1985 LIVING WILL LAWS

By The Society for the Right to Die. 128 pp. New York, Society for the Right to Die, 1986. $5.00 (softcover)

The *Handbook of 1985 Living Will Laws* is published by the Society for the Right to Die, which is located in New York City. The *Handbook* contains the text of model living will legislation drafted by the National Conference of Commissioners on Uniform State Laws (approved at its annual conference, August 2–9, 1985), the texts of 13 living will statutes enacted in 1985 (Arizona, Colorado, Connecticut, Indiana, Iowa, Maine, Maryland, Missouri, Montana, New Hampshire, Oklahoma, Tennessee, and Utah), and the text of the 1977 Texas Natural Death Act, which was significantly amended in 1985.

Although not covered in this publication, other handbooks also published by the Society for the Right to Die contain the texts of living will statutes published in other states. *Living Will Laws: 1981–1984* contains the texts of statutes enacted between May 1981 and July 1984 (Alabama, Delaware, District of Columbia, Florida, Georgia, Louisiana, Mississippi, Vermont, Virginia, West Virginia, Wisconsin, and Wyoming), and *The Handbook of Enacted Laws* covers the first ten living will laws that were enacted (Arkansas, California, Idaho, Kansas, Nevada, New Mexico, North Carolina, Oregon, Texas, and Washington).

The term "living will" is a generic term applied to legislation that addresses the rights of individuals to refuse in advance the continuation of medical treatment if it is determined that the patient has become terminally ill and that the continuation of medical treatment will be futile and will only prolong the dying process. No two state laws are exactly alike and the circumstances in which an individual may execute a living will and the procedures that must be followed in the execution and implementation of a living will may vary substantially from state to state.

Realizing the need for uniformity, in August 1985 the National Conference of Commissioners on Uniform State Laws (NCCUSL), whose role it is to develop model legislation when the commissioners see a need for nationwide uniformity, approved a "Uniform Rights of the Terminally Ill Act," recommending it for adoption throughout the United States. The *Handbook of 1985 Living Will Laws* contains the text of this model act, along with commentaries that explain in simple language the more salient features of the act.

Although a separate commentary does not accompany the text of each state law included in the *Handbook*, a commentary is provided concerning what the writer believes to be the emerging trend in living will legislation. The *Handbook* provides statutory citations so that the reader can refer directly to the state code for amendments that may be enacted in the future.

The *Handbook* may be a useful reference source to individuals who are interested in the topic of living will legislation by enabling the reader to quickly identify how other states have addressed living will issues, including such issues as the formal procedures to be followed for a declaration to be valid, the scope of immunity provided to a health practitioner in complying with a declaration, the penalties that may be incurred by a health practitioner in failing to comply with a declaration, the validity of a declaration if a declarant is pregnant, the validity of provisions regarding the withholding of nutrition and hydration, etc.

Although New York is not among the states that have enacted living will legislation to date, individuals who are concerned with the issues pertaining to living wills may wish to be acquainted with the NCCUSL model act and the enactments of other states since legislatures will often look to the laws of sister states in drafting and considering legislation.

DONALD MOY, JD
General Counsel
Medical Society of the
State of New York
Lake Success, NY 11042

GOLDFRANK'S TOXICOLOGIC EMERGENCIES

Third Edition. By Lewis R. Goldfrank, MD, Neal E. Flomenbaum, MD, Neal A. Lewin, MD, et al. 929 pp, illustrated. East Norwalk, Conn, Appleton-Century-Crofts, 1986. $85.00 (hardcover)

The third edition of *Goldfrank's Toxicologic Emergencies* attempts to be a more comprehensive text on poisoning than previous editions. It also continues to provide very readable information on medical toxicology. Dr Goldfrank is one of the leading authorities on medical toxicology and poison control. His many years of experience as Director of Emergency Medical Services at Bellevue Hospital Center in New York City have provided him with a greater clinical experience in treating poisoned patients than most of us will have in a lifetime.

New sections in the text include discussions of different organ systems as they pertain to toxicology, an excellent chapter on withdrawal, and chapters on snakes, arthropods, anaphylaxis, and anaphylactoid reactions.

However, the continued strength of the book is its problem solving format. Fifty eight of the present 75 chapters start with a case presentation, followed by a discussion of the differential diagnosis, appropriate diagnostic work-up, pharmacology of the offending agent, and treatment. Some would argue that this format is also a weakness because it attempts to make the book a text on therapeutic pharmacology, general medicine, and poisonings all at once. For example, the chapter on antipsychotics mainly focuses on the pharmacology and adverse effects seen with the therapeutic use of phenothiazines. The chapter on lidocaine discusses the differentiation between supraventricular and ventricular rhythms, warning arrhythmias in the ischemic heart, and the role of prophylactic lidocaine in an acute myocardial infarction. These chapters do not provide extensive reviews on the acute toxicity seen in overdoses with these agents.

Those practicing medical toxicology, however, realize that medical toxicology is a mixture of pharmacology, general medicine, and poisoning. This book is not intended to be a comprehensive review of all the literature concerning medical toxicology. It is meant to cover many topics in toxicology that are pertinent to physicians in everyday practice in almost all specialties.

The text contains some typograpical errors, instances of unclear typesetting, and some inaccurate information. On page 349, the formula for the extraction fraction is unclear and should read $EF = (A - V)/A$. On page 638, the lead extraction ratio after an EDTA mobilization test is given incorrectly as mg lead/mg EDTA, rather than μg lead/mg EDTA. On page 758, IV calcium gluconate is recommended for severe envenomations from scorpions in the United States, even though this is not and would not be expected to be effective based on the physiology of the venom. Unfortunately, no mention is made of the highly effective antivenom available in Arizona, where most of these scorpions reside. Table 71-1 on page 756 incorrectly states that scorpions bite and that spiders sting. The reverse is true.

This book would best be used in combination with a more comprehensive resource such as *Poisoning and Drug Overdose* by Haddad, or the *POISINDEX* microfiche and computer system. If one wants an extensive review on lithium poisoning, one should read Hadad's book. If one wants to learn how to work up a patient with lithium poisoning, including a complete differential diagnosis, one should read Goldfrank.

Goldfrank's book should be in *all* medical libraries, emergency departments, and poison control centers. It should also be available to physicians in training who are called upon to treat poisoned patients. For those physicians interested in toxicology, this text provides very enjoyable reading in a format that will provide for much greater retention of material than do standard textbooks.

STEVEN C. CURRY, MD
Central Arizona Regional
Poison Management Center
Phoenix, AZ 85006

BASIC TOXICOLOGY. FUNDAMENTALS, TARGET ORGANS, AND RISK ASSESSMENT

By Frank C. Lu. 276 pp, illustrated. New York, Hemisphere Publishing Corp, 1985. $49.95 (hardcover)

As indicated in the title, this book is a general treatment of the subject of toxicology and is intended, according to the author, as an introductory text for students (presumably graduate students in biological sciences) and for toxicologists who want to review areas outside their specialty. It might also be of some interest to physicians who want an introduction to and broad background for understanding the process of drug and environmental chemical toxicity evaluation, a topic of general interest in the scientific and popular media. This is not a text for those seeking advice for evaluating or treating clinical drug or chemical toxicities.

The book is divided into three major sections. The first, on general principles of toxicology, has chapters dealing with the scope of the subject and its history, the disposition of toxicants, biotransformation (metabolism), a general consideration of toxic effects, factors that modify toxic effects on organisms, and the general approach to the evaluation of new chemicals for toxicity. The second part, on testing procedures, considers short- and long-term toxicity studies on animals and test methods for carcinogenesis, mutagenesis, and teratogenesis. The third section deals with testing procedures for evaluating specific organ toxicity including that of the lung, liver, kidney, skin, eye, nervous system, and the reproductive, cardiac, and immune systems.

The chapter on testing for mutagenic activity of chemicals clearly stands out as the best treatment of a topic in this book. It is a model of the goal the other chapters attempt to achieve. Some chapters come close—they are primarily those in sections two and three dealing with descriptions of animal test procedures for organ toxicity. A select group of readers may be looking for just that sort of text and this book would serve the purpose. Section one as a whole falls well short of the goal. The discussion of chemical disposition in chapter 2 is particularly disappointing. For example, the discussion of toxicokinetics is completed in three paragraphs and illustrated by two rudimentary figures. The discussion of metabolism is similarly brief (chapter 3), and although the appendix giving examples of substances undergoing bioactivation is somewhat unique, I do not believe it will substitute for the discussion of fundamentals that most of the intended readers would want or need. The other area in which I expected this text to be strong was its explanation of the methods of calculating risk levels, and this again was not the case. In the chapters on organ toxicity, although the test descriptions are useful, the discussions of mechanisms are also weak. (About the time I started reading the book there was a review article in a journal of clinical medicine that covered the subject of liver toxicity in a more satisfactory manner than chapter 13 of this book. I decided to search for similar articles to substitute for other chapters and found it an easy task.)

On the other hand, the author is to be congratulated on his clear, straightforward writing style. This reviewer particularly appreciated the lack of jargon.

In summary, this book's strong points are in its general description of animal toxicity test procedures. As such it is a rather unique addition to the library, and I would recommend it to the interested reader on that basis alone. As a discussion of scientific fundamentals, however, it falls short. There are other books that will substitute in that area, such as Casarett and Doull's *Toxicology*, now in its second edition.

JOHN J. AMBRE, MD, PHD
Northwestern University Medical School
Chicago, IL 60611

THE FIFTEEN MINUTE HOUR. APPLIED PSYCHOTHERAPY FOR THE PRIMARY CARE PHYSICIAN.

Edited by Marian R. Stuart, PHD, and Joseph A. Lieberman III, MD. 199 pp. New York, Praeger Publishers Inc, 1986. $38.95 (hardcover)

Stuart and Lieberman offer the primary care physician a textbook of office therapeutic skills the likes of which has not been provided since Castelnuevo-Tedesco did so two decades ago in *The Twenty Minute Hour*. In so doing they make it clear that this text offers the primary care physician a workbook of usable therapeutic skills within the context of the ordinary patient visit, whether the patient is suffering primarily from clinical depression, an unfulfilling job, or a sore throat. As a team, trained respectively in social psychology and family medicine, and having experience in both the academic and private settings, the authors are eminently qualified to discuss and present such material.

What is important to the primary care physician may be found in chapters 3–8, in which the authors present a concise and practical framework for incorporating therapeutic principles within the routine office visit. Chapter 3 discusses some of the necessary qualifications for primary care physicians interested in encouraging therapeutic change in their patients. In chapter 5 the authors eloquently distill core therapeutic principles common to many schools of psychotherapy in a way which takes much of the magic and mystique out of the therapeutic process, and in so doing make it clear and understandable for the primary care physician. In addition, the chapter contains an extensive discussion of models for helping and coping, adapted from Brickman. This serves as a thread for much of the rest of the text. In all these chapters the authors focus on a broad range of therapeutic skills. These skills are applicable to a wide variety of patients who are experiencing some form of distress, whether it be from a primary psychiatric problem, a medical illness, or the wear and tear of everyday living. Particularly helpful is a description in chapter 6 of a technique developed by the authors called BATHE, which refers to a systematic way of understanding the nature of a patient's problem from his/her own perspective.

My concerns about this text have to do with the relevance and appropriateness of various chapters. Chapter 1 might be interesting to the primary care physician who has never heard of the biopsychosocial model. Otherwise it repeats an approach which has been discussed in great detail in primary care texts and journals over the past ten years. Chapter 9 appears to be particularly out of context with the rest of the text. It is hard to follow its relevance vis-a-vis the preceding chapters. It focuses on dealing with special problem patients, but its treatment is quite superficial. The chapter concludes with 12 brief rules for physician survival which are interesting and helpful but which do not clearly follow from what precedes them in the chapter. Chapter 10 discusses ways in which physicians and patients are likely to differ as a result of applying the authors' recommendations. It essentially says that physicians will feel better if they employ therapeutic techniques in their practices and that patients will be more self-actualized in many aspects of their lives. The chapter appears to me to be somewhat self-aggrandizing and adds little to the stated goals of the book.

A final criticism relates to a number of comments the authors make concerning the willingness of primary care physicians to employ therapeutic modalities with their patients as described in the book, compared to that of other medical specialists, in particular psychiatrists. The authors seem to suggest that the primary care physician has something of a monopoly on "caring skills" and that psychiatry is so oriented to psychiatric diagnosis and pharmacologic treatment as to have lost a sense of caring about patients' overall well-being. Such impressions by the authors are not documented and would certainly be seen as offensive to practicing psychiatrists.

This book, particularly in its middle chapters, provides excellent practical suggestions for primary care physicians interested in the overall well-being of their patients. It also contains practical therapeutic principles capable of influencing the lives of their patients. One gets the feeling, however, that the authors chose to extend their original theme in order to make the manuscript suitable for textbook form. It seems akin to taking a beautiful poem and trying to turn it into a short story.

RICHARD AMSTOTT, MD, PHD
University of Colorado
Health Sciences Center
Denver, CO 80220

AIDS definition clarified

The Centers for Disease Control (CDC) established a "surveillance definition" of acquired immunodeficiency syndrome (AIDS) to facilitate epidemiologic monitoring of the disease. This definition "is restricted to the more severe manifestations that are consistently reported and are relatively specific for HIV [human immunodeficiency virus] infection," according to CDC spokesmen writing in the *New England Journal of Medicine* (1986; 315:761).

The confusion surrounding this definition can result in misclassification of immunosuppressed patients. For example, patients with other conditions that can cause immunodeficiency—such as autoimmune diseases that require immunosuppressive therapy or certain lymphoid malignant diseases—may not be identified as having AIDS, even though HIV infection is present.

The CDC recommends that the determination of HIV infection be based on tests for serum antibody to HIV. The surveillance definition should not be used as the sole criterion for establishing a diagnosis of AIDS.

Recombinant human erythropoietin used to treat anemia

Patients on long-term maintenance hemodialysis due to chronic renal failure or loss of a kidney often suffer from anemia. In many cases, the anemia is severe enough to warrant blood transfusions. A reduced level of the hormone erythropoietin—which is made primarily by the kidney and which regulates the formation of red blood cells—is believed to be the primary cause of the anemia.

Replacement therapy with erythropoietin has been made possible by the cloning of the gene that codes for human erythropoietin. (Previously, there was no source that could provide sufficient amounts of the hormone to make replacement therapy a feasible treatment option.) Purified, recombinant human erythropoietin (RHE) has been shown to be effective in increasing the number of red blood cells and eliminating the need for blood transfusions among patients on long-term maintenance hemodialysis.

Winearls et al (*Lancet* 1986; 2:1175) used replacement therapy with RHE to treat ten anemic patients who required hemodialysis three times a week. The patients ranged in age from 26 to 65 years. Four patients required regular blood transfusions.

The patients received RHE intravenously after each dialysis treatment. This study, aimed at determining the efficacy and safety of RHE, called for an escalating dose regimen, in which an increasing dose of RHE was administered until the hemoglobin level rose by 2 g/dL or more—indicating a response—above the mean level measured during a two-week period prior to the therapy.

Within 11 weeks of treatment all patients exhibited a response in hemoglobin concentration. The four patients who had received regular blood transfusions no longer needed transfusions. Adverse effects of the treatment included one episode of hypertensive encephalopathy and complete and partial clotting of arteriovenous fistulas in two patients.

The findings suggest that replacement therapy with erythropoietin was able to correct for the anemia associated with renal failure. Comparison of recombinant human erythropoietin to human urinary erythropoietin revealed identical amino acid sequences and very similar carbohydrate structure. Furthermore, no differences in biological activity or immunologic characteristics of the two forms of the hormone have been found.

Winearls et al cite three beneficial results of erythropoietin therapy for these patients: the correction of anemia; the discontinuing of blood transfusions; and the perceived improvement in overall well-being. The authors emphasize the need for further studies to determine the appropriate dose of RHE needed to maintain an adequate hemoglobin concentration and the need for studies to evaluate the risk of thrombotic complications.

New cholesterol-reducing drug announced

Lovastatin, a new drug introduced by Merck Sharp and Dohme, reduces cholesterol levels in the body. The drug works by inhibiting an enzyme system involved in the synthesis of low density lipoprotein. Studies in patients with nonfamilial hypercholesterolemia revealed about a one-third reduction in total cholesterol and in low density lipoprotein, both of which have been implicated in heart disease (*JAMA* 1986; 256:2829). Previous studies have suggested that, on average, a 1% decrease in serum cholesterol can reduce the rate of heart disease by about 2%. Therefore, the dramatic effects of lovastatin could have a substantial impact on the risk of coronary heart disease in patients with hypercholesterolemia.

Lovastatin also produced a slight increase in the levels of high density lipoprotein—which exerts a protective effect against heart disease—and a moderate decrease in the levels of total plasma triglycerides.

The company filed a New Drug Application with the Food and Drug Administration in November 1986. Lovastatin is expected to come to market in the United States by the middle to end of 1988.

In a related article in *JAMA* (1986; 256:2823), researchers involved in the Multiple Risk Factor Intervention Trial (MRFIT) reported a continuous, graded association between serum cholesterol levels and the risk of death due to coronary heart disease: the higher the cholesterol level, the greater the risk of fatal heart disease, and vice versa.

The authors concluded that the majority of American men aged 35 to 57 years would benefit from a reduction in serum cholesterol—cholesterol levels above about 180 mg/dL are associated with an increased risk of premature death due to heart disease.

NEWS BRIEFS are compiled and written by Vicki Glaser.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Charles Leslie Allen, MD, South Norwalk, Connecticut. Died November 20, 1986, age 89. Dr Allen was a 1934 graduate of Université de Paris Faculté de Médecine. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Anthony Joseph Altieri, MD, Phoenix, Maryland. Died November 4, 1986; age 75. He was a 1938 graduate of Georgetown University School of Medicine, Washington, DC. Dr Altieri was a Fellow of the American Academy of Family Practice and a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Samuel Avnet, MD, Beverly Hills, California. Died October 30, 1986; age 75. Dr Avnet was a 1937 graduate of New York Medical College, Flower and Fifth Avenue Hospitals, New York. He was a Diplomate of the American Board of Orthopedic Surgery. His memberships included the Academy of Medicine, the American Academy of Orthopaedic Surgeons, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Abner Berkwits, MD, Brooklyn. Died November 11, 1986; age 65. He was a 1944 graduate of the University of Cincinnati College of Medicine. Dr Berkwits was a Fellow of the American College of Anesthesiologists and a Diplomate of the American Board of Anesthesiology. His memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Labeeb Najib Bokhair, MD, Brooklyn. Died October 31, 1986; age 70. Dr Bokhair was a 1944 graduate of American University of Beirut School of Medicine, Syria. Dr Bokhair was a Qualified Fellow of the International College of Surgeons, a Diplomate of the American Board of Surgery, and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Erich P. Bosshold, MD, Brewster. Died January 12, 1986; age 67. He was a 1944 graduate of New York Medical College, Flower and Fifth Avenue Hospitals, New York. Dr Bosshold was a member of the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Medical Society of the County of Westchester, and the Medical Society of the State of New York. .

Frederick David Bridge, MD, New York. Died September 28, 1986; age 81. Dr Bridge was a 1930 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American College of Chest Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the New York County Medical Society and the Medical Society of the State of New York.

Pauline Feldmann, MD, Riverdale. Died April 28, 1986; age 102. Dr Feldmann was a 1918 graduate of Medizinische Fakultät der Universität, Wien. She was a member of the New York County Medical Society and the Medical Society of the State of New York.

Abraham Florin, MD, Brooklyn. Died October 6, 1986; age 77. He was a 1934 graduate of Universität Bern Medizinische Fakultät. Dr Florin was a Fellow of the American Academy of Family Practice and a member of the American Thoracic Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Eugene Frederick Galvin, MD, Rosendale. Died October 27, 1986; age 87. Dr Galvin was a 1925 graduate of the Albany Medical College of Union University. He was a member of the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Medical Society of the County of Ulster, and the Medical Society of the State of New York.

Louis Goldman, MD, Bronx. Died November 16, 1986; age 80. Dr Goldman was a 1933 graduate of Vanderbilt University School of Medicine, Nashville. He was a member of the American Academy of Family Practice, the American Geriatrics Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

Norman L. Higinbotham, MD, New York. Died November 12, 1986; age 86. He was a 1926 graduate of McGill University Faculty of Medicine, Montreal. Dr Higinbotham was a Fellow of the American College of Surgeons, and a Diplomate of the American Board of Surgery. His memberships included the Academy of Medicine, the New York State Surgical Society, the American Association for the Surgery of Trauma, the Society of Surgical Oncology, the New York County Medical Society, and the Medical Society of the State of New York.

Mark G. Kantor, MD, North Miami Beach, Florida. Died November 5, 1986; age 89. Dr Kantor was a 1922 graduate of State University of New York Health Science Center at Brooklyn. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Abraham Kopp, MD, Teaneck, New Jersey. Died October 22, 1986; age 89. He was a 1921 graduate of State University of New York Health Science Center at Brooklyn. Dr Kopp was a Fellow of the American Academy of Family Practice and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Emory Ladany, MD, New York. Died October 3, 1986; age 84. He was a 1927 graduate of Eotvos Lorand Tudomanyegyetem Orvostudományi Fakultas, Budapest. Dr Ladany was a Diplomate of the American Board of Dermatology, Inc, and a member of the New York State Dermatological Society, the American Academy of Dermatology, the Society for Investigative Dermatology, the New York County Medical Society, and the Medical Society of the State of New York.

Harry Lebow, MD, West Hempstead. Died November 17, 1986; age 76. Dr Lebow was a 1936 graduate of Rush Medical College, Chicago. He was a Fellow of the American Academy of Family Practice and a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Gerald P. Metzler, MD, Syracuse. Died October 15, 1986; age 89. Dr Metzler was a 1922 graduate of State University of New York Health Science Center at Syracuse. He was a member of the Academy of Medicine, the Onondaga County Medical Society, and the Medical Society of the State of New York.

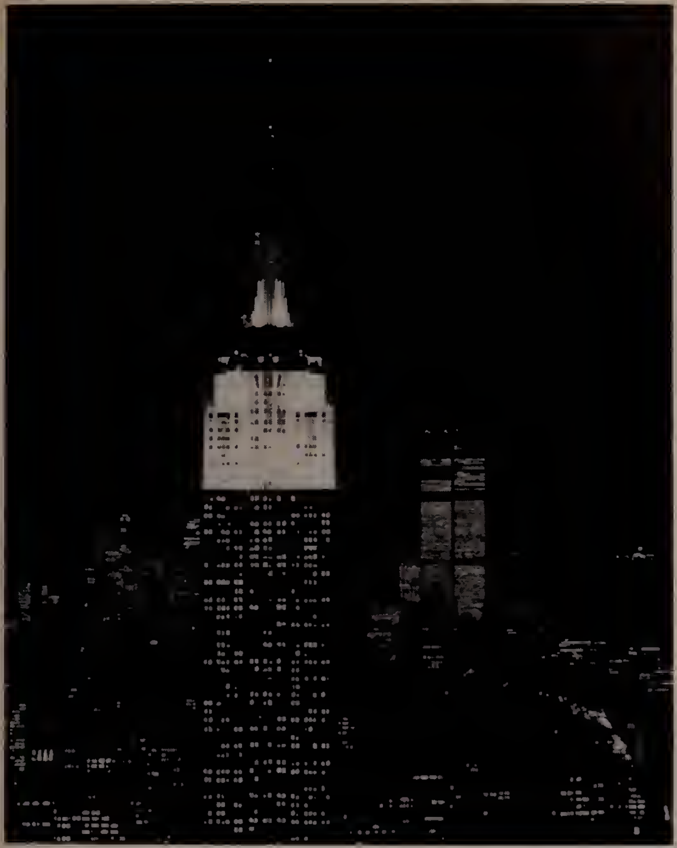
Henry Nowell Reid, MD, Rome. Died November 17, 1986; age 76. Dr Reid was a 1937 graduate of Washington University School of Medicine, St Louis. He was a member of the Academy of Medicine, the American Academy of Family Practice, the International Academy of Proctology, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Archie Scribner, MD, Chappaqua. Died June 21, 1986; age 77. He was a 1937 graduate of Columbia University College of Physicians and Surgeons, New York. Dr Scribner was a member of the Academy of Medicine, the American Rheumatism Association, the New York County Medical Society, and the Medical Society of the State of New York.

Mary C. Tyson, MD, Haworth, New Jersey. Died October 18, 1986; age 75. She was a 1937 graduate of New York University College of Medicine, New York. Dr Tyson was a member of the International Society of Hematology, the New York County Medical Society, and the Medical Society of the State of New York.

William H. Weshta, MD, Brooklyn. Died November 2, 1986; age 70. Dr Weshta was a 1941 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Joseph George Zimring, MD, Long Beach. Died November 20, 1986; age 76. He was a 1936 graduate of the University of Maryland School of Medicine, Baltimore. Dr Zimring was a Fellow of the American Academy of Family Practice. His memberships included the Academy of Medicine, the American Geriatrics Society, the Nassau County Medical Society, and the Medical Society of the State of New York.



COURTESY N.Y. CONVENTION & VISITORS BUREAU

Medical Society of the State of New York

ANNUAL CME ASSEMBLY

Clinical Dilemmas & Controversies

March 13–15, 1987

and the

Annual Meeting of the House of Delegates

March 12–15, 1987

The New York Hilton
New York City

HIGHLIGHTS . . .

- 22 Sessions
- Panel Discussions
- Technical Exhibits
- Two Receptions
- President's Reception & Dinner Dance

CLIP & MAIL

Director of Front Office Operations
The New York Hilton
1335 Avenue of the Americas
New York, N.Y. 10019

Phone: 212-586-7000

for hotel use only

NAME ______________________ TITLE ______________
FIRM ______________________ PHONE ______________
STREET ______________________________________
CITY/STATE/ZIP ______________________________
REMARKS _____________________________________
(please print)

NOTE: Reservations must be received no later than February 19, 1987 and will be held only until 6 p.m. on day of arrival unless later hour is specified.

please print

ARRIVAL DATE ______________________
☐ 2-4 p.m. ☐ 4-6 p.m. ☐ 6-8 p.m. ☐ after 8 p.m.
DEPARTURE DATE ______________________
(checkout time is 1 p.m.)

HAVE A CREDIT CARD? You can register at our QUICK CHECK desk. If you wish to take advantage of this credit card PAYMENT GUARANTEED reservation, please complete the information below.

☐ AMERICAN EXPRESS
☐ DINERS CLUB
☐ CARTE BLANCHE

Expiration Date ______________
____
Credit Card No.

Signature ______________________

MEDICAL SOCIETY OF THE STATE OF NEW YORK
Annual Meeting of the House of Delegates
March 12–15, 1987
Annual CME Assembly
March 13–15, 1987

Circle Preferred rate:

Singles: $130 $145 $155
Doubles/Twins: $155 $170 $180
Executive Tower: $200 Single/Double
Suites: (P&1): $360 $395
Suites: (P&2): $460 $495

Rates subject to 8¼% N.Y. State sales tax; $2.00 per room per night, and 5% N.Y.C. occupancy taxes. (If room at the rate requested is unavailable, one at the nearest available rate will be reserved.)

GENERAL INFORMATION

All physicians, members of the allied professions, and their guests are invited to attend the Medical Society of the State of New York's Annual CME Assembly to be held March 13–15, 1987, at the New York Hilton. The two and one-half days of meetings will provide the individual physician with a possible 18 credits out of a total of 93 Category 1, CME credit hours being offered. The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians. Over 125 outstanding speakers, including a number from out-of-state, will present papers at 20 sessions.

The theme of the CME Sessions will be "Clinical Dilemmas and Controversies." A few of the subjects to be presented will be, Terrorism & Medicine; Computers in Medicine; AIDS in Pregnancy; Humanism and Medical Ethics in Our Technological Age; Intensive Care for AIDS Victims; Thoracic Aortic Balloon Occlusion: Methods and Indications; and the Role of the Physician as the Patient's Advocate. For details, see the Preliminary Program which appears in the December 1986 issue of the *New York State Journal of Medicine.* Our program will offer the clinician timely approaches in diagnosis and treatment of diseases in a wide range of medical specialties.

Here are comments from some of the attendees at the 1986 CME Assembly: "The sessions were educational, and the topics were timely," "There was an excellent choice of qualified speakers," "I found the material was practical, concise and clinically useful." Don't miss this opportunity to gain category 1 credits over a weekend when you are free from office hours. Also, bring your family to New York City to enjoy the sights. There is no registration fee for MSSNY members or members of any other state medical society.

Professional representatives from more than 60 companies will be in the Rhinelander Gallery Exhibit Hall on Friday and Saturday to discuss their products and services. The Technical exhibits will include drug products, office management systems, medical and surgical suppliers, health insurance plans, pension plans and more.

SPECIAL EVENTS

Thursday, March 12

The Annual Dinner Dance, in honor of our President, John Finkbeiner, M.D., will be held in the Trianon Ballroom of the New York Hilton. The reception will be held in the Mercury Ballroom starting at 7:00 p.m. All attendees are welcome. Tickets are $75.00 per person and should be ordered in advance from the Medical Society.

UNITED NATIONS HEADQUARTERS
— *Courtesy N.Y. Convention & Visitors Bureau*

Friday, March 13 and Saturday, March 14

A reception for physicians, their guests, the allied professions and exhibitors will be held both days in the Exhibit Hall between 5:00 p.m. and 6:00 p.m. A free drawing for a color TV set will take place at both receptions.

HOTEL RESERVATIONS

Physicians must make their own reservations with the New York Hilton no later than February 19, 1987. After that date, rooms are on an availability basis only. If making reservations by telephone, please mention that you are attending the Medical Society Convention for billing at the special rate.

REGISTRATION

CME Registration will take place in the Exhibit Hall on Friday, March 13, from 8:00 a.m. to 5:30 p.m., and Saturday, March 14 from 8:30 a.m. to 5:30 p.m., and on Sunday, March 15, registration will take place on the 2nd floor Promenade from 8:30 a.m. to 12:30 p.m.

House of Delegates registration will take place on the 2nd and 3rd floor Promenades at the following hours: Wednesday, March 11, from 12 Noon to 6:00 p.m.; Thursday, March 12, Friday, March 13, and Saturday, March 14, from 8:30 a.m.–5:30 p.m.; and Sunday, March 15, from 8:30 a.m. to 12 Noon. All MSSNY members are invited to attend the meetings.

CME MEETING HOURS

Friday, March 13 —	9am – 12:30pm
	2pm – 5pm
Saturday, March 14	10am – 1pm
	2pm – 5pm
Sunday, March 15	10am – 1pm

EXHIBIT HOURS

Friday, March 13 —	9am – 6pm
Saturday, March 14	9am – 6pm

1987 ANNUAL CME ASSEMBLY

Medical Society of the State of New York
The New York Hilton, New York City

Clinical Dilemmas and Controversies

FRIDAY, MARCH 13

9:15 a.m. - 12:30 p.m. Plenary Session: Clinical and Ethical Dilemmas, *Gramercy A & B*

2:00 p.m. - 5:00 p.m.
Chest Diseases, *Regent Parlor*
Cardiovascular Diseases, *Murray Hill B*
Pediatrics/Orthopedic Surgery, *Murray Hill A*
Neurology/Alcoholism/Physical Medicine & Rehabilitation, *Nassau A*
Emergency Medicine/Critical Care/Otolaryngology, *Gramercy B*
Medical Liability, *Gramercy A*

SATURDAY, MARCH 14

10:00 a.m. - 1:00 p.m.
Internal Medicine (All Day), *Regent Parlor*
Ophthalmology (All Day), *Sutton Parlor Center*
Psychiatry (All Day), *Murray Hill B*
Hand Surgery/Orthopedic Surgery/Physical Medicine & Rehabilitation (All Day), *Murray Hill A*
Urology/Pathology, *Nassau A*
Dermatology, *Nassau B*

2:00 p.m. - 5:00 p.m.
Internal Medicine, *Regent Parlor*
Ophthalmology, *Sutton Parlor Center*
Psychiatry, *Murray Hill B*
Hand Surgery/Orthopedic Surgery/Physical Medicine & Rehabilitation, *Murray Hill A*
Computers in Medicine, *Madison Suite*
Terrorism and Medicine, *Sutton Parlor South*

SUNDAY, MARCH 15

10:00 a.m. - 1:00 p.m.
Allergy and Immunology, *Murray Hill B*
General Surgery/Radiology/Anesthesiology, *Murray Hill A*
Obstetrics and Gynecology/Women's Medical Society of New York State, *Gramercy B*
Family Practice/Orthopedic Surgery, *Nassau A*
Neurosurgery, *Morgan Suite*

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis. The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians.

◆

Technical Exhibits

The Brooklyn Bridge (foreground) and the Manhattan Bridge spanning the East River

Advance Registration Form

1987 Annual CME Assembly, March 13–15
The New York Hilton, New York City

Complete and return this form to the Medical Society address as indicated below.

Name____
PLEASE PRINT LAST FIRST

Address______________________________________

City/State/Zip__________________________________

Medical Specialty_______________________________

REGISTRATION FEE *Please check categories*

☐ Non-Member Physician* $50.00
☐ Allied Profession $10.00

☐ Nurse
☐ Physician Assist.
☐ Dentist

☐ Lab Technician
☐ PT/OT
☐ Other______________
Specify

NO FEE *Please check category*

☐ Active Member*
☐ MSSNY Life Member
☐ Resident/Intern

☐ Medical Students
☐ Medical Assist.
☐ Speaker

☐ Spouse/Family
☐ News Media/Medical Writer

REMITTANCE ENCLOSED $______________

* of any state medical society

Please make check payable to:
Medical Society of the State of New York, and return this form with remittance to: Division of Meeting Services/MSSNY, 420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042.

Important: Your ID Badge will be held at the MSSNY Registration Desk located in the Exhibit Hall, on the second floor of the New York Hilton.

(*Meetings continued from p 14A*)

ha. Contact: Marge Adey, University of Nebraska Medical Center, Center for Continuing Education, 42nd and Dewey Ave, Omaha, NE 68105. Tel: (402) 559-4152.

NORTH DAKOTA

Mar 14. **The Aging Nervous System: Dementia and Behavioral Changes in the Elderly.** Contact: Dr Harry A. Whitaker, The Neuropsychiatric Institute, 700 First Ave S, Fargo, ND 58107. Tel: (701) 235-5354.

TEXAS

Mar 4–6. **1987 Pan American Allergy Society Training Course and Seminar.** Four Seasons Hotel, San Antonio. Info: Betty Kahler, Pan American Allergy Society, 229 Parking Way, Lake Jackson, TX 77566. Tel: (409) 297-8964.

UTAH

Mar 1–6. **Primary Care—Current Diagnosis & Treatment.** 25 Cat 1 Credits. Park City. Info: Merritt Peralta Health Education Center, 400 Hawthorne Ave, Oakland, CA 94609. Tel: (415) 420-6161.

Mar 1–7. **Update '87—Office Obstetrics and Gynecology.** Shadow Ridge Resort, Park City. Info: Scott and White, 2401 South 31 St, Temple, TX 76508. Tel: (817) 774-4073.

Mar 11–13. **9th Annual Common Problems in Pediatrics.** Salt Lake City. Info: Katharine C. Blosch, Preferred Meeting Management, Inc, 565 E 2100S, Salt Lake City, UT 84106. Tel: (801) 466-3500.

AROUND THE WORLD

CANADA

Mar 5–7. **30th Anniversary of the British Columbia Heart Foundation Cardiac Symposium.** Hyatt Regency Hotel, Vancouver, British Columbia. Info: BC Heart Foundation Symposium, Venue West Conference Management Services, #801-750 Jervis St, Vancouver, BC V6E 2A9. Tel: (604) 681-5226.

Medical Society of the
State of New York

Annual Dinner Dance

in honor of
John A. Finkbeiner, M.D.
President

Thursday
March 12, 1987
Trianon Ballroom
The New York Hilton
53rd Street & Avenue of Americas

Cocktail Hour
7 P.M.

Superlative
Dinner

Subscription $75.00 per person

Reservation Form

Make check payable to:
Medical Society of the State of New York
Attn: Div. of Management & Meeting Services
420 Lakeville Road, P.O. Box 5404
Lake Success, New York 11042

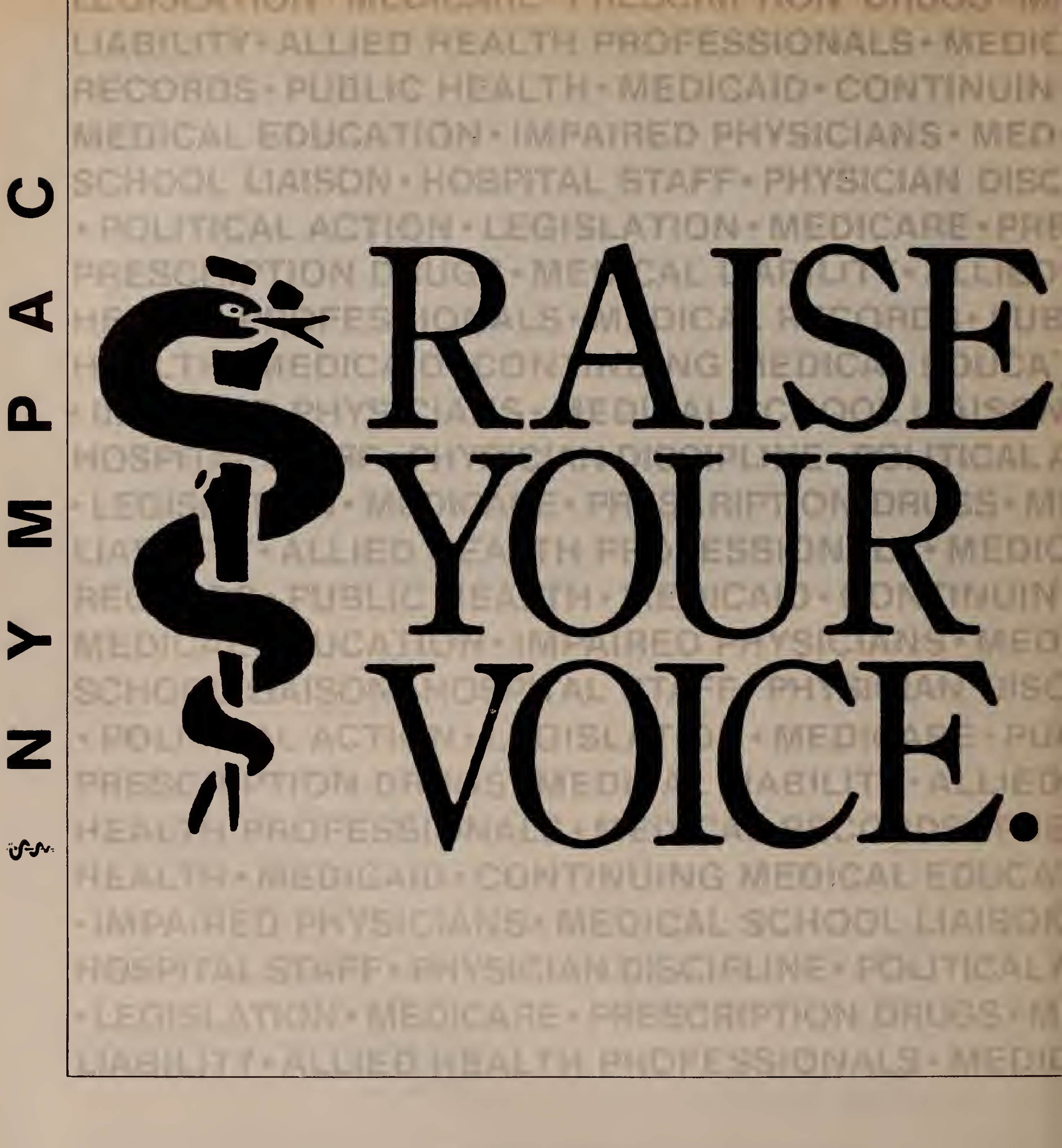
Please send me ______ tickets for the
Annual Dinner Dance on March 12, 1987
Check enclosed for $________

PLEASE NOTE: Reservations and ticket sales will close at 12 Noon on Thursday, March 12, one hour before the House Convenes.

Name____________________________________

Address__________________________________

________________________Zip______________



New York Medical Political Action Committee
An Arm of the Medical Society of the State of New York
420 Lakeville Road, Lake Success, NY 11042 • 516-488-6100

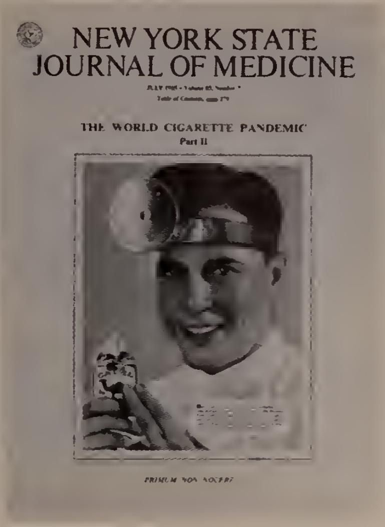
Visit Our Exhibit at the CME Assembly

Political Action for Physician

Essential reading for every physician

NEW YORK STATE JOURNAL OF MEDICINE

THE WORLD CIGARETTE PANDEMIC


NEW YORK STATE JOURNAL OF MEDICINE

THE WORLD CIGARETTE PANDEMIC
Part II

NEW YORK STATE JOURNAL OF MEDICINE

CIGARETTE SMOKING, TOBACCO, AND HEALTH
INTERNATIONAL PERSPECTIVES

The December 1983, July 1985 and September 1986 issues of the **New York State Journal of Medicine** constitute the most comprehensive coverage on the subject of smoking ever published by a medical journal.

SEND A COPY TO COLLEAGUES AND MEDICAL STUDENTS in the US and abroad who might not normally receive the Journal. Or order an extra copy for your patients and yourself.

Extra press runs have enabled the New York State Journal of Medicine to offer single or bulk copies of its issues on smoking. As the official publication of the Medical Society of the State of New York since 1900, the Journal is one of the foremost independent peer-reviewed general medical journals in the country. Providing timely and stimulating commentary, challenging research papers, analysis of clinical trends, debate on topical issues, and in-depth coverage of medical news, the Journal makes important reading—and a welcome gift—to physicians and students in the United States or abroad. A free copy of the July 1985 issue will be included with each subcription.

The Following, Reduced Rates Are Now Being Offered On The Smoking Issues

Quantity	**Per Copy***
1–24	$4.00
25–49	$3.50
50–99	$3.00
100–149	$2.50
150–500	$2.00

ORDER FORM

Name____

Address____

City/State/Zip____________________________________

Please send ________ copies of December 1983, ________ copies of July 1985, ________ copies of September 1986 issue of the Journal.

Enter my subscription to the Journal. ☐ $25.00 U.S. & Canada; ☐ $33.00 Foreign

Total amount enclosed $ __________ ***Note:** Domestic postage included. Please add New York state & local sales tax where applicable, or sales tax exemption certificate. Foreign postage additional.

Mail to: Circulation Department, **New York State Journal of Medicine**
420 Lakeville, Rd., P.O. Box 5404, Lake Success, N.Y. 11042.

MEDICAL OFFICE SPACE FOR LEASE

2 ½ Blocks from St. Vincent's Medical Center

222 W. 14th St., N.Y.C. Between 7th & 8th Avenues

1000 to 9600 SQUARE FEET

Ground Floor & Mezzanine Available

- NEW HI-RISE CONDO BLDG
- EXCELLENT BUS & SUBWAY ACCESS
- PARKING GARAGE IN BUILDING
- AVAILABLE SPRING-1987

For additional information or inspection contact:

COLDWELL BANKER
COMMERCIAL REAL ESTATE SERVICES

COLDWELL BANKER
COMMERCIAL REAL ESTATE SERVICES
Emanuel A. Campisi • Paul Kutscera
(212) 223-7171
437 Madison Avenue, New York, N.Y. 10022

MISCELLANEOUS

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

ARE YOU HAVING A PROBLEM RELATING TO CHEMICAL DEPENDENCE? If your answer is affirmative, contact Susan Kaplan, Attorney-at-Law, (212) 877-5998. Practice limited to legal problems directly involving your license to practice medicine—with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professionals), and as an Assistant District Attorney in Nassau County. Susan Kaplan, Esq., 165 West End Avenue, Suite 27P, New York, N.Y. 10023.

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuarial certification and employee statements of participation—partnership agreements and professionals incorporation—No insurance required—references upon request—Wachstock and Dienstag Attorneys at Law, 122 Cutter Mill Road, Great Neck, NY 11021 (516) 773-3322.

FIRM OF GERALD GOLDBERG, CPA-ATTORNEY, formerly with IRS, announces his availability to prepare taxes and plan under changing tax environment, accounting, tax appeals, legal representation on practice transfers. (212) 406-1156 and (516) 565-6260.

MISCELLANEOUS—CONT'D

HAVE YOU RECEIVED AN INQUIRY FROM THE OFFICE OF PROFESSIONAL MEDICAL CONDUCT? For consultation, contact Susan Kaplan, Attorney-at-Law, (212) 877-5998. Practice limited to the crucial legal problems directly affecting your license to practice medicine—with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professions), and as an Assistant District Attorney in Nassau County. Susan Kaplan, Esq., 165 West End Avenue, Suite 27P, New York, N.Y. 10023.

50% OFF PREVIOUSLY OWNED MEDICAL— Laboratory, x-ray, ultrasound equipment in excellent condition. We buy, sell, broker, repair. Ask about our Holter Scanning Services. Call anytime: Medical Equipment Resale, 24026 Haggerty, Farmington Hills, MI 48018. (313) 477-6880.

ATTORNEY David E. Ruck, former chief, Criminal Division, office of the New York State Deputy Attorney General for Medicaid Fraud Control is pleased to announce the opening of his office for the practice of law at 230 Park Ave., New, York, NY 10169 (212) 661-8866.

CRAIG PERSONNEL AGENCY, INC., Medical placement specialists: medical office managers, secretaries, assistants and technicians; full-time, part-time, temp. NYC. (212) 302-8686, 25 West 43 Street, NY, NY. Queens (718) 225-4379, 40-18 Bell Blvd., Bayside, NY.

COMPUTERIZE YOUR BILLING—Remarkably easy-to-use software automatically prints bills, statements, insurance forms. Menu-driven. Reports ageing balances, referral sources, income by time period, procedure & more. Installs automatically. IBM and compatibles. Solo practice only $399. Group practice, only $499. Free information. Demonstration disk with complete manual, $10. Call or write REM Systems, Inc., Dept. NY2, 281 Dug Hill Road, Brownsboro, AL 35741 (205) 534-3457. VISA, MC Accepted.

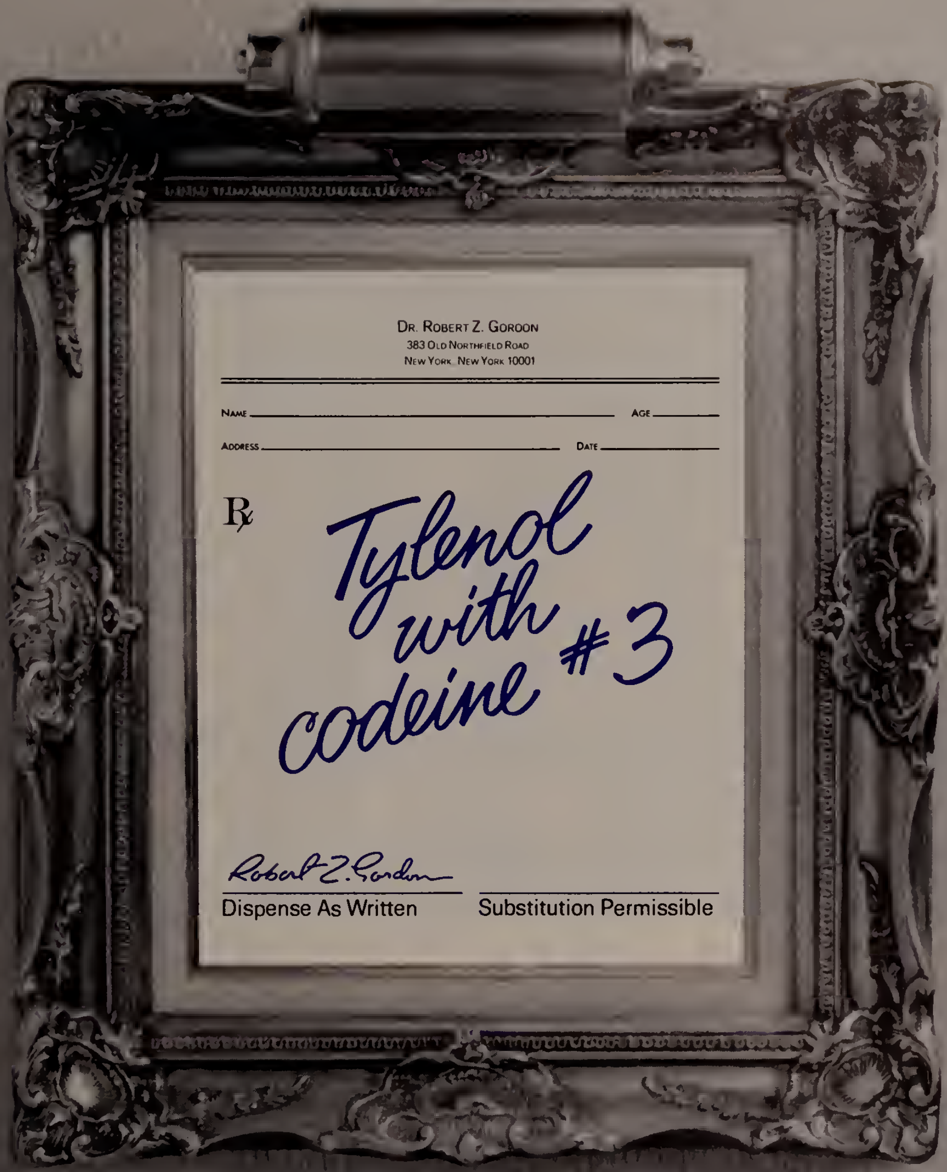
MISCELLANEOUS—CONT'D

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta, GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

WEIGHT LOSS TUTORIAL, NO FEE for physicians and their staff. Individual help in setting up a medically documented, effective, economical, uncomplicated weight loss program in your office practice. Contact Allan Lazar, M.D., 420 Grand Avenue, Englewood, N.J. 07631. (201) 568-3742.

EXPERIENCED MEDICAL DISCIPLINARY ATTORNEY. Litigator/trial attorney experienced in representing physicians at hearings before the Department of Health and the Office of Professional Medical Conduct. Michael S. Kelton, Esq., c/o Lippman & Krasnow, P.C., 475 Fifth Avenue, Suite 1212, New York, N.Y. 10017, telephone number (212) 532-0710.

PROFESSIONAL MISCONDUCT ATTORNEYS. William L. Wood Jr., formerly Executive Director of the New York State Office of Professional Discipline and Anthony Z. Scher, formerly Director of Prosecutions, announce the opening of their law office, Wood & Scher. Our recent tenure as chief enforcement officers has given us experience which will allow us to represent physicians in professional misconduct proceedings, malpractice licensure, license restoration, controlled drug proceedings, reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practitioners. Wood and Scher, Attorneys at Law, One Chase Road, Scarsdale, New York 10583. Telephone 914-723-3500.



Dr. Robert Z. Goroon
383 Old Northfield Road
New York, New York 10001

Name ____________________ Age ________

Address ____________________ Date ________

℞ Tylenol with codeine #3

Robert Z. Gordon
Dispense As Written

Substitution Permissible

CREATE A SIGNED ORIGINAL

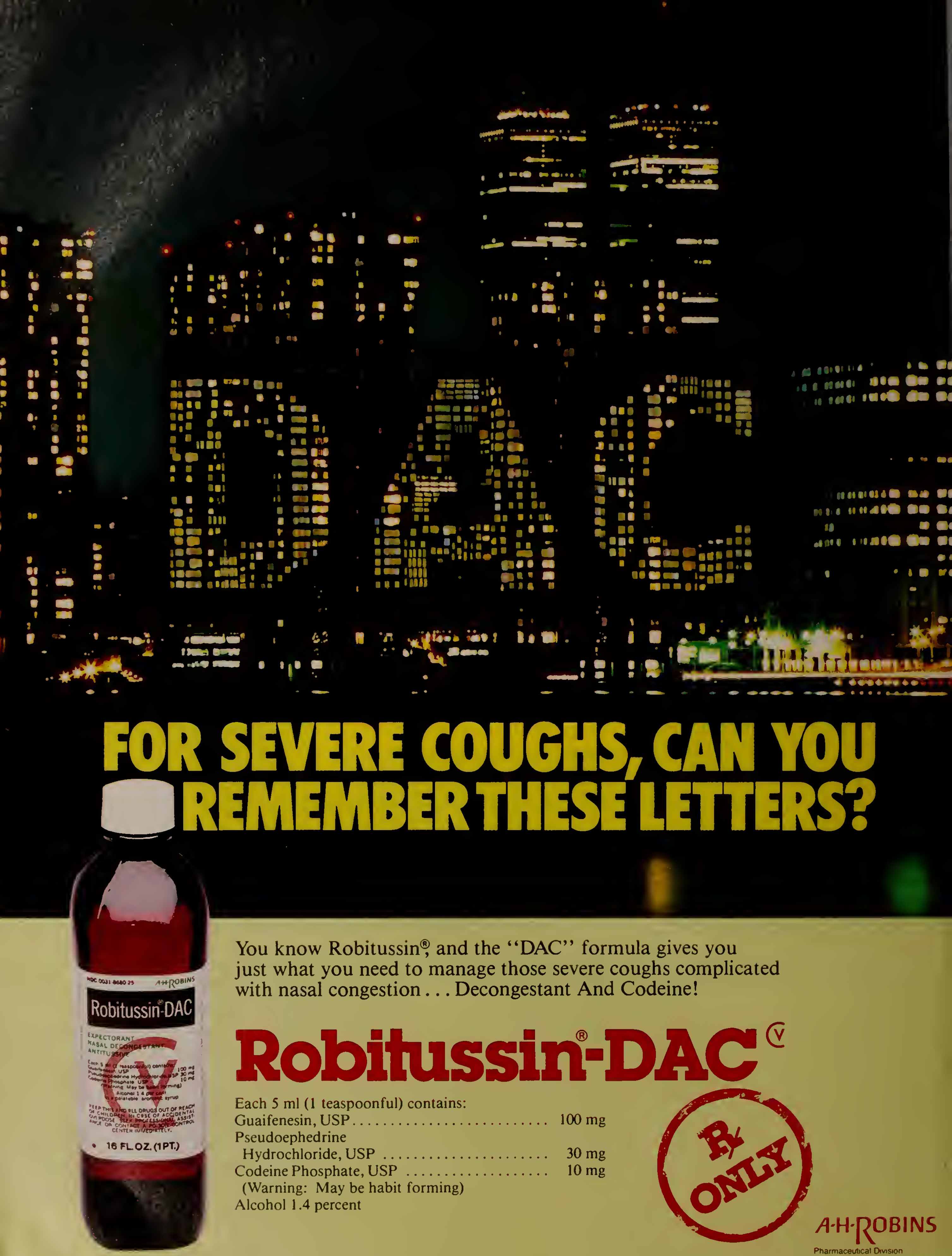
to be sure your patients get...

Tylenol®
ACETAMINOPHEN
with codeine
phosphate
TABLETS Ⓒ-III ELIXIR Ⓒ-V

Tablets: Contain Codeine Phosphate*: No. 3 — 30 mg;
No. 4 — 60 mg — plus Acetaminophen 300 mg.
Elixir: Each 5 mL contains 12 mg Codeine Phosphate* plus
120 mg Acetaminophen (Alcohol 7%).
*Warning: May be habit forming.

© McNEILAB, INC., 1987

TYCT 6445-A



FOR SEVERE COUGHS, CAN YOU REMEMBER THESE LETTERS?

You know Robitussin®, and the "DAC" formula gives you just what you need to manage those severe coughs complicated with nasal congestion . . . Decongestant And Codeine!

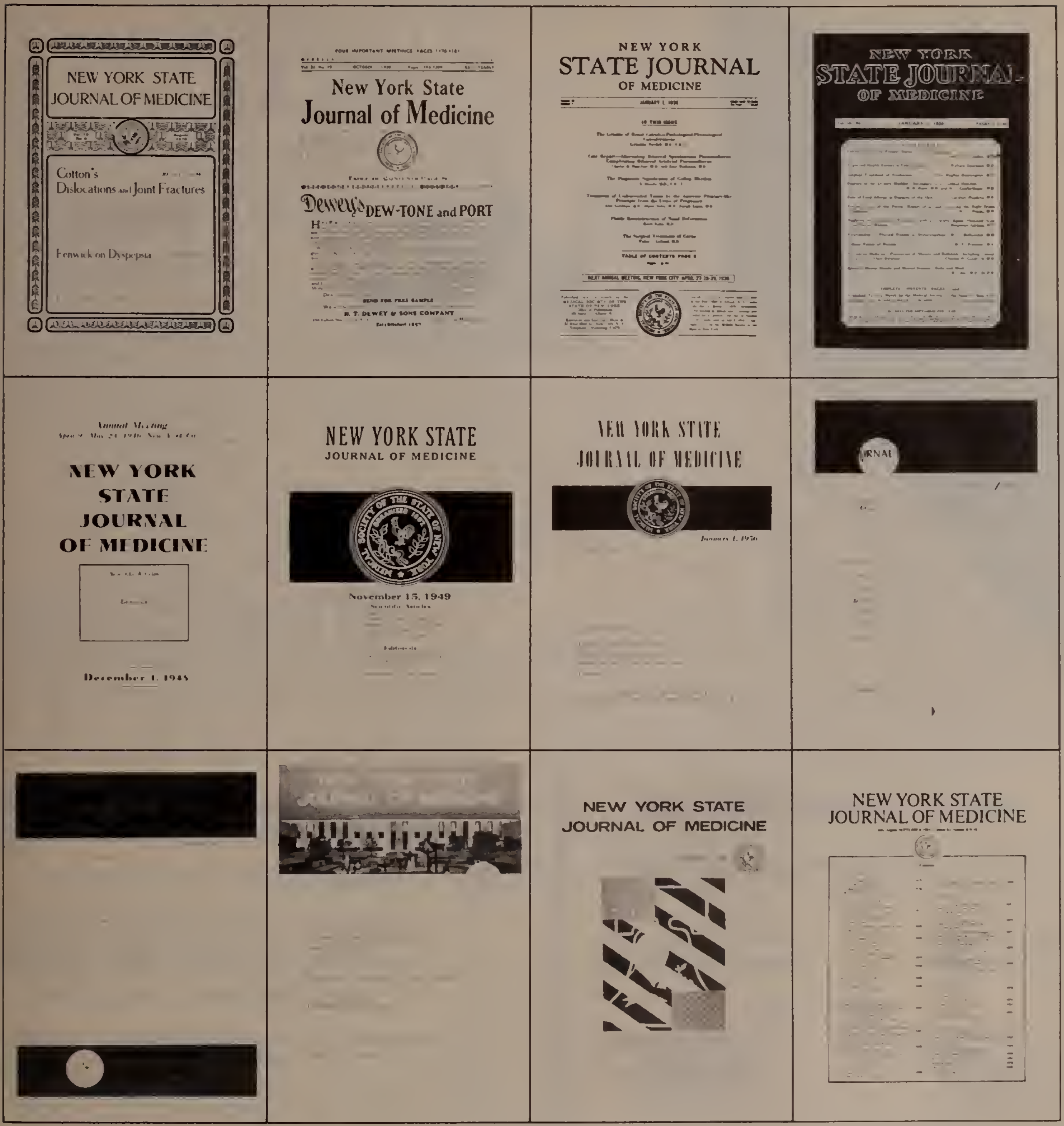
Robitussin®-DAC ⓥ

Each 5 ml (1 teaspoonful) contains:	
Guaifenesin, USP	100 mg
Pseudoephedrine Hydrochloride, USP	30 mg
Codeine Phosphate, USP	10 mg
(Warning: May be habit forming)	
Alcohol 1.4 percent	

℞ ONLY

A-H-ROBINS
Pharmaceutical Division
Richmond, Virginia 23261-6609

Cover to cover . . . . always good reading.


NEW YORK STATE JOURNAL OF MEDICINE
Cotton's Dislocations and Joint Fractures
Fenwick on Dyspepsia
New York State Journal of Medicine
Dewey's DEW-TONE and PORT
B. T. DEWEY & SONS COMPANY
NEW YORK STATE JOURNAL OF MEDICINE
NEW YORK STATE JOURNAL OF MEDICINE
NEW YORK STATE JOURNAL OF MEDICINE
December 1, 1945
NEW YORK STATE JOURNAL OF MEDICINE
November 15, 1949
NEW YORK STATE JOURNAL OF MEDICINE
NEW YORK STATE JOURNAL OF MEDICINE
NEW YORK STATE JOURNAL OF MEDICINE

In 1987, the *New York State Journal of Medicine* promises to provide timely and stimulating commentary, challenging research papers, analysis of clinical trends, debate on topical issues, and in-depth coverage of medical news. The journal will make a welcome gift to a colleague or student in the United States or abroad.

A free copy of the July 1985 issue, a special issue on smoking, will be included with each subscription while the supply lasts.

SUBSCRIPTION ORDER FORM Clip and mail

Circulation Department
New York State Journal of Medicine
420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042

Name______________________________

Address______________________________

____

	1 Year	2 Years	3 Years
U.S. & Canada	☐ $25	☐ $45	☐ $60
Foreign	☐ $30	☐ $55	☐ $75

Payment required with order

CRAIG MEDICAL AGENCY, INC.
Affiliated with Health Care Since 1964

Medical Placement Specialists
Full Time, Part Time, Temporary
Medical Office Managers/Secretaries/
Assistants/RN's/Technicians/
Receptionists

Manhattan: 25 West 43rd Street, NY, NY 10036
(212) 302-8686
Visit Booth 32
March 13-14
New York
Hilton

Portfolio Management As Individual As You Are.

Whether your financial objective is maximum income or maximum growth, the Private Clients Group individually manages your portfolio for maximum results–profitable performance for you. Our dedicated investment research team and full business integration in support of the private investor make us unique. No one serves the individual investor better.

To discuss your portfolio of $500,000 or more, write or call Caroline M. Calder, Marketing Officer, Private Clients Group, 280 Park Avenue, New York, N.Y. 10017. Tel. 212/850-1473.

© 1986 Bankers Trust Company
Member FDIC

Bankers Trust Company
Private Clients Group
We Make Money For People Who Make Money

AIDS SYMPOSIUM

Mt. Sinai Medical Center, New York City
Saturday, April 4, 1987 9 am—6 pm

Peter Herman, M.D.
Course Director

David M. Simpson, M.D.
Associate Course Director

Topics Included:
1. Infectious Diseases
2. Epidemiology
3. Virology
4. Neurology
5. Mental Health
6. Oncology
7. Pediatrics
8. Therapeutics

Guest Speakers from NIH, Mass. General Hospital, University of California, CDC Georgia, Faculty of Mt. Sinai Medical Center and Salpetriere Hospital, Paris, France.

Fees: $75 ($25 for Residents, Interns and Medical Students)

Credit Hours: 8

Send Check to: Peter Herman, M.D.
AIDS SYMPOSIUM
Mt. Sinai Medical Center
P.O. Box 1282
New York, New York 10029

4TH ANNUAL BONE MARROW TRANSPLANTATION SYMPOSIUM

March 12, 1987—3:00 to 7:00 P.M.

Sponsored by: Division of Neoplastic Diseases
New York Medical College, Valhalla, NY 10595

Credits: CME Category I, 3 hours, no fee

Information contact Tauseef Ahmed, M.D., Director
(914) 993-4400

DORSCH PROSTHETICS AND ORTHOTICS, INC.

Now with two locations:

New York City—109 East 29th St., New York, NY 10016
Westchester—200 East Post Rd., White Plains, NY 10601

PROUDLY SERVING THE NEEDS OF THE MEDICAL COMMUNITY SINCE 1898

Visit Booth 68, March 13–14, New York Hilton

Classified Advertising

New York State Journal of Medicine

GENERAL INFORMATION

All classified advertisements are payable in advance, except for government agencies and advertising agencies; a 15% commission to recognized advertising agencies.

Frequency: Monthly
Mailing date: 7th of the month
Closing date: 30 days before mailing date

Circulation: 27,000 (mainly physicians & medical libraries)

DISPLAY ADVERTISING

	1 time	*6 time*	*12 time*	*Width*		*Depth*
1/4-page	$540	$480	$420	3-1/2″	×	4-7/8″
1/6-page	252	228	204	2-1/8″	×	4-7/8″
1/8-page	228	204	180	3-1/2″	×	2-3/8″
1/16-page	114	102	90	3-1/2″	×	1-1/8″
1/18-page	84	72	60	2-1/8″	×	1-1/2″

PARAGRAPH ADVERTISING

The first several words are set in bold face caps.

Rates: $35.00 per insertion for 50 words or less; additional words are 40 cents each. On request, Dept. numbers will be assigned by the Journal for replies to be forwarded to the advertiser. Dept. numbers are 50 cents per insertion. There is no frequency discount for paragraph ads.

Counting Words: Two initials, each abbreviation, figures consisting of 5 numerals or less, are counted as separate words. For replies, your name and address, or telephone number should appear at the end of the ad and be counted in the total number of words.

For further information, please call the Advertising Department at 516-488-6100.

CLASSIFIED ADVERTISING ORDER FORM

New York State Journal of Medicine
Attn: Classified Advertising
420 Lakeville Rd., P.O. Box 5404
Lake Success, N.Y. 11042

Date__________________

Name____________________________________

Address__________________________________

City/State/Zip____________________________

Payment enclosed: $__________

Number of Insertions: __________

Assign Dept. Number: __________

Please make check payable to New York State Journal of Medicine.

Don't play games with your lungs.

Your lungs are an incredible piece of equipment. But they're also incredibly delicate. Every time you smoke, you damage your lungs. Puff after puff, day after day, your lungs get weaker and weaker until finally, they're useless. Like a flat football. Don't let that happen to your precious lungs.

—*Eric Dickerson*

TAKE CARE OF YOUR LUNGS. THEY'RE ONLY HUMAN.

AMERICAN LUNG ASSOCIATION®
The Christmas Seal People®

Doctor . .

Do you, or a physician you know, have a problem with alcohol, drugs, or emotional illness?

If so, please contact the Medical Society of the State of New York's Committee for Physicians' Health, whose function is to assist, *confidentially.*

Telephone: 516-488-7777.

Index to Advertisers

Ayerst Laboratories 4A, 5A, 6A, 31A, 32A, 33A, 34A
Bankers Trust Company 28A
St. Clare's Hospital 7A
Classified Advertising 8A, 24A, 28A
Coldwell Banker 24A
Craig Personnel Agency 28A
Dorsch Prosthetics and Orthotics, Inc. 28A
Knoll Pharmaceuticals 11A, 12A, 13A
Eli Lilly and Company 16A
Marion Laboratories 1A, 2A
McNeil Laboratories 25A
Physician's Reciprocial Insurers 10A
A. H. Robins Company 9A, 26A
Roche Laboratories . . . . 3rd & 4th Cover
Summit Health Ltd. 14A
Upjohn Company . . . . 2nd Cover

To show you how many hypertensives stayed on

INDERAL® LA
(PROPRANOLOL HCl)

after a major nationwide trial...

...we had
to find
just the
right room.

60,073 patients (90%) who started on INDERAL® LA stayed on INDERAL LA.[1*]

Surprising? Not really.

Because most patients on INDERAL LA (propranolol HCl) don't even know it's working.

A recent double-blind, placebo-controlled, crossover study in 138 hypertensive patients[2] revealed that INDERAL LA has a side effects profile unsurpassed by atenolol or metoprolol—which shows how well-tolerated once-daily INDERAL LA can be.

Sole therapy or concomitant therapy? Fifty-nine percent of the time, INDERAL LA stood on its own.

The patients in the nationwide compliance trial were no different from yours. Generally when the antihypertensive regimen is complicated, compliance may become a problem. So, the effectiveness of INDERAL LA as once-daily monotherapy is a big plus. Of the remaining hypertensives in the program, 36% were controlled merely with the addition of a diuretic to INDERAL LA.

For the noncompliant patients in your practice, INDERAL LA may well be the answer.

Almost 20,000 of the patients in the nationwide compliance trial were identified as having been noncompliant with their previous antihypertensive therapy. Their physicians reported that 88% showed improved compliance when placed on once-daily INDERAL LA.

Control, comfort, and compliance

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl) LONG ACTING CAPSULES

Like conventional INDERAL Tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree, and bronchial asthma.

*After a 30-day trial with INDERAL LA, physicians reported that 90% of the patients would remain on INDERAL LA.

The one you know best keeps looking better

Please see next page for brief summary of prescribing information.

The one you know best keeps looking better

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride. Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules.

CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately.

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate. Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours. When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets. The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially.

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period.

The mechanism of the antihypertensive effect of INDERAL has not been established. Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain. Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use. Effects on plasma volume appear to be minor and somewhat variable. INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients.

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction. Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period. The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity.

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential. The significance of the membrane action in the treatment of arrhythmias is uncertain.

The mechanism of the antimigraine effect of propranolol has not been established. Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain.

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient. But there are also situations in which sympathetic stimulation is vital. For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved. In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction. Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm.

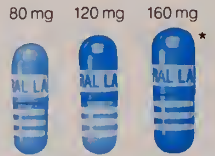
Propranolol is not significantly dialyzable.

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl)

LONG ACTING CAPSULES



INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension; it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies.

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris.

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache. The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use.

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope. INDERAL LA also improves exercise performance. The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary.

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL.

WARNINGS. CARDIAC FAILURE: Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible).

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema) — PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

MAJOR SURGERY: The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e.g., dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA: Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes. In these patients, it may be more difficult to adjust the dosage of insulin.

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case, this resulted after an initial dose of 5 mg propranolol.

PRECAUTIONS. *General:* Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies.

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

Clinical Laboratory Tests. Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug.

Pregnancy: Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose.

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers: INDERAL is excreted in human milk. Caution should be exercised when INDERAL is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy.

Cardiovascular: bradycardia, congestive heart failure, intensification of AV block, hypotension, paresthesia of hands, thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type.

Central Nervous System: lightheadedness, mental depression manifested by insomnia, lassitude, weakness, fatigue, reversible mental depression progressing to catatonia, visual disturbances, hallucinations; an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics.

Gastrointestinal: nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

Allergic: pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress.

Respiratory: bronchospasm.

Hematologic: agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura.

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported.

Miscellaneous: alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily. If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained. INDERAL LA should not be considered a simple mg for mg substitute for INDERAL. INDERAL LA has different kinetics and produces lower blood levels. Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval.

HYPERTENSION — *Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic. The dosage may be increased to 120 mg once daily or higher until adequate blood-pressure control is achieved. The usual maintenance dosage is 120 to 160 mg once daily. In some instances a dosage of 640 mg may be required. The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks.

ANGINA PECTORIS — *Dosage must be individualized.* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained. Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily. In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established.

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS).

MIGRAINE — *Dosage must be individualized.* The initial oral dose is 80 mg INDERAL LA once daily. The usual effective dose range is 160-240 mg once daily. The dosage may be increased gradually to achieve optimum migraine prophylaxis. If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued. It may be advisable to withdraw the drug gradually over a period of several weeks.

HYPERTROPHIC SUBAORTIC STENOSIS — 80-160 mg INDERAL LA once daily.

PEDIATRIC DOSAGE — At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use.

*The appearance of these capsules is a registered trademark of Ayerst Laboratories.

REFERENCES:

1. INDERAL LA National Compliance Evaluation Program. Data on file, Ayerst Laboratories.
2. Ravid M, Lang R, Jutrin I: The relative antihypertensive potency of propranolol, oxprenolol, atenolol, and metoprolol given once daily. *Arch Intern Med* 1985;145:1321-1323.

6412/187

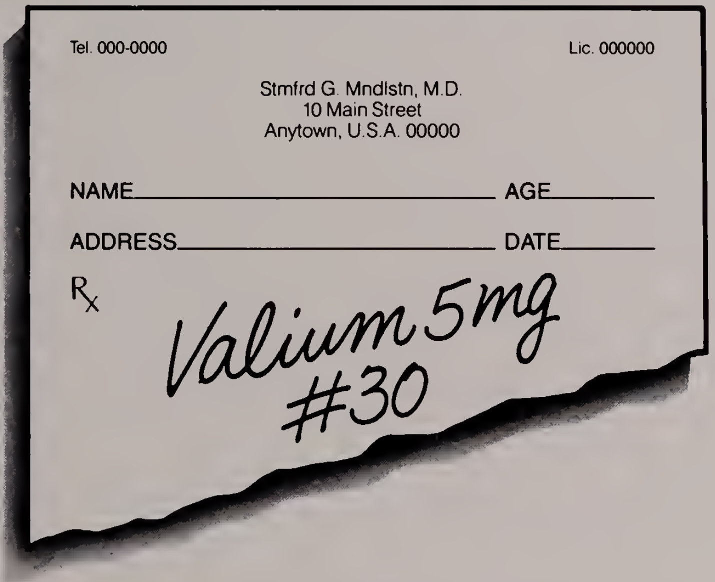
Ayerst®
AYERST LABORATORIES
New York, NY 10017

© 1986 Ayerst Laboratories

Do Not Substitute

VALIUM® (IV)
brand of
diazepam/Roche

The One You Know Best



Tel. 000-0000 Lic. 000000

Stmfrd G. Mndlstn, M.D.
10 Main Street
Anytown, U.S.A. 00000

NAME ______ AGE ______

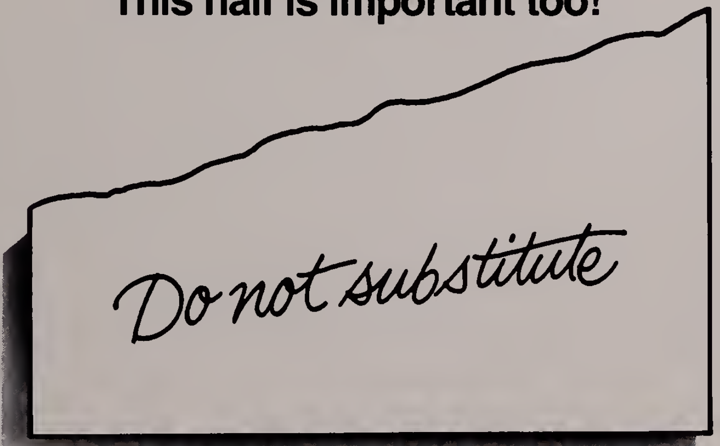
ADDRESS ______ DATE ______

℞

Valium 5mg
#30

Are you writing only half a prescription for it?

This half is important too!



Do not substitute

Be sure to write a complete prescription.

Specify "Dispense as written" or "Do not substitute" or "Medically necessary."

ROCHE®
Roche Products Inc.
Manati, Puerto Rico 00701

ROCHE

BECAUSE ONLY VALIUM

IS ALWAYS VALIUM

IS ALWAYS VALIUM IS ALWAYS VALIUM

IS ALWAYS VALIUM

IS ALWAYS VALIUM IS ALWAYS VALIUM

IS ALWAYS VALIUM

IS ALWAYS VALIUM IS ALWAYS VALIUM

IS ALWAYS VALIUM

IS ALWAYS VALIUM IS ALWAYS VALIUM

IS ALWAYS VALIUM

IS ALWAYS VALIUM IS ALWAYS VALIUM

IS ALWAYS VALIUM®

brand of diazepam/Roche Ⓘ

REMEMBER TO WRITE "DO NOT SUBSTITUTE."
IT'S THE ONE YOU KNOW BEST.

Copyright © 1987 by Roche Products Inc. All rights reserved.

The cut out "V" design is a registered trademark of Roche Products Inc.

STACKS

NEW YORK STATE JOURNAL OF MEDICINE

MARCH 1987 • Volume 87, Number 3

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA
M. D. S.
MAR 23 1987

Contents

COMMENTARIES

Parkinson disease 139
JOSEPH H. FRIEDMAN, MD

Mesenteric cysts 140
PHILIP CAROPRESO, MD

Adverse effects of cyclosporine A on the microcirculation of the cold preserved kidney 141
DAVID ANAISE, MD; WAYNE WALTZER, MD; ANGELO N ARNOLD, PhD; FELIX T. RAPAPORT, MD

RESEARCH PAPER

Urinary calcium excretion in nonfamilial hyperparathyroidism 143
MURRAY LAS, MD, URIEL S. BARZEL, MD

REVIEW ARTICLES

Update on Parkinson disease 147
ABRAHAM N LIEBERMAN, MD

Evaluation and management of chronic aspiration 154
ANDREW BLITZER, DDS, MD

HISTORY

Arthur Donaldson Smith, MD: Physician, explorer, naturalist, and diplomat—Part II 161
PASCAL JAMES IMPERATO, MD

CASE REPORTS

Mesenteric cyst 177
VINCENT CALDAROLA, MD, JOHN LaDUCA, MD

Ethylene glycol intoxication: A new stage in the clinical syndrome 179
STEWART A. FACTOR, DO, NEIL S. LAVA, MD

Acute disseminated intravascular coagulation as a complication of systemic lupus erythematosus 181
LESLIE DUBIN KERR, MD; HARRY SPIERA, MD; LOUIS N ALEDORT, MD

LETTERS TO THE EDITOR

Male to female transmission of AIDS 184
HERBERT BERGER, MD

Emergency medical services 184
JULIUS E STOLFI, MD

A "pack of trouble" for smoking drivers 184
KIMBERLY HALL FERNANDEZ, MD; FRANK M TAMARIN, MD; ROBERT D BRANDSTETTER, MD

Breast carcinoma in a man following local trauma 186
MARIO C RAVIGLIONE, MD, PETER E GRAHAM, MD

BOOK REVIEWS 188

NEWS BRIEFS 192

GUIDELINES FOR AUTHORS 194

MEDICAL MEETINGS AND LECTURES 7A

Philadelphia
19 S. 22nd St. Dec. 87
Phila, PA 19103

Motrin® 800 TABLETS mg
ibuprofen

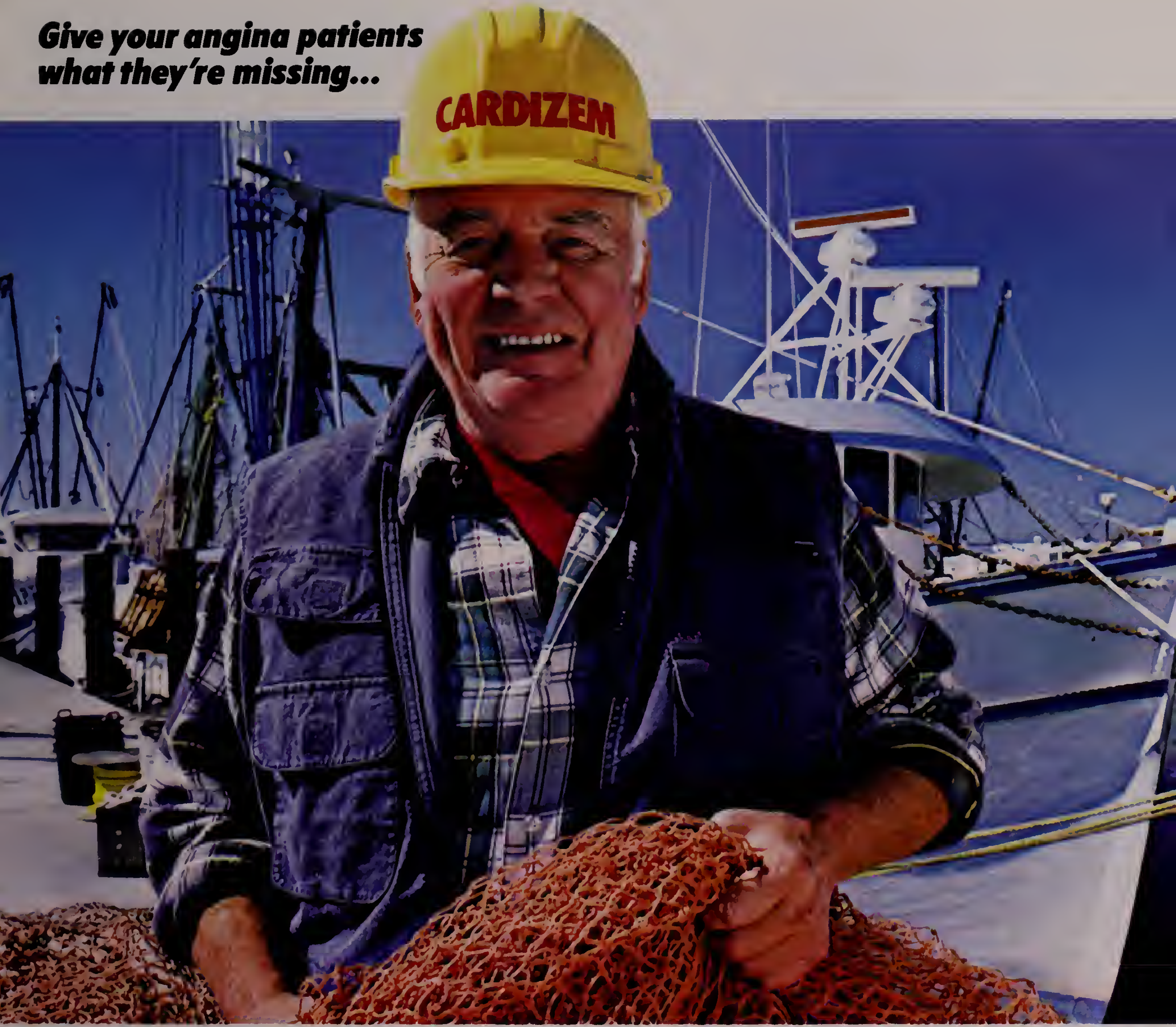
Extra strength
Convenience
Economy

Upjohn
100
A Century
of Caring
1886·1986

©1986 The Upjohn Company

J-6138 January 1986

Give your angina patients what they're missing...



CARDIZEM®: FEW SIDE EFFECTS

diltiazem HCl/Marion

- ***Antianginal action includes dilatation of coronary arteries, a decrease in vascular resistance/afterload, and a reduction in heart rate***
- ***Proven efficacy when used alone in angina[1]***
- ***Compatible with other antianginals[2,3]****
- ***A safe choice for angina patients with coexisting hypertension, asthma, COPD, or PVD[4,5]***

****See Warnings and Precautions.***

Please see brief summary of prescribing information on the next page.

CARDIZEM® diltiazem HCl/Marion FEW SIDE EFFECTS IN ANTIANGINAL THERAPY

60 mg tid or qid

Brief Summary
Professional Use Information

CARDIZEM®
(diltiazem HCl) 30 mg and 60 mg Tablets

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. ***Cardiac Conduction.*** *CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1,243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.*
2. ***Congestive Heart Failure.*** *Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt).*

   Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. ***Hypotension.*** *Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.*
4. ***Acute Hepatic Injury.*** *In rare instances, significant elevations in enzymes such as alkaline phosphatase, CPK, LDH, SGOT, SGPT, and other symptoms consistent with acute hepatic injury have been noted. These reactions have been reversible upon discontinuation of drug therapy. The relationship to CARDIZEM is uncertain in most cases, but probable in some. (See PRECAUTIONS.)*

PRECAUTIONS

General. *CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.*

Drug Interaction. *Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)*

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. *A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.*

Pregnancy. *Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.*

There are no well-controlled studies in pregnant women, therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. *Diltiazem is excreted in human milk. One report suggests that concentrations in breast milk may approximate serum levels. If use of CARDIZEM is deemed essential, an alternative method of infant feeding should be instituted.*

Pediatric Use. *Safety and effectiveness in children have not been established.*

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences as well as their frequency of presentation are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%). In addition, the following events were reported infrequently (less than 1%):

Cardiovascular:	*Angina, arrhythmia, AV block (first degree), AV block (second or third degree — see conduction warning), bradycardia, congestive heart failure, flushing, hypotension, palpitations, syncope.*
Nervous System:	*Amnesia, gait abnormality, hallucinations, insomnia, nervousness, paresthesia, personality change, somnolence, tinnitus, tremor.*
Gastrointestinal:	*Anorexia, constipation, diarrhea, dysgeusia, dyspepsia, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH (see hepatic warnings), vomiting, weight increase.*
Dermatologic:	*Petechiae, pruritus, photosensitivity, urticaria.*
Other:	*Amblyopia, dyspnea, epistaxis, eye irritation, hyperglycemia, nasal congestion, nocturia, osteoarticular pain, polyuria, sexual difficulties.*

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: alopecia, gingival hyperplasia, erythema multiforme, and leukopenia. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established. Issued 7/86

See complete Professional Use Information before prescribing.

References: 1. *Pepine CJ, Feldman RL, Hill JA, et al: Clinical outcome after treatment of rest angina with calcium blockers: Comparative experience during the initial year of therapy with diltiazem, nifedipine, and verapamil. Am Heart J 1983, 106(6):1341-1347.* ***2.*** *Shapiro W: Calcium channel blockers: Actions on the heart and uses in ischemic heart disease. Consultant 1984,24(Dec):150-159.* ***3.*** *Johnston DL, Lesoway R, Humen DP, et al: Clinical and hemodynamic evaluation of propranolol in combination with verapamil, nifedipine and diltiazem in exertional angina pectoris: A placebo-controlled, double-blind, randomized, crossover study. Am J Cardiol 1985,55:680-687.* ***4.*** *Cohn PF, Braunwald E: Chronic ischemic heart disease, in Braunwald E (ed): Heart Disease: A Textbook of Cardiovascular Medicine, ed 2. Philadelphia, WB Saunders Co, 1984, chap 39.* ***5.*** *Schroeder JS: Calcium and beta blockers in ischemic heart disease: When to use which. Mod Med 1982;50(Sept):94-116*

Another patient benefit product from
PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC.
KANSAS CITY, MO 64137

0246M6

NEW YORK STATE JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
FRANK C. MAXON, JR, MD
ELIZA H. CALDWELL, MD
JOHN T. PRIOR, MD
STANFORD WESSLER, MD

Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Consulting Editor and Book Review Editor	RICHARD B. BIRRER, MD
Consulting Editor	NAOMI R. BLUESTONE, MD, MPH
Consulting Editor	CARL POCHEDLY, MD
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Managing Editor	CAROL L. MOORE
Librarian	ELLA ABNEY
Assistant Librarian	ELEANOR BURNS
Consulting Medical Writer	VICKI GLASER
Editorial Assistant	MILDRED J. ARFMANN
Secretary	ELIZABETH J. SOMERS
Advertising Director	ROBERT MILLER

ASSOCIATE EDITORIAL BOARD 1986

MICHAEL E. BERLOW, MD
RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MYLES S. GOMBERT, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
JAMES M. MORRISSEY, ESQ
STEPHEN NORDLICHT, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. United Media Associates, Inc, 16 Bruce Park Ave, Greenwich, CT 06830. Telephone: (203) 661-9702.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

JOHN A. FINKBEINER, MD,	*President*
DANIEL F. O'KEEFFE, MD,	*Past-President*
SAMUEL M. GELFAND, MD,	*President-Elect*
CHARLES D. SHERMAN, MD,	*Vice-President*
JOHN H. CARTER, MD,	*Secretary*
GEORGE LIM, MD,	*Assistant Secretary*
MORTON KURTZ, MD,	*Treasurer*
ROBERT A. MAYERS, MD,	*Assistant Treasurer*
CHARLES N. ASWAD, MD,	*Speaker*
SEYMOUR R. STALL, MD,	*Vice-Speaker*

Councilors

Term Expires 1987

DAVID M. BENFORD, MD,	New York
DUANE M. CADY, MD,	Onondaga
JAMES M. FLANAGAN, MD,	Wayne
M. THEODORE TANENHAUS, MD,	Kings
KATHLEEN E. SQUIRES, MD,	New York

(*Councilor representing the resident physician membership*)

FRANCES HARTENSTEIN-COLLICHIO,	Monroe

(*Councilor representing the medical student membership*)

Term Expires 1988

JAMES H. COSGRIFF, JR, MD,	Erie
RICHARD A. HUGHES, MD,	Warren
ANTONIO F. LASORTE, MD,	Broome
SIDNEY MISHKIN, MD,	Nassau

Term Expires 1989

ROBERT E. FEAR, MD,	Suffolk
STANLEY L. GROSSMAN, MD,	Orange
THOMAS D. PEMRICK, MD,	Rensselaer
RALPH E. SCHLOSSMAN, MD,	Queens

Trustees

RALPH M. SCHWARTZ, MD,	Kings
EDGAR P. BERRY, MD,	New York
RICHARD D. EBERLE, MD,	Onondaga
JOSEPH F. SHANAPHY, MD,	Richmond
VICTOR J. TOFANY, MD,	Monroe
BERNARD J. PISANI, MD,	New York
DANIEL F. O'KEEFFE, MD,	Warren

Executive Vice-President	DONALD F. FOY
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Vice-President Emeritus	GEORGE J. LAWRENCE, JR, MD
Director, Division of Policy Coordination	IRMA A. ERICKSON
Director, Division of Scientific Publications	PASCAL JAMES IMPERATO, MD
Director, Division of Communications	EDWARD A. HYNES
Director, Division of Computer Services	IVAN H. NAJMAN
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Governmental Affairs	GERARD L. CONWAY, ESQ
General Counsel	DONALD R MOY, JD
Director, Division of Management and Meeting Services	ROBERT MILLER
Director, Division of Membership Support Services	EUNICE M. SKELLY
Director, Division of Scientific and Educational Activities	PETER B. FARNSWORTH, MD
Director, Division of Socio-Medical Economics	MARIO V. MENGHINI
Medical Director, Committee for Physicians' Health	BARTON A. HARRIS, MD
Consultant	EDWARD SIEGEL, MD

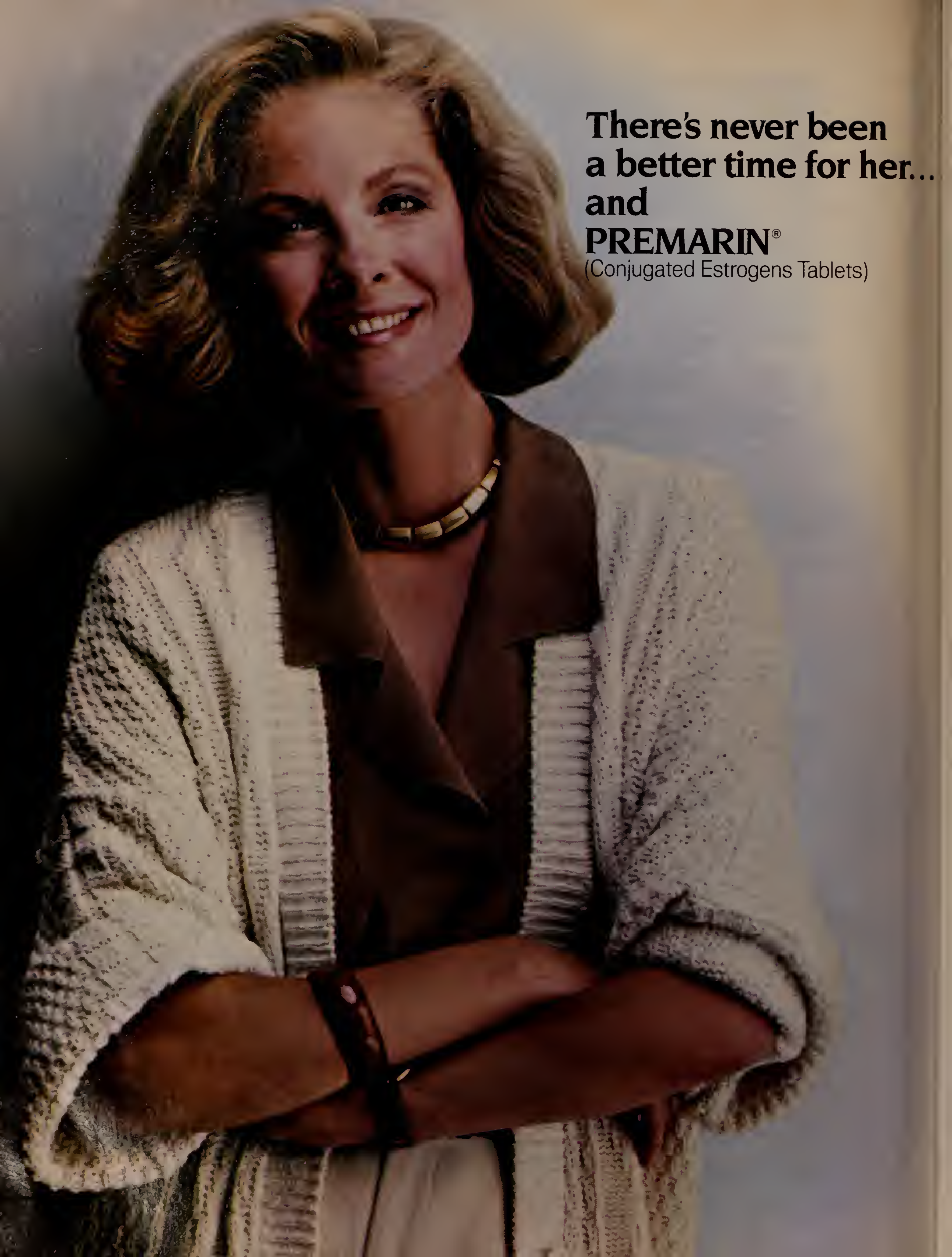
The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York.

Copyright © 1987, Medical Society of the State of New York. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.



There's never been
a better time for her...
and
PREMARIN®
(Conjugated Estrogens Tablets)

Now the evidence looks better than ever

Significantly reduced risk of endometrial hyperplasia

Endometrial hyperplasia was significantly reduced when progestin was added to PREMARIN therapy for more than ten days a month.[1-4] The risk of endometrial hyperplasia may also be reduced through cyclic administration of unopposed, low-dose PREMARIN.

Effect on lipids—an important feature

PREMARIN used alone does not adversely affect lipid levels. In fact, a clinical study has shown a significant increase in HDL cholesterol—from 49.7 mg/dL to 56.4 mg/dL—and decrease in LDL cholesterol—from 165.1 mg/dL to 138.1 mg/dL—after one year of therapy with PREMARIN, 0.625 mg.[5]

Low-dose control of menopausal symptoms*

PREMARIN effectively relieves vasomotor symptoms, such as hot flashes. When estrogen deficiency is limited to atrophic vaginitis, PREMARIN® (conjugated estrogens) Vaginal Cream restores the vaginal environment to its premenopausal state.

The most widely used, most extensively studied estrogen worldwide.

PREMARIN®

(Conjugated Estrogens Tablets)

Most trusted for more reasons

*PREMARIN is indicated for moderate-to-severe vasomotor symptoms.

Please see following page for brief summary of prescribing information.

For moderate-to-severe vasomotor symptoms

PREMARIN®

(Conjugated Estrogens Tablets)

0.3 mg 0.625 mg 0.9 mg 1.25 mg 2.5 mg

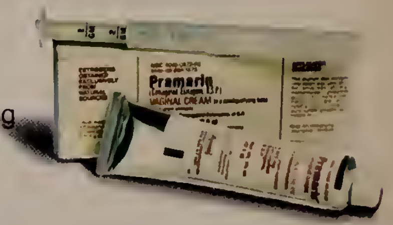
The appearance of these tablets is a trademark of Ayerst Laboratories.

For atrophic vaginitis

PREMARIN®

(Conjugated Estrogens)

Vaginal Cream
0.625mg/g



BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION AND PATIENT INFORMATION, SEE PACKAGE CIRCULARS.)*

PREMARIN® Brand of conjugated estrogens tablets, USP
PREMARIN® Brand of conjugated estrogens Vaginal Cream in a nonliquefying base

> 1. ESTROGENS HAVE BEEN REPORTED TO INCREASE THE RISK OF ENDOMETRIAL CARCINOMA.
>
> Three independent case control studies have reported an increased risk of endometrial cancer in postmenopausal women exposed to exogenous estrogens for more than one year. This risk was independent of the other known risk factors for endometrial cancer. These studies are further supported by the finding that incidence rates of endometrial cancer have increased sharply since 1969 in eight different areas of the United States with population-based cancer reporting systems, an increase which may be related to the rapidly expanding use of estrogens during the last decade. The three case control studies reported that the risk of endometrial cancer in estrogen users was about 4.5 to 13.9 times greater than in nonusers. The risk appears to depend on both duration of treatment and on estrogen dose. In view of these findings, when estrogens are used for the treatment of menopausal symptoms, the lowest dose that will control symptoms should be utilized and medication should be discontinued as soon as possible. When prolonged treatment is medically indicated, the patient should be reassessed on at least a semiannual basis to determine the need for continued therapy. Although the evidence must be considered preliminary, one study suggests that cyclic administration of low doses of estrogen may carry less risk than continuous administration; it therefore appears prudent to utilize such a regimen. Close clinical surveillance of all women taking estrogens is important. In all cases of undiagnosed persistent or recurring abnormal vaginal bleeding, adequate diagnostic measures should be undertaken to rule out malignancy. There is no evidence at present that "natural" estrogens are more or less hazardous than "synthetic" estrogens at equiestrogenic doses.
> 2. ESTROGENS SHOULD NOT BE USED DURING PREGNANCY.
> The use of female sex hormones, both estrogens and progestogens, during early pregnancy may seriously damage the offspring. It has been shown that females exposed in utero to diethylstilbestrol, a non-steroidal estrogen, have an increased risk of developing in later life a form of vaginal or cervical cancer that is ordinarily extremely rare. This risk has been estimated as not greater than 4 per 1,000 exposures. Furthermore, a high percentage of such exposed women (from 30% to 90%) have been found to have vaginal adenosis, epithelial changes of the vagina and cervix. Although these changes are histologically benign, it is not known whether they are precursors of malignancy. Although similar data are not available with the use of other estrogens, it cannot be presumed they would not induce similar changes. Several reports suggest an association between intrauterine exposure to female sex hormones and congenital anomalies, including congenital heart defects and limb reduction defects. One case control study estimated a 4.7-fold increased risk of limb reduction defects in infants exposed in utero to sex hormones (oral contraceptives, hormone withdrawal tests for pregnancy, or attempted treatment for threatened abortion). Some of these exposures were very short and involved only a few days of treatment. The data suggest that the risk of limb reduction defects in exposed fetuses is somewhat less than 1 per 1,000. In the past, female sex hormones have been used during pregnancy in an attempt to treat threatened or habitual abortion. There is considerable evidence that estrogens are ineffective for these indications, and there is no evidence from well controlled studies that progestogens are effective for these uses. If PREMARIN is used during pregnancy, or if the patient becomes pregnant while taking this drug, she should be apprised of the potential risks to the fetus, and the advisability of pregnancy continuation.

DESCRIPTION: PREMARIN (conjugated estrogens, USP) contains a mixture of estrogens, obtained exclusively from natural sources, blended to represent the average composition of material derived from pregnant mares' urine. It contains estrone, equilin, and 17α-dihydroequilin, together with smaller amounts of 17α-estradiol, equilenin, and 17α-dihydroequilenin as salts of their sulfate esters Tablets are available in 0.3 mg, 0.625 mg, 0.9 mg, 1.25 mg, and 2.5 mg strengths of conjugated estrogens. Cream is available as 0.625 mg conjugated estrogens per gram.
INDICATIONS AND USAGE: PREMARIN (conjugated estrogens tablets, USP): Moderate-to-severe *vasomotor* symptoms associated with the menopause (There is no evidence that estrogens are effective for nervous symptoms or depression without associated vasomotor symptoms and they should not be used to treat such conditions.) **Osteoporosis (abnormally low bone mass)**. Atrophic vaginitis. Kraurosis vulvae. Female castration.

PREMARIN (conjugated estrogens) Vaginal Cream is indicated in the treatment of atrophic vaginitis and kraurosis vulvae. PREMARIN HAS NOT BEEN SHOWN TO BE EFFECTIVE FOR ANY PURPOSE DURING PREGNANCY AND ITS USE MAY CAUSE SEVERE HARM TO THE FETUS (SEE BOXED WARNING).
Concomitant Progestin Use: The lowest effective dose appropriate for the specific indication should be utilized. Studies of the addition of a progestin for 7 or more days of a cycle of estrogen administration have reported a lowered incidence of endometrial hyperplasia. Morphological and biochemical studies of the endometrium suggest that 10 to 13 days of progestin are needed to provide maximal maturation of the endometrium and to eliminate any hyperplastic changes. Whether this will provide protection from endometrial carcinoma has not been clearly established. There are possible additional risks which may be associated with the inclusion of progestin in estrogen replacement regimens. (See PRECAUTIONS.) The choice of progestin and dosage may be important; product labeling should be reviewed to minimize possible adverse effects.
CONTRAINDICATIONS: Estrogens should not be used in women (or men) with any of the following conditions: 1. Known or suspected cancer of the breast except in appropriately selected patients being treated for metastatic disease. 2. Known or suspected estrogen-dependent neoplasia 3. Known or suspected pregnancy (See Boxed Warning). 4 Undiagnosed abnormal genital bleeding 5. Active thrombophlebitis or thromboembolic disorders. 6 A past history of thrombophlebitis, thrombosis, or thromboembolic disorders associated with previous estrogen use (except when used in treatment of breast or prostatic malignancy).
WARNINGS: Long-term continuous administration of natural and synthetic estrogens in certain animal species increases the frequency of carcinomas of the breast, cervix, vagina, and liver. There are now reports that estrogens increase the risk of carcinoma of the endometrium in humans. (See Boxed Warning.) At the present time there is no satisfactory evidence that estrogens given to postmenopausal women increase the risk of cancer of the breast, although a recent study has raised this possibility There is a need for caution in prescribing estrogens for women with a strong family history of breast cancer or who have breast nodules, fibrocystic disease or abnormal mammograms. A recent study has reported a 2- to 3-fold increase in the risk of surgically confirmed gallbladder disease in women receiving postmenopausal estrogens.

Adverse effects of oral contraceptives may be expected at the larger doses of estrogen used to treat prostatic or breast cancer or postpartum breast engorgement; it has been shown that there is an increased risk of thrombosis in men receiving estrogens for prostatic cancer and women for postpartum breast engorgement. Users of oral contraceptives have an increased risk of diseases, such as thrombophlebitis, pulmonary embolism, stroke, and myocardial infarction Cases of retinal thrombosis, mesenteric thrombosis, and optic neuritis have been reported in oral contraceptive users. An increased risk of postsurgery thromboembolic complications has also been reported in users of oral contraceptives. If feasible, estrogen should be discontinued at least 4 weeks before surgery of the type associated with an increased risk of thromboembolism, or during periods of prolonged immobilization. Estrogens should not be used in persons with active thrombophlebitis, thromboembolic disorders, or in persons with a history of such disorders in association with estrogen use. They should be used with caution in patients with cerebral vascular or coronary artery disease. Large doses (5 mg conjugated estrogens per day), comparable to those used to treat cancer of the prostate and breast, have been shown to increase the risk of nonfatal myocardial infarction, pulmonary embolism and thrombophlebitis. When doses of this size are used, any of the thromboembolic and thrombotic adverse effects should be considered a clear risk.

Benign hepatic adenomas should be considered in estrogen users having abdominal pain and tenderness, abdominal mass, or hypovolemic shock. Hepatocellular carcinoma has been reported in women taking estrogen-containing oral contraceptives. Increased blood pressure may occur with use of estrogens in the menopause and blood pressure should be monitored with estrogen use. A worsening of glucose tolerance has been observed in patients on estrogen-containing oral contraceptives. For this reason, diabetic patients should be carefully observed. Estrogens may lead to severe hypercalcemia in patients with breast cancer and bone metastases.
PRECAUTIONS: Physical examination and a complete medical and family history should be taken prior to the initiation of any estrogen therapy with special reference to blood pressure, breasts, abdomen, and pelvic organs, and should include a Papanicolaou smear. As a general rule, estrogen should not be prescribed for longer than one year without another physical examination being performed. Conditions influenced by fluid retention such as asthma, epilepsy, migraine, and cardiac or renal dysfunction, require careful observation. Certain patients may develop manifestations of excessive estrogenic stimulation, such as abnormal or excessive uterine bleeding, mastodynia, etc. Prolonged administration of unopposed estrogen therapy has been reported to increase the risk of endometrial hyperplasia in some patients. Oral contraceptives appear to be associated with an increased incidence of mental depression. Patients with a history of depression should be carefully observed. Preexisting uterine leiomyomata may increase in size during estrogen use. The pathologist should be advised of estrogen therapy when relevant specimens are submitted. If jaundice develops in any patient receiving estrogen, the medication should be discontinued while the cause is investigated. Estrogens should be used with care in patients with impaired liver function, renal insufficiency, metabolic bone diseases associated with hypercalcemia, or in young patients in whom bone growth is not complete. If concomitant progestin therapy is used, potential risks may include adverse effects on carbohydrate and lipid metabolism.

The following changes may be expected with larger doses of estrogen:

a. Increased sulfobromophthalein retention.

b Increased prothrombin and factors VII, VIII, IX, and X; decreased antithrombin 3; increased norepinephrine-induced platelet aggregability.

c. Increased thyroid binding globulin (TBG) leading to increased circulating total thyroid hormone, as measured by PBI, T4 by column, or T4 by radioimmunoassay. Free T3 resin uptake is decreased, reflecting the elevated TBG; free T4 concentration is unaltered.

d. Impaired glucose tolerance.

e. Decreased pregnanediol excretion.

f. Reduced response to metyrapone test.

g Reduced serum folate concentration.

h. Increased serum triglyceride and phospholipid concentration. As a general principle, the administration of any drug to nursing mothers should be done only when clearly necessary since many drugs are excreted in human milk.
ADVERSE REACTIONS: The following have been reported with estrogenic therapy, including oral contraceptives: breakthrough bleeding, spotting, change in menstrual flow; dysmenorrhea; premenstrual-like syndrome; amenorrhea during and after treatment; increase in size of uterine fibromyomata; vaginal candidiasis, change in cervical erosion and in degree of cervical secretion; cystitis-like syndrome; tenderness, enlargement, secretion (of breasts); nausea, vomiting, abdominal cramps, bloating; cholestatic jaundice; chloasma or melasma which may persist when drug is discontinued; erythema multiforme; erythema nodosum; hemorrhagic eruption; loss of scalp hair; hirsutism; steepening of corneal curvature; intolerance to contact lenses; headache, migraine, dizziness, mental depression, chorea; increase or decrease in weight; reduced carbohydrate tolerance; aggravation of porphyria; edema; changes in libido.
ACUTE OVERDOSAGE: May cause nausea, and withdrawal bleeding may occur in females.
DOSAGE AND ADMINISTRATION:
PREMARIN® Brand of conjugated estrogens tablets, USP
1. *Given cyclically for short-term use only.* For treatment of moderate to severe *vasomotor* symptoms, atrophic vaginitis, or kraurosis vulvae associated with the menopause (0.3 to 1.25 mg or more daily). The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible. Administration should be cyclic (eg, three weeks on and one week off). Attempts to discontinue or taper medication should be made at three- to six-month intervals.
2. *Given cyclically:* Female castration. Osteoporosis. Female castration—1.25 mg daily, cyclically Adjust upward or downward according to response of the patient. For maintenance, adjust dosage to lowest level that will provide effective control. Osteoporosis —0.625 mg daily. Administration should be cyclic (eg, three weeks on and one week off).

Patients with an intact uterus should be monitored for signs of endometrial cancer and appropriate measures taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding.
PREMARIN® Brand of conjugated estrogens Vaginal Cream
Given cyclically for short-term use only. For treatment of atrophic vaginitis or kraurosis vulvae.

The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible.

Administration should be cyclic (eg, three weeks on and one week off).

Attempts to discontinue or taper medication should be made at three-to-six month intervals.

Usual dosage range: 2 to 4 g daily, intravaginally, depending on the severity of the condition.

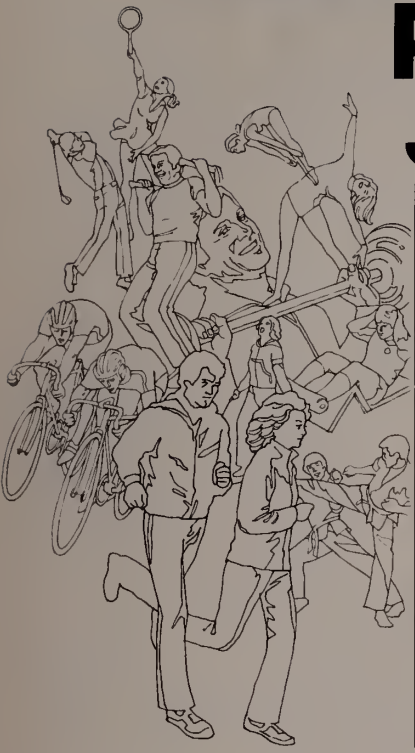
Treated patients with an intact uterus should be monitored closely for signs of endometrial cancer and appropriate diagnostic measures should be taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding.
References:
1. Whitehead MI, Townsend PT, Pryse-Davies J, et al: Effects of estrogens and progestins on the biochemistry and morphology of the postmenopausal endometrium. *N Engl J Med* 1981;305:1599-1605 **2.** Paterson MEL, Wade-Evans T, Sturdee DW, et al: Endometrial disease after treatment with oestrogens and progestogens in the climacteric. *Br Med J* 1980;280:822-824. **3.** Magos AL, Brincat M, Studd JWW, et al: Amenorrhea and endometrial atrophy with continuous oral estrogen and progestogen therapy in postmenopausal women. *Obstet Gynecol* 1985; 67: 496-499. **4.** Whitehead MI, Lane G, Siddle N, et al: Avoidance of endometrial hyperstimulation in estrogen-treated postmenopausal women. *Semin Reprod Endocrinol* 1983;1:1;41-52. **5.** Barnes RB, Roy S, Lobo RA: Comparison of lipid and androgen levels after conjugated estrogen or depo-medroxyprogesterone acetate treatment in postmenopausal women. *Obstet Gynecol* 1985;66:216-219.

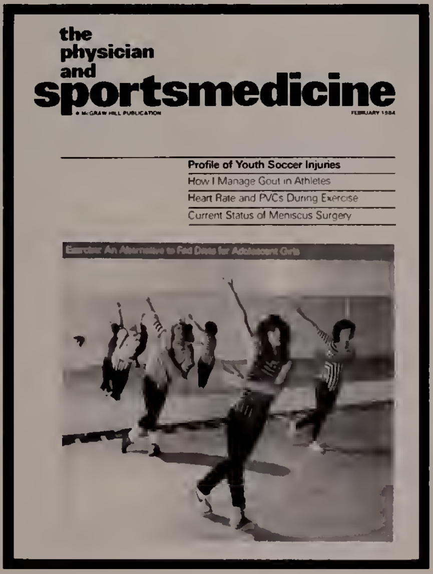
© 1986 Ayerst Laboratories

Ayerst®
AYERST LABORATORIES
New York, NY 10017

T6194/886

OVER 66,000 FAMILY PHYSICIANS READ THIS JOURNAL





the physician and sportsmedicine

FEBRUARY 1984

Profile of Youth Soccer Injuries
How I Manage Gout in Athletes
Heart Rate and PVCs During Exercise
Current Status of Meniscus Surgery

Exercise: An Alternative to Fad Diets for Adolescent Girls

Practical information on the medical aspects of fitness and exercise.

Tennis elbow: Joint resolution by conservative treatment.

Hypertrophic cardiomyopathy and the athlete.

Effects of sunscreen use during exercise in the heat.

Overuse injuries to the knee in runners.

How I manage ingrown toenails.

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices unless the information is submitted at least three months prior to the event.*

APRIL 1987

AROUND THE STATE

BRONX

Apr 29. **Forensic Aspects of Gambling.** Contact: Mark L. Taff, MD, Nassau County Medical Examiner's Office, PO Box 160, East Meadow, NY 11554. Tel: (516) 542-2400.

MANHATTAN

Apr 2–3. **Uveitis Update 1987.** 16 Cat 1 Credits. Lenox Hill Hospital's Einhorn Auditorium. Contact: Betty C. Agree, Assistant Director, Department of Public Relations, Lenox Hill Hospital, 100 East 77th St, New York, NY 10021. Tel: (212) 439-2400.

Apr 4. **Basic Microsurgery.** Manhattan Eye, Ear & Throat Hospital. Contact: Martha Klapp, Manhattan Eye, Ear & Throat Hospital, 210 East 64 St, New York, NY 10021. Tel: (212) 605-2762.

Apr 6. **Transmissible and Non-Transmissible Dementias in Amyloidosis of the Brain.** Stern Auditorium, Annenberg Building, The Mount Sinai Medical Center. Contact: The Mount Sinai Medical Center, School of Medicine, One Gustave L. Levy Place, New York, NY 10029. Tel: (212) 650-7301.

Apr 6–8. **Hypothalamic-Pituitary-Adrenal Axis Revisited in Honor of Dorothy Krieger.** 1 Cat 1 Credit. Vista International Hotel. *Apr 27–29.* **Hemochromatosis Conference.** 1 Cat 1 Credit. Vista International Hotel. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd St, New York, NY 10021.

Apr 7–11. **Electromyography and Electrodiagnosis.** 31 Cat 1 Credits. Mayflower Hotel. Contact: Daoud B. Karam, MD, New York Medical College, Rehabilitation Medicine, Coler Memorial Hospital, Roosevelt Island, NY 10044. Tel: (212) 688-9400, ext 285.

Apr 23–25. **Advances in Dermatology.** 17 Cat 1 Credits. NYU Medical Center. Contact: NYU Medical Center Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Apr 25. **59th Annual Scientific Session, New York Cardiological Society.** 5 Cat 1 Credits. Hotel Pierre. Info: New York Cardiological Society, 84 Grove St, Suite Two, New York, NY 10014. Tel: (212) 924-5346.

(*continued on p 12A*)

Classified Advertising

PHYSICIANS WANTED

ARIZONA BASED PHYSICIAN RECRUITMENT firm has opportunities Coast to Coast. "Professionals working with Professionals". Over 13 years experience. Call (602) 990-8080, or send CV to: Mitchell & Associates, Inc., P.O. Box 1804, Scottsdale, AZ 85252.

EMERGENCY MEDICINE POSITIONS. Full/part time emergency medicine physicians sought by muiti-state professional association. Openings in metropolitan NY, NJ, PA, MD, DC, FL and throughout U.S. Contact or send CV to Liberty Healthcare Corporation, 399 Market Street, Suite 400, Philadelphia, PA 19106, (215) 592-7400 or outside PA (800) 331-7122.

GENERAL SURGERY—Solo General Surgical Practice looking for a young associate to begin Spring/Summer 1987. General and some vascular surgery. Delightful upstate location. Prefer candidates completing residency or with recent training. Send confidential letter and C.V. to P.O. Box 505, New Hartford, New York 13413.

DO YOU WANT TO SHARE A THRIVING DERMATOLOGY PRACTICE: Do you enjoy year around outdoor recreation? You should contact us—a progressive multi-specialty group of 68 physicians in a pleasant, growing community. We offer a liberal financial package and outstanding benefits. Send curriculum vitae and references to: Michael T. Anderson, M.D., Willmar Medical Center, 101 Willmar Ave. SW, Willmar, MN 56201.

PHYSICIANS needed for 75 children's private residential camps July and/or August. Contact: Association of Independent Camps (JM), 43 West 23rd Street—Sixth Floor, New York, N.Y. 10010 (212) 645-6620.

BE A "WINTER TEXAN" INTERNIST. Enjoy the warm, beautiful Rio Grande Valley while practicing internal medicine with an internist. Texas license essential. Salary, living accommodations, and malpractice insurers. Send curriculum vitae to Luis F. Arango, M.D., P.A., 104 South Bryan Road, Mission, Texas 78572 or call (512) 585-2783 for more information.

NEW YORK, WESTERN: Seeking primary care trained physicians for full time Emergency Department positions. Moderate volume. Attractive hourly compensation, plus malpractice insurance, Director position available. Contact: Emergency Consultants, Inc., 2240 S. Airport Road, Room 42, Traverse City, MI 49684: 1-800-253-1795 or in Michigan 1-800-632-3496.

ACTIVELY SEEKING INDIVIDUAL who is board certified, prepared in emergency, internal medicine, family practice or surgery. 475 bed teaching hospital with 45,000 ed visits annually. Attractive salary and benefits; bonus for talented, career oriented applicant. Send C.V. to Dr. R. Hart, 101 Union Avenue, Suite 817, Syracuse, New York 13203 or call (315) 478-1977.

PHYSICIANS WANTED—CONT'D

PHYSICIAN WANTED to share office space with an older general practitioner with intention of sale later on retirement. Pleasant, rural community in Finger Lakes area. Situated on lake. Hospital nearby. Terms negotiable. Days call (315) 536-4456, nights (315) 536-8084 or write H. A. Mikk, M.D., Medical Arts Building, Pen Yan, NY 14527.

WANTED Primary care board certified/board eligible Internists or Family Physician for four doctor primary care medical group on Long Island. Great opportunity and future. Start about July 1, 1987. Reply Dept. 438 c/o NYSJM.

FAMILY PRACTICE OPPORTUNITY. Two established Mid-Island physicians seeking senior resident to enter Family practice July 1, 1987. Reply Dept. #436 c/o NYSJM.

INTERNIST IS WANTED AS PARTNER to family physician practice in Queens. Yugoslavian origin preferred, but not necessary. Eventually take over this well established practice. Reply Dept. 435 c/o NYSJM.

MULTI-SPECIALTY NINE PHYSICIAN GROUP located in a rural setting adjacent to Western New York metropolitan area is seeking the following medical specialties: Internal Medicine/Gastroenterology/Cardiology—board certified/eligible Internist is needed. Sub-specialty interest welcome particularly in Gastroenterology or Cardiology. Approximately one-half of practice could be in either of these fields. Family Practice/Generalist—board certified/eligible Family Practitioner experienced and/or desirous of working in a group practice setting. Obstetrical practice available but not required.

We are currently involved in a wide variety of third party payor programs to include a prepaid (H.M.O.) component. Group is quite progressive and can offer the medical professional a broad array of in-house medical support services. Please direct responses to the address listed below and please include a summary of your education and experience along with salary expectations. Reply Dept. 437 c/o NYSJM.

POSITIONS WANTED

RADIOLOGIST, A.B.R. & A.B.N.M. is seeking a salaried position with a group or a hospital for 4–5 years. Group partnership not a consideration. Expertise in Angiography including neuro (20 years). Special competence in nuclear cardiology. Mammography & xeromammography (10 years), cat-head & body (6 years). Artrography, special procedures, ultrasonography & diagnostic radiology. Location limited to 100 mile radius of New York City. Reply to Dept. 432 c/o NYSJM.

PRACTICES AVAILABLE

6-YEAR INTERNAL MEDICINE PRACTICE FOR SALE in Dutchess County. Good location, near two hospitals, in rapidly growing area. Leaving area, reasonable sale price. Reply Dept. #431 c/o NYSJM.

WELL ESTABLISHED, WELL EQUIPPED, Pediatric practice to sell in Hampton, VA. Located close to a major community hospital. Lease or sale of office property negotiable. (804) 245-5283.

PRACTICES AVAILABLE—CONT'D

"SELLING PRACTICES IS OUR ONLY BUSINESS"

Want to maximize the return on your investment? Before you buy or sell, call for an appraisal to assure the best financial and transfer terms. We guide you through the entire sales process from initial meeting to closing. Serving NY, CT, NJ and MA.

Countrywide Business Brokerage, Inc.
319 East 24th St., Suite 23-G
New York, NY 10010
(212) 686-7902 (203) 869-3666

OPTOMETRIC PRACTICE FOR SALE. Lucrative income. Jamaica, Queens. Dr. Dubin, (718) 739-5454.

EQUIPMENT

DISCOUNT HOLTER SCANNING SERVICES starting at $35.00. Spacelabs holter recorder (cassette) available from $1,275.00. Cardionostic holter recorder (cassette) available from $1,125.00. Smallest and lightest holters update. Fast service (24–48 hours) turn over. Hookup kits starting at $4.95. Special introductory offer of three free tests with any purchase or lease of the recorder. Cardiologist overread available for $15.00. If interested call (301) 870-3626.

50% OFF PREVIOUSLY OWNED MEDICAL—Laboratory, x-ray, ultrasound equipment in excellent condition. We buy, sell, broker, repair. Ask about our Holter Scanning Services. Call anytime: Medical Equipment Resale, 24026 Haggerty, Farmington Hills, MI 48018. (313) 477-6880.

REAL ESTATE FOR SALE OR RENT

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

EAST SIXTIES, PROFESSIONAL BUILDING. The preferred location for internists, cardiologist, rheumatologist. 7 treatment rooms, 2 reception areas to share with three other internists. Share NYC licensed lab, radiology, thermography. Full or part time. (212) 838-2860.

MEDICAL OFFICE FOR SUBLEASE—prime Manhattan location—4 rooms, secretaries station and waiting room. (212) 362-4848.

WESTCHESTER COUNTY, NEW YORK STATE, medical facility (building) for sale or lease. Fully equipped, 350 x-ray, Kodak processor pulmonary function, EKG laboratory setup. Suitable for 2 or 3 physicians (internist, gastroenterologist, orthopedist, GP, FP, surgeon or combination). Upper middle class area convenient to transportation, churches, schools, post office, banks, parking. Call (914) 835-4848.

FOR SEVERE COUGHS, CAN YOU REMEMBER THESE LETTERS?

You know Robitussin®, and the "DAC" formula gives you just what you need to manage those severe coughs complicated with nasal congestion . . . Decongestant And Codeine!

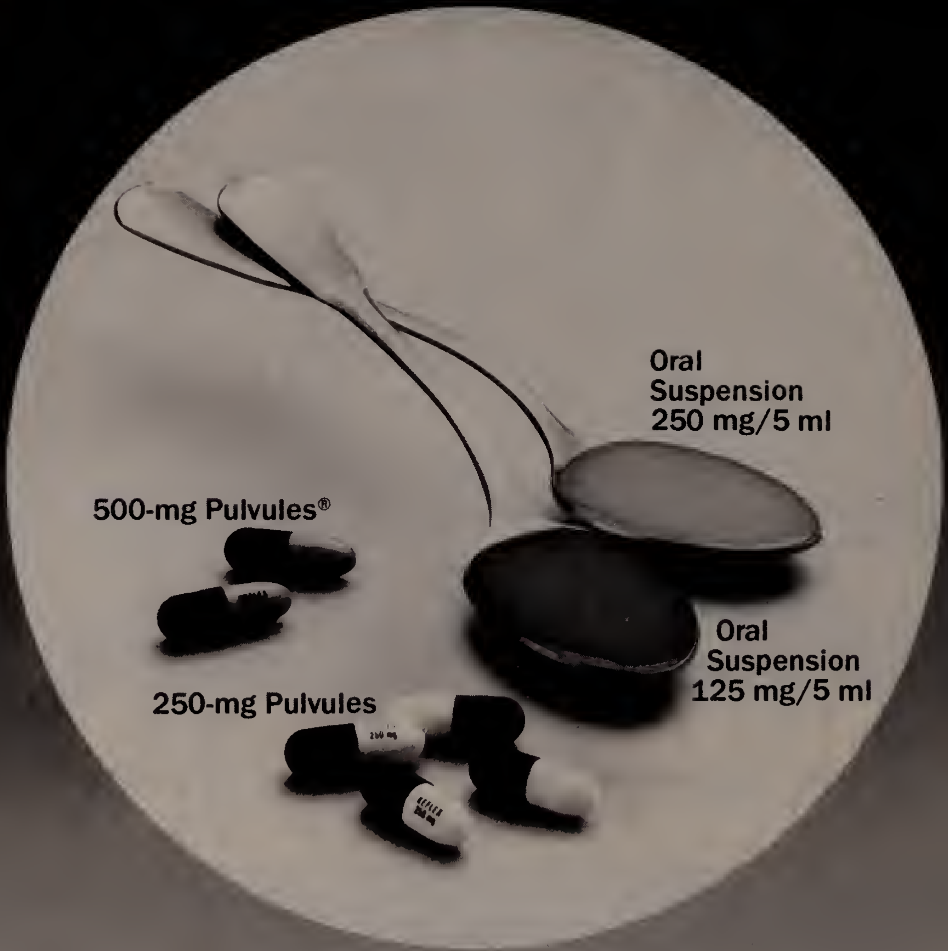
Robitussin®-DAC Ⓥ

Each 5 ml (1 teaspoonful) contains:
Guaifenesin, USP . . . . 100 mg
Pseudoephedrine
Hydrochloride, USP . . . . 30 mg
Codeine Phosphate, USP . . . . 10 mg
(Warning: May be habit forming)
Alcohol 1.4 percent

℞ ONLY

A-H-ROBINS
Pharmaceutical Division
Richmond, Virginia 23261-6609

Easy To Take



Keflex®
cephalexin

Additional information available to the profession on request.

DISTA
420113

Dista Products Company
Division of Eli Lilly and Company
Indianapolis, Indiana 46285
Mfd. by Eli Lilly Industries, Inc.
Carolina, Puerto Rico 00630

NEW YORK STATE
JOURNAL OF MEDICINE

March 1987 • Volume 87, Number 3

COMMENTARIES

Parkinson disease

Parkinson disease (PD) occupies a special niche in neurological history. It is the first disease for which a therapy was rationally designed. Although the histopathologic basis of the disease had been known for many years, it was not until 1963[1] that the major neurochemical deficit, the ten-fold reduction of levels of dopamine in the basal ganglia, was recognized. In retrospect, one can recognize that this was something of a lucky break. It turns out that many neurotransmitters are reduced in the basal ganglia in PD.[2,3] Imagine the difficulties of working out clinical trials of serotonin, enkephalins, gamma-aminobutyric acid (GABA), etc. A few negative trials with these agents might have pushed levodopa (L-dopa) off the scene, leaving us with better stereotactic surgery as the only advance in treatment in the last 25 years. Recent events, involving an incredible series of ironies,[4] leading to a fortuitous observation in an otherwise tragic situation, have pushed PD research into a second stage of development.

Looking back on the development of L-dopa is a useful exercise in understanding where we are now. Clearly, the modern era for the treatment of PD was ushered in with the recognition that L-dopa was an effective treatment. However, the initial trials of the drug were not auspicious.[5] Major side effects precluded the use of therapeutic doses of L-dopa until a regimen was devised in which a low dose was given, followed by slow titration upwards into the therapeutic range.[6] A doubting neurologist witnessing the early, unsuccessful trials of L-dopa made the analogy that PD was like a car running out of gas, with dopamine being the gas, and that giving L-dopa was tantamount to pouring gas all over the car and expecting it to run. But run it did. Like the beneficial effects of penicillin, those of L-dopa were clear and, in some cases, were so striking that they appeared to be miraculous. In 1973, Oliver W. Sacks published a book called *Awakenings*, describing the miraculous effect of L-dopa on a group of patients with postencephalitic PD in a rehabilitation center.[7] Subsequent editions of this book have been published in the U.S. in 1974 (Doubleday), 1976 (Vintage), and 1983 (Dutton). These people had been frozen in both space and time and were suddenly resurrected from their petrified state.

Unfortunately, the early miracle wore thin. In a manner reminiscent of another miracle drug, prednisone, L-dopa went through the phases of doubt, appreciation, disappointment, and finally understanding of its risks and benefits. The dramatic responses to L-dopa initially prompted hope that the drug might be a therapeutic, rather than simply a symptomatic agent; that L-dopa could halt the progression of the disease. This hope was not realized. Then the problems began. Dementia became a more obvious problem. Was it a side effect of the drug, or was this increased prevalence a result of the more advanced patients now being able to communicate? We now know that dementia is related to progression of the underlying degenerative process and is not an iatrogenic disorder. Other problems now known to be related to L-dopa remain, and dealing with them forms the core about which the art of parkinsonian therapy is drawn: wearing off, on-off, variable response, dyskinesias, dystonias, myoclonus, toxic delirium, etc. And finally, what can be done for the patient who no longer responds to drug therapy?

These were (and still are) critical issues confronting neurology, when two new developments, initially quite independent of each other, arose: first, the slowly developing area of brain implantation. While surely the stuff of science fiction, it has become increasingly clear that brain from one animal could be transplanted into the brain of another animal. More remarkable is the fact that not only do these implants survive, but they become functional both biochemically and behaviorally.[8] Anticipating the obvious ethical difficulties of harvesting fetal brain cells, work is in progress to demonstrate that cross species grafting can be accomplished even without immunosuppression,[9] although the use of immunosuppressive agents improves the results.

Equally important for the future of PD research was the discovery of MPTP,[10] a "designer drug," inadvertently manufactured, which selectively destroys cells in the substantia nigra, creating a model of Parkinson's disease in intravenous drug abusers who used MPTP. "Disease is nature's way of experimenting," said Pasteur, and the results of this awful experiment were immediately recognized by Langston and colleagues.[11] Using MPTP, they developed the first good animal model of PD. Young primates injected parenterally with MPTP not only lose cells in the pars compacta of the substantia nigra, but develop clinical signs of PD. The cell loss can be titrated to create mild or severe disease. Old primates treated with MPTP not only suffer cell losses in the pars compacta but also in the locus ceruleus, another brain area affected in PD, and even develop Lewy-type bodies, the sine qua non of PD.[12] This breakthrough will hopefully lead to a better understanding of the physiologic state in PD and, more importantly, to an understanding of what goes wrong with prolonged L-dopa therapy. Then, presumably, ways to avoid these problems can be determined. The use of MPTP also allows for the development of new treatment protocols using nonhuman models. The response to brain implantation in an MPTP-induced parkinsonian primate is much more convincing evidence for starting human trials than is the response of a rat previously rendered parkinsonian by a chemosurgical ablation.

MPTP can also be used to create parkinsonian-like models in nonmammals, such as frogs,[13] salamanders,[13] and even a leech.[14] Studies of such simple neurologic networks will provide basic insights into the problems of more complex parkinsonian systems.

The discovery of MPTP (initially developed as a potential antiparkinsonian drug) has spurred interest in exploring the environment for causes of PD, as MPTP, while not a naturally occurring chemical, is closely related to a large number of compounds which do occur naturally.

Parkinson disease is a common affliction. It affects about 1% of the American population above the age of 65.[15] All physicians, aside from pediatricians, must deal with it on some level. This issue of the *Journal* contains a superb review article that describes current events in the field of Parkinson disease, with practical points about diagnosis and management.[16] For the first time in more than 20 years, we are optimistic about being close to taking a giant step forward in our understanding of this disease. We are closer to better treatment, maybe even to halting progress of PD, and, possibly, to preventing this disease.

JOSEPH H. FRIEDMAN, MD
Assistant Professor
Division of Neurology
Brown University

Chief
Division of Neurology
Roger Williams General Hospital
Providence, RI 02908

1. Hornykiewicz O: Die topische Lokalisation und das Verhalten Von Noradrenalin und Dopamin (3-hydroxytyramin) in der substantia nigra des normalen und Parkinsonkranken Menschen. *Wien Klin Wchnschr* 1963; 75:309-312.
2. Hornykiewicz O: Brain neurotransmitter changes in Parkinson's disease, in Marsden CD, Fahn S (eds): *Movement Disorders.* London, Butterworth Scientific, 1982, pp 41-58.
3. Uhl GR, Hackney GO, Torchia M, et al: Parkinson's disease: Nigral receptor changes support peptidergic rise in nigrostriatal modulation. *Ann Neurol* 1986; 20:194-203.
4. Lewin R: Trail of ironies to Parkinson's disease. *Science* 1984; 224:1083-1085.
5. Fehling C: Treatment of Parkinson's disease with L-dopa: A double blind study. *Acata Neurol Scan* 1966; 42:367-372.
6. Cotzias CG, Van Woert MH, Schiffer LM: Aromatic amino acids and modification of Parkinsonism. *N Engl J Med* 1967; 276:374-379.
7. Sacks OW: *Awakenings.* London, Duckworth, 1976.
8. Perlow MJ, Freed WJ, Hoffer BJ, et al: Brain grafts reduce motor abnormalities produced by destruction of nigrostriatal dopamine system. *Science* 1979; 204:643-646.
9. Bjorklund A, Stenevi U, Dunnett SB, et al: Cross species neural grafting in a rat model of Parkinson's disease. *Nature* 1982; 298:652-654.
10. Langston JW, Ballard P, Tetrud JW, et al: Chronic parkinsonism in humans due to a product of meperidine analogue synthesis. *Science* 1983; 219:979-980.
11. Langston JW, Forno LS, Rebert CS, et al: Selective nigral toxicity after systemic administration of 1-methyl-4-phenyl-1,2,5,6-tetrahydropyridine (MPTP) in the squirrel monkey. *Br Res* 1984; 292:390-394.
12. Forno LS, Langston AW, DeLanney LE, et al: Locus ceruleus lesions and eosinophilic inclusions in MPTP-treated monkeys. *Ann Neurol* 1986; 20:449-455.
13. Barbeau A, Dallaire L, Buu NT, et al: New amphibian models for the study of 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP). *Life Sci* 1985; 36:1125-1134.
14. Lent CM: MPTP depletes neuronal monoamines and impairs the behavior of the medicinal leech, in Markey SP, Castagnoli N, Trevor AJ, et al (eds): *MPTP: A Neurotoxin Producing a Parkinsonian Syndrome.* New York, Academic Press, 1986, pp 105-118.
15. Kessler II: Parkinson's disease in epidemiological perspective. *Adv Neurology* 1978; 19:355-384.
16. Lieberman AN: Update on Parkinson disease. *NY State J Med* 1987; 87:147-153.

Mesenteric cysts

In 1897, Berkeley Moynihan wrote that "cysts of the mesentery are among the surgical rarities."[1] He observed that only 100 cases had been reported by then. By 1954, some 600 reported cases were found in the literature according to a review published by Oh, Danese, and Dreiling.[2] In 1966, McKeown and Burks[3] put forth the view that mesenteric cysts were medical curiosities. Although these lesions are rare, they should enter into consideration in the differential diagnosis of abdominal masses.

Early in my residency, before the advent of computed tomography (CT) and ultrasonography, I had the opportunity of operating on two patients with mesenteric cysts. The diagnosis had not been made preoperatively. As Caldarola and LaDuca[4] note in their article in this issue of the *Journal*, CT scanning and ultrasound have greatly facilitated the preoperative diagnosis of mesenteric cyst.

As a consequence of my operative experience with mesenteric cysts, I reviewed the medical literature on the subject and even had the opportunity of visiting Dr Wilbur Emory Burnett, who had coauthored an important paper on the subject in 1950.[5] Over the years, many surgeons have discovered mesenteric cysts during the course of abdominal surgical procedures and some have published reports of their cases along with a review of the literature, as I did in 1974.[6] The report and review by Caldarola and LaDuca[4] in this issue of the *Journal* is thorough, informative, and current.

Many classifications have been devised for mesenteric cysts. Vaughn et al[7] described them as either simple or neoplastic. Ford,[8] however, elaborated a more extensive classification. The older descriptions of mesenteric cysts, particularly those in the papers of Moynihan[1] and Tillaux,[9] are of great interest. All surgeons should recall the diagnostic signs of Tillaux—a fluctuating tumor toward the umbilicus, great mobility in a transverse direction and around a central axis, and a zone of resistance over the mass—when considering the diagnosis of mesenteric cyst on physical examination.[1]

Surgeons will be better prepared to evaluate and treat abdominal masses after reading this current review, which is in many ways a rediscovery of mesenteric cysts in the 1980s. It is interesting to note that the patient described by Caldarola and LaDuca had undergone four cesarean sections. It would be worthwhile to review all of the reported cases in the literature in order to determine the frequency of surgical procedures in patients who subsequently developed mesenteric cysts. While the precise etiology of mesenteric cysts is unknown, they are generally thought to be developmental in origin. An unanswered question is whether or not previous and unrelated surgical procedures promote the later formation of mesenteric cysts, by somehow uncovering and fostering these developmental abnormalities.

PHILIP CAROPRESO, MD
1023 2nd Street, SW
Mason City, Iowa 50401

1. Moynihan BGA: Mesenteric cysts. *Ann Surg* 1897; 26:1-30.
2. Oh C, Danese CA, Dreiling DA: Chylous cysts of mesentery. *Arch Surg* 1967; 94:790-793.
3. McKeown MJ, Burks JL: Mesenteric cysts: A diagnostic conundrum. *Northwest Med* 1966; 65:748-750.
4. Caldarola V, LaDuca J: Mesenteric cyst. *NY State J Med* 1987; 87:177-179.
5. Burnett WE, Rosemond GP, Bucher RM: Mesenteric cysts: Report of 3 cases in one of which calcified cyst was present. *Arch Surg* 1950; 60:699-706.
6. Caropreso PR: Mesenteric cysts: A review. *Arch Surg* 1974; 108:242-246.
7. Vaughn AM, Lees WM, Henry JW: Mesenteric cysts: Review of literature and report of calcified cyst of mesentery. *Surgery* 1948; 23:306-317.
8. Ford JR: Mesenteric cysts: Review of the literature with a report of an unusual case. *Am J Surg* 1960; 99:878-884.
9. Tillaux PJ: These de Paris (as cited by Moynihan, 1897).

Adverse effects of cyclosporine A on the microcirculation of the cold preserved kidney

The use of cyclosporine A (CyA) in organ transplantation and in a variety of disease states associated with altered immunologic reactivity has been lauded by the biomedical literature and the lay press.[1] The isolation of CyA marks the development of an immunosuppressive agent capable of selective rather than broad spectrum activity. Its selective effect(s) on helper T cells, with only minimal effects on myeloid cells, constitutes a clear advantage over conventional immunosuppressive agents, such as azathioprine. Numerous studies have shown the superiority of CyA over azathioprine in preventing rejection crises and in prolonging the graft survival of many solid organs. CyA is also credited as a major factor in the development of safer techniques for heart and liver transplantation.

Possibly because of the relatively large doses of CyA used in the initial management of renal transplant recipients (20-25 mg/kg/day), the toxic effects of this agent were documented at a relatively early stage of its clinical trials. In the first clinical trial of CyA by Calne et al[2] (1978), only one of seven renal allografts functioned. Acute tubular necrosis (ATN) developed in the remainder, and they were lost. Calne et al[2] then showed that patients who suffered from ATN and were treated with CyA lost their grafts, while patients who exhibited immediate graft function rarely developed CyA nephrotoxicity. Similar findings were reported by the Canadian Multicenter Trial,[3] in which the overall results of kidney transplantation with CyA appeared to be superior to those achieved by conventional therapy.

Certain surgical variables were capable of magnifying the nephrotoxicity of CyA. CyA was shown to have a particularly adverse effect on subsequent renal function in patients given cadaveric renal allografts, with 24 hours or more of machine perfusion preservation prior to transplantation, or in patients who required 45 minutes or more for completion of the renal vascular anastomoses. These risk factors were not relevant when azathioprine/prednisone immunosuppression was used. A greater risk of graft loss in patients treated with CyA in whom ATN develops is a frequent finding. Keown and colleagues[4] recently reported a 19% decrease in three-month graft survival in cadaveric kidneys that exhibited ATN. Furthermore, the three-month graft survival rate in recipients of second allografts that did not function immediately was only 14%. In a randomized prospective study conducted by Simmons et al[5] at the University of Minnesota, the rate of ATN among recipients of cadaveric allografts who were treated with CyA was found to be similar to the rate among recipients of renal allografts who were treated with azathioprine. However, patients treated with CyA required twice as many dialyses, the recovery rate from ATN was much slower, and the median serum creatinine level at one year was 2.1 mg %, versus 1.5 mg % in patients treated with azathioprine. These observations led this group to administer CyA only after transplantation, and only when renal function had been fully recovered. This policy negates the beneficial effects of CyA on the recognitive phase of the immune response, and thus may not be the best strategy to address the problem of nephrotoxicity in allografts with ATN.

Combatting the nephrotoxic effects of CyA on the ischemic kidney requires a better understanding of the mechanisms responsible. Unfortunately, this effect seems to be specific for the human species, and there is no good animal model for studying CyA nephrotoxicity. Some useful information, however, has been derived from studies in the rat, dog, and pig.[6-8] Humes et al[6] studied the nephrotoxic effects of CyA in rats. After administration of 100 mg of CyA/kg of body weight, elevations of blood urea nitrogen and creatinine were noted. Despite such derangement in renal function, no toxic effect on renal tubular cells could be demonstrated in vitro or in vivo. There was, however, a significant decrease in total renal blood flow (RBF). In a related study, McKenzie et al[7] have shown that administration of as little as 2.5 mg/kg of CyA to mongrel dogs, combined with renal artery occlusion for 30 minutes, produced significant decreases in RBF. These data suggest that CyA nephrotoxicity primarily affects the renal microcirculation rather than the renal tubules. The precise mechanism by which CyA nephrotoxicity impairs RBF in the transplanted kidney is unclear. The acute nature of the decrease in RBF after intravenous administration of CyA and the reversibility of this effect suggest, however, that structural lesions are not required to produce CyA-mediated renal hypoperfusion.[8] Rather, the adverse effects of CyA on the renal microcirculation may be brought about through impairment of the regulation of the RBF. It seems that the delicate balance between the levels of renal hormones responsible for RBF regulation may be impaired in patients with CyA toxicity.

High levels of thromboxane have been found in the urine of patients suffering from CyA nephrotoxicity.[4] Inhibition of prostaglandin synthesis by Meclofenamate accentuated CyA-induced renal vasoconstriction.[9] Activation of the renin-angiotensin system, resulting in cortical hypoperfusion, has also been observed.[10] In addition, renal vasoconstriction mediated by alpha adrenergic stimulation may aggravate CyA nephrotoxicity. CyA-induced vasoconstriction can be ameliorated by the administration of phenoxybenzamine and by renal denervation.[9] The importance of this finding on the induction of CyA nephrotoxicity in the denervated transplanted kidney is unclear, but this mechanism may play a role in the induction of CyA nephrotoxicity in heart and liver transplant recipients.

Structural arteriolar damage leading to vascular thrombosis has also been reported.[11] CyA was found to inhibit the secretion of prostacyclin stimulating factor, which is responsible for maintaining the integrity of the vascular endothelium.[12] In addition, CyA was found to increase platelet aggregation[13] and procoagulant activity in monocytes,[14] thus leading to intravascular coagulation.

The strong correlation noted between CyA nephrotoxicity and preservation damage implies that CyA nephrotoxicity acts in concert with hypothermic ischemia to damage the cortical microcirculation, resulting in acute tubular necrosis.

Recent studies conducted in our laboratories with dogs[15,16] have shown that prolonged cold storage preservation results in a progressive deterioration of the integrity of the renal cortical microcirculation. The addition of the calmodulin inhibitor trifluoperazine to Collins' flush solution protected the cortical microcirculation and resulted in dramatic improvements in renal viability after transplantation. Further studies have also shown that maintaining the integrity of the cortical microcirculation is a prerequisite for graft function after transplantation in human allograft recipients.[17] The recent observation by Totterman et al[18] that CyA concentration in the renal cortex increased markedly in kidneys with hypoperfusion, despite the occurrence of normal trough levels in the serum, provides another mechanism for the nephrotoxic effects of CyA on an already-damaged renal microcirculation.

One helpful strategy to reduce CyA nephrotoxicity might include measures designed to protect the integrity of the renal microcirculation during organ preservation. The use of agents such as free radical scavengers, calcium inhibitors, and prostaglandins during organ preservation is one approach to reducing the currently prohibitive incidence of post-transplant ATN, as well as averting complications due to CyA nephrotoxicity. Clinical experience using these new therapeutic modalities is limited, however, and further clinical trials are required.

DAVID ANAISE, MD
WAYNE C. WALTZER, MD
ANGELO N. ARNOLD, PhD
FELIX T. RAPAPORT, MD
Department of Surgery
State University of New York
at Stony Brook
Stony Brook, NY 11794-8192

1. Strom TB: Immunosuppressive agents in renal transplantation. *Kidney Int* 1984; 26:353-365.
2. Calne RY, White DJG, Thiru S, et al: Cyclosporin A in patients receiving renal allografts from cadaver donors. *Lancet* 1978; 2:1323-1327.
3. Stiller C: The Canadian trial of cyclosporine: Cyclosporine therapy compared to standard immunosuppression in renal transplants: An exploration of nephrotoxicity. *Cyclosporine Biological Activity & Clinical Applications* 1983; 1:263-268.
4. Keown PA, Stiller CR, Wallace AC, et al: Cyclosporine nephrotoxicity: Exploration of the risk factors and prognosis of the renal injury. *Transplant Proc* 1985; 17(suppl):247-253.
5. Simmons RL, Canafax DM, Strand M, et al: Management and prevention of cyclosporine nephrotoxicity after renal transplantation: Use of low doses of cyclosporine, azathioprine, and prednisone. *Transplant Proc* 1985; 17(suppl):266-275.
6. Humes HD, Jackson NM, O'Connor RP, et al: Pathogenetic mechanisms of nephrotoxicity: Insights into cyclosporine nephrotoxicity. *Transplant Proc* 1985; 17(suppl):51-62.
7. McKenzie N, Devineni R, Vezina W et al: The effect of cyclosporine on organ blood flow. *Transplant Proc* 1985; 17:1973-1975.
8. Cunningham C, Gavin MP, Whiting PH, et al: Serum cyclosporine levels, hepatic drug metabolism and renal tubulotoxicity. *Biochem Pharmacol* 1984; 33:2857-2861.
9. Paller MS, Murray BM: Renal dysfunction in animal models of cyclosporine toxicity, in Kahan BD (ed): *Cyclosporine, volume III, Diagnosis and Management of Associated Renal Injury*. New York, Grune & Stratton, Inc, 1985, p 153.
10. Siegl H, Ryffel B, Petric R, et al: Cyclosporine, the renin-angiotensin-aldosterone system, and renal adverse reactions. *Cyclosporine Biological Activity & Clinical Applications* 1983; 1:503-509.
11. Kahan B, in Kahan BD (ed): *Cyclosporine, volume III, Diagnosis and Management of Associated Renal Injury*. New York, Grune & Stratton, Inc, 1985, p 162.
12. Leithner C, Sinzinger H, Pohanka E, et al: Occurrence of hemolytic uremic syndrome under cyclosporine treatment: Accident or possible side effect mediated by a lack of prostacyclin-stimulating plasma factor? *Cyclosporine Biological Activity & Clinical Applications* 1983; 1:571-573.
13. Neild GH, Ivory K, Williams DG: Glomerular thrombi and infarction in rabbits with serum sickness following cyclosporine therapy. *Cyclosporine Biological Activity & Clinical Applications* 1983; 1:566-570.
14. Carlsen E, Courillon Mallet AC, Prydz H: Effects of cyclosporine A on procoagulant activity in mononuclear blood cells and monocytes in vitro. *Clin Exp Immunol* 1985; 60:407-410.
15. Anaise D, Bachvaroff RJ, Sato K, et al: Enhanced resistance to the effects of hypothermic ischemia in the preserved canine kidney. *Transplantation* 1984; 38:570-574.
16. Anaise D, Sato K, Atkins H, et al: Scintigraphic evaluation of the viability of cold preserved kidneys before transplantation. *J Nucl Med* 1984; 25:1304-1309.
17. Anaise D, Lehr H, Atkins H, et al: Preservation of the cortical renal microcirculation—a prerequisite for immediate renal allograft function. *Transplant Proc* (in press).
18. Totterman TH, Sjoberg O, Claesson K: Monitoring of cyclosporine levels by radioimmunoassay in kidney biopsies. *Transplant Proc* 1986; 18:52-54.

Urinary calcium excretion in nonfamilial hyperparathyroidism

MURRAY LAS, MD, URIEL S. BARZEL, MD

ABSTRACT. Urinary volume, specific gravity, and calcium excretion were examined in 33 patients with mild, asymptomatic, nonfamilial hyperparathyroidism who were on their usual diets. Hyposthenuria (urinary specific gravity less than 1.010 in first morning specimen) was found in 11 cases. Polyuria (24-hour urinary volume greater than 2,000 mL) was present in only three cases. Urinary calcium excretion was less than 100 mg per 24 hours in 13 cases, and less than 200 mg per 24 hours in 26 cases. Hypercalciuria (urinary calcium greater than 250 mg per day) was found in only five cases. All four patients with hyperparathyroidism and nephrolithiasis, on the other hand, had calciuria of 220 mg per 24 hours or greater. We conclude that hypercalciuria is less frequent than hypocalciuria in mild sporadic hyperparathyroidism.

(NY State J Med 1987; 87:143–146)

It had been well established in the past that hyperparathyroidism was associated with hypercalciuria, and the latter had been considered a necessary element in the diagnosis of this condition.[1] Urine concentrating deficiency and polyuria were also expected in the hyperparathyroid patient. In the present work, we have reexamined the urinary specific gravity, urinary volume, and the prevalence of hypercalciuria in patients in whom hyperparathyroidism had been diagnosed as a result of multiphasic biochemical screening. We found that urine specific gravity is rarely low, urine volume is generally normal, calciuria is frequently normal, and at times low, and that hypercalciuria is uncommon in this patient population.

PATIENTS AND METHODS

We reviewed data of patients in whom hyperparathyroidism was diagnosed following the discovery of hypercalcemia on multiphasic screening. All patients were seen at the Montefiore Medical Group, a large primary care facility, during the period 1980-1985. All patients were carefully questioned about the presence in family members of hyperparathyroidism, hypercalcemia, kidney stone disease, and metabolic bone disease. Members of two families with familial hyperparathyroidism secondary to hyperplasia of all four glands, diagnosed surgically in at least one member of each family, were excluded. Patients with abnormal kidney function were also excluded. In all, 29 women and eight men with nonfamilial hyperparathyroidism, ranging in age from 40 to 80 years, were studied. All except two patients (nos. 7 and 23, Table I) had an elevated blood level of c-terminal parathyroid hormone in the face of hypercalcemia. In 13 patients, surgical confirmation of the diagnosis was obtained, and in 20 patients, the diagnosis remained presumptive. During the same time period, four additional patients with hyperparathyroidism were identified who had kidney stone disease, all of whom were operated on, and thus had proven hyperparathyroidism. With the exception of one patient with kidney stone disease (no. 34), none of the patients were on diuretic therapy at the time of urine collection.

The levels of creatinine and calcium in the blood as well as blood urea nitrogen (BUN) were determined by sequential multiple analysis using a computer (Technicon Co, Tarrytown, NY). Twenty-four-hour urine collections were done by patients while on their customary diets, as part of the initial workup. Urinary calcium was determined on a Technicon autoanalyser by the method of Kessler and Wolffman.[2] Urine specific gravity was determined on an American Optical refractometer (AO TS Meter 10423) on a separate fasting morning urine specimen. C-terminal parathyroid hormone radioimmunoassays were obtained from commercial laboratories as indicated in Table I and are presented in the table as multiples of the upper limit of normal as well as the actual reported levels. Statistical analysis was by the Students' t test.

RESULTS

Postmenopausal women predominated in this series, as in other series of patients with hyperparathyroidism. The mean ages of the patients in the three groups (proven hyperparathyroidism, presumptive hyperparathyroidism, and proven nephrolithiasis-associated hyperparathyroidism) were nearly identical (Table I). By definition, the BUN level was 25 mg/dL or lower in all cases of hyperparathyroidism. The mean BUN level was virtually identical in the proven and the presumptive hyperparathyroid groups, but was higher ($p < 0.025$) in the nephrolithiasis as compared to the presumptive hyperparathyroid group. Serum creatinine levels were 1.4 mg/dL or lower in the non-nephrolithiasis groups and were not significantly different among all three groups. The serum calcium levels varied in each group within a very narrow range. The levels were 12 mg/dL or lower in all but three patients—with one of these patients representing each of the three groups. The mean serum calcium level was lowest in the presumptive hyperparathyroid group ($p < 0.025$, compared with the confirmed hyperparathyroid group). The parathyroid hormone level in the presumptive hyperparathyroid group was also significantly lower ($p < 0.025$) compared with the other two groups.

From the Division of Endocrinology and Metabolism, Department of Medicine, Montefiore Medical Center, and the Albert Einstein College of Medicine, Bronx, NY.

Address correspondence to Dr Barzel, Division of Endocrinology and Metabolism, Department of Medicine, Montefiore Medical Center, 111 East 210th St, Bronx, NY 10467.

TABLE I. Patients with Confirmed Hyperparathyroidism, Presumptive Hyperparathyroidism, or Nephrolithiasis

Patient	Sex	Age	Blood Urea Nitrogen (mg/dL)	Serum Creatinine (mg/dL)	Serum Calcium (mg/dL)	Immunoassayable C-terminal Blood Parathyroid Hormone		24-Hour Urinary Calcium Excretion (mg/24 hr)	Calcium Clearance ÷ Creatinine Clearance[‡]	Comment
						X[*]	V[†]			
					Confirmed Hyperparathyroidism					
1	M	70	13	1.2	11.3	4.7	1798[§]	71	0.0063	Adenoma
2	F	68	21	1.0	11.2	1.9	723[§]	145	0.0123	Adenoma
3	F	66	17	0.9	11.6	2.7	1000[§]	264	0.0237	Adenoma
4	F	47	12	0.9	13.3	1.6	64[‖]	394	0.0249	Adenoma
5	F	60	15	—	11.6	2.2	840[§]	174	—	Hyperplasia
6	F	60	25	1.3	11.8	5.3	1986[§]	35	0.0040	Adenoma
7	F	67	13	0.8	11.6	1.3	50[‖]	282	0.0189	Adenoma
8	F	73	22	0.9	11.8	0.7	249[§]	145	0.0115	Adenoma
9	F	80	25	1.1	11.9	1.6	65[‖]	82	0.0106	Adenoma
10	F	62	20	1.0	11.7	2.6	975[§]	152	0.0107	Adenoma
11	F	63	14	0.9	10.9	2.5	942[§]	140	0.0095	Hyperplasia
12	M	61	15	1.2	12.0	1.6	722[¶]	329	0.0207	Adenoma
13	F	67	11	0.8	10.7	2.8	1035[§]	151	0.0128	Adenoma
Mean		65.8	18.4	1.03	11.68	2.4	1296[§] 61[‖]	238.4	0.0138	
Standard Deviation		7.3	4.8	0.17	0.62	1.3	508 7.4	103.5	0.0067	
					Presumptive Hyperparathyroidism					
14	M	64	19	1.4	10.9	1.2	444[§]	71	0.0100	
15	M	40	13	1.2	10.7	1.2	446[§]	375	0.0217	
16	F	72	19	1.1	12.9	1.2	449[§]	133	0.0114	
17	F	70	12	1.3	11.5	3.1	1149[§]	178	0.0235	
18	F	73	14	1.0	11.1	1.4	56[‖]	24	0.0030	
19	F	79	18	1.1	11.0	1.0	384[§]	87	0.0147	
20	M	68	18	1.1	11.0	2.4	96[‖]			
						7.0	2610[§]	22	0.0017	
21	F	54	15	0.9	10.5	1.4	623[¶]	104	0.0066	
22	F	78	18	0.9	10.7	1.0	390[§]	38	0.0037	
23	F	47	18	0.8	11.3	0.7	264[§]	24	0.0010	
24	F	65	16	0.9	11.0	1.1	401[§]	36	0.0030	
25	F	79	17	1.3	11.0	1.4	543[§]	41	0.0063	
26	F	54	16	1.0	11.4	1.0	378[§]	101	0.0088	
27	F	64	14	1.1	10.9	1.1	424[§]	144	0.0141	
28	F	59	19	0.8	11.4	1.1	45[‖]	245	0.0228	
29	F	65	16	1.2	11.0	1.2	455[§]	36	0.0039	
30	F	80	18	0.9	11.2	3.1	1181[§]	165	0.0189	
31	F	41	9	0.9	11.1	1.9	74[‖]	59	0.0032	
32	M	70	17	1.3	11.6	1.3	53[‖]	216	0.0281	
33	M	65	16	1.4	10.6	1.4	525[§]	183	0.0203	
Mean		66.5	16.5	1.11	11.19	1.6	651[§] 70[‖]	185.2	0.0113	
Standard Deviation		10.8	2.3	0.20	0.51	1.4	344 19	103.6	0.0086	
						Nephrolithiasis				
34	M	67	29	1.6	11.1	1.9	717[§]	227	0.0235	Adenoma
35	F	51	15	0.9	11.5	1.6	610[§]	234	0.0196	Adenoma
36	F	74	22	1.0	10.8	2.2	808[§]	243	0.0301	Adenoma
37	F	64	17	1.0	12.9	8.6	3209[§]	320	0.0261	Adenoma
Mean		65.1	22.2	1.19	11.6	3.5	2214[§]	261.5	0.0248	
Standard Deviation		8.0	5.6	0.3	0.8	3.4	1219	39.9	0.0044	

[*] Multiples of highest limit of normal
[†] Actual value
[‡] Calculated by the formula creatinine clearance = (140 − age) × body weight (kg)/72 × serum creatinine (mg/dL)
[§] Upjohn Exotic Tests Lab; highest limit of normal, 375 pgeq/mL
[‖] Mayo Medical Laboratory; highest limit of normal, 40 μLeq/mL
[¶] Smith Kline Clinical Lab; highest limit of normal, 450 pg/mL

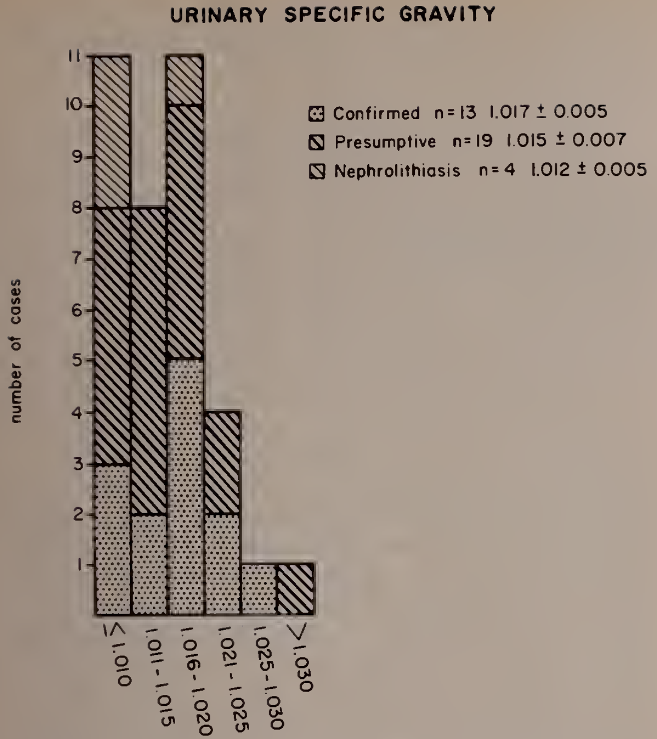


FIGURE 1. Specific gravity of first morning urine in 37 cases of primary hyperparathyroidism.

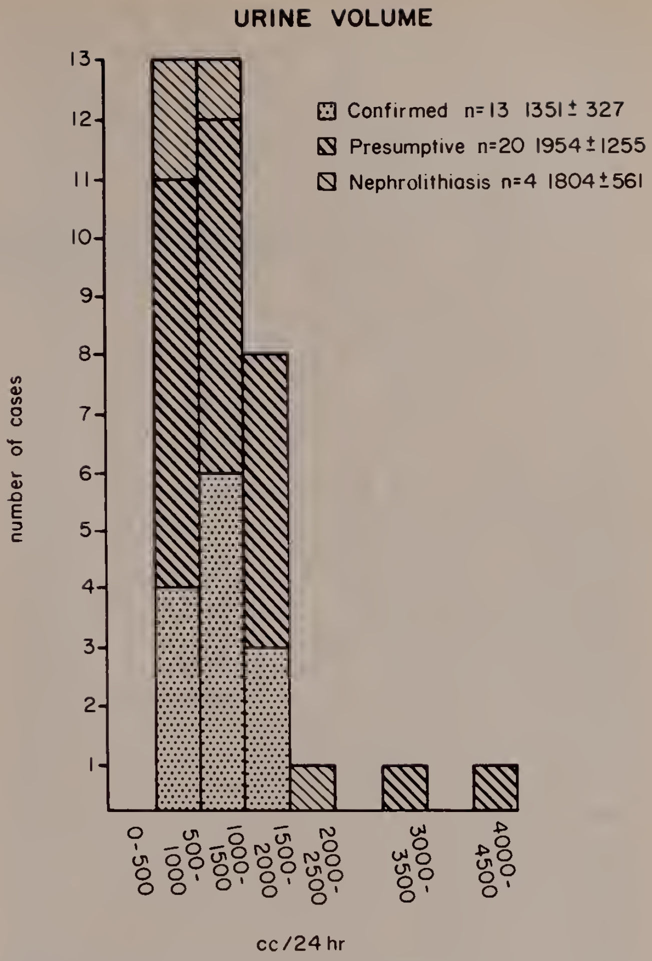


FIGURE 2. Urinary volume (cc per 24 hours) in 37 cases of primary hyperparathyroidism.

Urinary specific gravity and urine volume were not significantly different between the groups. Specific gravity (Fig 1) ranged from 1.002 to 1.026. Only two patients had values below 1.009, six had specific gravity of 1.009, and all the others had levels of 1.010 and higher. Urine volumes (Fig 2) were lower than 2,000 mL in all but three patients—two in the presumptive group and one in the nephrolithiasis group. There was no relationship between urine volume, which is governed by fluid intake, and urine specific gravity.

Urinary calcium excretion (Table 1 and Fig 3) varied greatly in all three groups, ranging between 22 mg/day and 394 mg/day. The mean level was not significantly different among the three groups, but tended to be higher in the nephrolithiasis group than in the other two. Urinary calcium excretion was less than 100 mg/day in three patients with proven hyperparathyroidism and in nine patients with presumptive hyperparathyroidism (23% and 45%, respectively). The level was less than 200 mg/day in nine of the proven and 17 of the presumptive hyperparathyroid cases (69% and 85%, respectively). Calciuria of 200 mg/day or more was found in three of the 13 patients with proven hyperparathyroidism (23%), and in four of the 20 patients with presumptive hyperparathyroidism (20%). In the nephrolithiasis hyperparathyroid group, calciuria was greater than 220 mg/day in all patients. An estimation of the ratio of calcium clearance to calculated creatinine clearance revealed the ratio to be less than 0.0100 in three members and less than 0.0150 in eight members of the confirmed hyperparathyroid group, and in 10 and 14 members, respectively, of the presumptive group.

Regression analysis revealed no correlation between blood calcium levels and urinary calcium excretion and no correlation between parathyroid hormone levels and urinary calcium excretion.

DISCUSSION

The first person with proven hyperparathyroidism in the United States presented in 1928 with collapsing vertebrae and stag-horn calculi.[3] His presentation of combined bone and kidney stone disease, associated with elevated levels of serum calcium, lowered serum phosphorus levels, an elevated serum alkaline phosphatase level, and hypercalciuria became the sine qua non for hyperparathyroidism in the following decade. No sooner had this syndrome been accepted by the medical profession than it began to be revised: Albright et al[4] demonstrated in 1937 that hy-

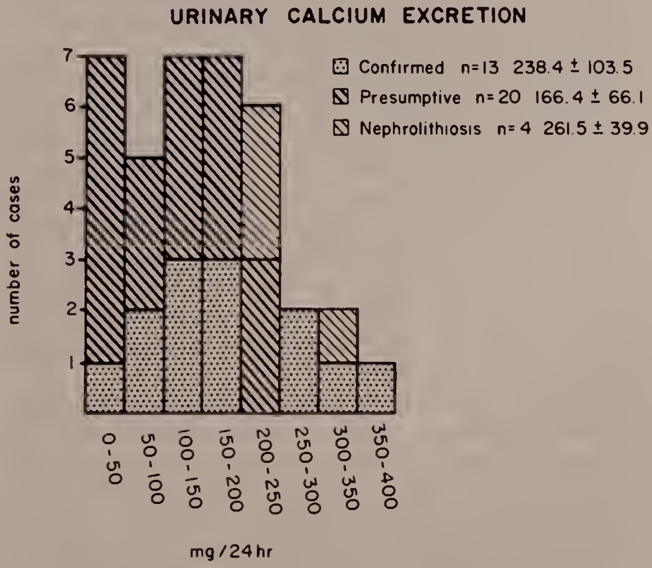


FIGURE 3. Calciuria (mg per 24 hours) in 37 cases of primary hyperparathyroidism.

perparathyroidism could exist with little or no bone disease and with normal levels of serum alkaline phosphatase. Subsequently, kidney stone disease became the primary clinical presentation of hyperparathyroidism. Hypercalciuria, together with hypercalcemia and hypophosphatemia, continued to be the major biochemical markers of the disease. Thus, Bartter[5] stated in 1967, before the radioimmunoassay for parathyroid hormone had become clinically available, that "the cardinal biochemical signs of primary hyperparathyroidism are lowered serum phosphorus, elevated serum calcium, and elevated urinary calcium." Hyposthenuria and polyuria were frequently associated with this syndrome.

With the introduction of multiphasic biochemical screening in the 1970s, physicians began to find hypercalcemia in patients with no history of disease. The majority of those patients with asymptomatic hypercalcemia are found, following appropriate workup, to have hyperparathyroidism. The disease in this patient population is generally mild: serum calcium levels tend to be no more than 1–2 mg/dL above the upper limit of the normal range, and nephrolithiasis and clinical bone disease are generally absent. We demonstrate here that hyposthenuria and polyuria occur infrequently in this patient population. Urinary calcium excretion is quite variable, but hypercalciuria is uncommon in this patient population: calciuria was less than 200 mg/day in 69% of patients with proven hyperparathyroidism and in 85% of the presumptive group, with slightly less severe disease. In the entire non-nephrolithiasis group, 12 of 33 patients (36%) had calciuria of less than 100 mg/day, and calciuria was less than 200 mg/day in 26 patients (78%). These data contrast with those of the patients with nephrolithiasis, who all had calciuria greater than 220 mg/day. The notion that urinary calcium levels may be normal and not increased in hyperparathyroidism was first proposed in 1966 by Pyrah et al,[6] who observed that 32% of patients had urinary calcium in the normal range (lower than 300 mg/24 hours). Similar data can be gleaned from later series.[7-11] We suggest, on the basis of the data presented here and in the series mentioned above,[6-11] that hypercalciuria is more likely to be present in patients with nephrolithiasis, and that patients without stone disease generally have normal calciuria.

The fact that urinary calcium levels may be normal or low in patients with hyperparathyroidism should come as no surprise to the physiologist. It is well established that parathyroid hormone stimulates calcium reabsorption by the renal tubule.[12] Excess parathyroid hormone secretion can be expected to accentuate this effect and to increase the fractional reabsorption of calcium by the kidney. Hypercalciuria, if it occurs in hyperparathyroidism, must therefore result from tubular damage caused by the disease process or from a marked increase in the postprandial filtered load of calcium.[8]

Hypocalciuria, expressed as total calcium excretion of less than 200 mg in 24 hours, or as an estimated creatinine clearance of less than 0.015, was present in nine out of 13 patients with proven hyperparathyroidism. We can therefore state that hypocalcemia is quite common in hyperparathyroidism, a fact that has particular significance in relation to the recently described entity of familial hypocalciuric hypercalcemia.[13-17] In view of these data, we believe that a low urinary calcium level cannot be used to make the latter diagnosis in any given individual, unless the parathyroid hormone level is normal, in which case a familial pattern should be investigated.

The diagnostic workup for hyperparathyroidism depends heavily on the determination of serum calcium, phosphorus, and parathyroid hormone levels. Because urinary calcium can be low, normal, or high in hyperparathyroidism, the determination of 24-hour excretion of calcium is useful primarily for the prescription of a therapeutic regimen for the patient with hyperparathyroidism. Because, based on our experience, more than 90% of patients are cured by surgery,[18] we recommend that all patients with nonfamilial hyperparathyroidism, even those with asymptomatic disease, should undergo surgical neck exploration if there is no medical contraindication and if the patient's life expectancy is longer than five years.[19] In patients whose urinary calcium excretion is excessive, the recommendation for surgery should be made with greater urgency, as they may have an increased risk of developing kidney stones.[8,9] These patients require control of their dietary calcium intake, because of the risk of nephrolithiasis, until they undergo surgery. The majority of patients with hyperparathyroidism, on the other hand, need not change their dietary habits at all, since their urinary calcium excretion may be well within normal limits while they maintain their regular diets.

REFERENCES

1. Keating FR Jr: Diagnosis of primary hyperparathyroidism. Clinical and laboratory aspects. *JAMA* 1961; 178:547–555.
2. Kessler G, Wolfman M: An automated procedure for the simultaneous determination of calcium and phosphorus. *Clin Chem* 1964; 10:686–703.
3. Albright F, Reifenstein FG Jr: *The Parathyroid Glands and Metabolic Bone Disease.* Baltimore, Williams and Wilkins, 1948.
4. Albright F, Sulkowitch HW, Bloomberg E: Further experience in the diagnosis of hyperparathyroidism, including discussion of cases with minimal degree of hyperparathyroidism. *Am J Med Sci* 1937; 193:800–812.
5. Bartter FC: Primary hyperparathyroidism, in Beeson PB, McDermott W (eds): *Cecil-Loeb Textbook of Medicine*, ed 12. Philadelphia, WB Saunders, 1967, pp 1359–1361.
6. Pyrah LN, Hodgkinson A, Anderson CK: Primary hyperparathyroidism. *Br J Surg* 1966; 53:245–316.
7. Hammonds JC, Williams JL, Harvey L: Primary hyperparathyroidism—a review of cases in the Sheffield area. *Br J Urol* 1976; 48:539–548.
8. Broadus AE, Horst RL, Lang R, et al: The importance of circulating 1,25-dihydroxyvitamin D in the pathogenesis of hypercalciuria and renal-stone formation in primary hyperparathyroidism. *N Engl J Med* 1980; 302:421–426.
9. Pak CYC, Nicar MJ, Peterson R, et al: A lack of unique pathophysiologic background for nephrolithiasis of primary hyperparathyroidism. *J Clin Endocrinol Metab* 1981; 53:536–542.
10. Graham JJ, Harding PE, Hoare LL, et al: Asymptomatic hyperparathyroidism: An assessment of operative intervention. *Br J Surg* 1980; 67:115–118.
11. Halse J: Urinary 3′,5′-cyclic adenosine monophosphate in relation to serum and urinary calcium in acromegaly and primary hyperparathyroidism. *Acta Endocrinol* (Copenh) 1980; 94:468–474.
12. Nordin BEC, Peacock M: Role of kidney in regulation of plasma-calcium. *Lancet* 1969; 2:1280–1283.
13. Marx SJ, Spiegel AM, Brown EM, et al: Divalent cation metabolism. Familial hypocalciuric hypercalcemia versus typical primary hyperparathyroidism. *Am J Med* 1978; 65:235–242.
14. Heath H 3d, Purnell DC: Urinary 3′,5′-adenosine monophosphate responses to exogenous and endogenous parathyroid hormone in familial benign hypercalcemia and primary hyperparathyroidism. *J Lab Clin Med* 1980; 96:974–984.
15. Marx SJ, Attie MF, Stock JL, et al: Maximal urine-concentrating ability: Familial hypocalciuric hypercalcemia *versus* typical primary hyperparathyroidism. *J Clin Endocrinol Metab* 1981; 52:736–740.
16. Marx SJ, Attie MF, Levine MA, et al: The hypocalciuric or benign variant of familial hypercalcemia: Clinical and biochemical features in fifteen kindreds. *Medicine* 1981; 60:397–412.
17. Marx SJ, Spiegel AM, Levine MA, et al: Familial hypocalciuric hypercalcemia: The relation to primary parathyroid hyperplasia. *N Engl J Med* 1982; 307:416–426.
18. Silver CE, Schoenbach SF, Barzel US: Surgical treatment of primary hyperparathyroidism. *NY State J Med* 1977; 77:732–735.
19. Lifschitz BM, Barzel US: Parathyroid surgery in the aged. *J Gerontol* 1981; 36:573–575.

REVIEW ARTICLES

Update on Parkinson disease

Abraham N. Lieberman, MD

Parkinson disease (PD) is a progressive disorder of the central nervous system (CNS). The annual incidence is 20 new cases per 100,000 population. It is estimated that there are one million persons with PD in the United States. The prevalence of PD is age-specific, with increasing occurrence after age 50; however, the disease does appear in persons under 40 years of age. Harry Truman, Francisco Franco, Mao Tse Tung, and Adolf Hitler had PD. The list of prominent persons with PD spans every occupation and includes Chester Bowles, ambassador to India; Morris Udall, congressman from Arizona; Arthur Koestler, author; Ernie Bushmiller, cartoonist; Michael Redgrave and Terry Thomas, actors; Salvador Dali, painter; and Muhammed Ali, boxer.

PD is characterized by five main clinical features: rigidity, tremor, bradykinesia, postural instability, and a gait disorder.[1] Other signs may include depression, dementia, postural deformity, speech impairment, and autonomic insufficiency including dysphagia, sialorrhea, orthostasis, impotence, and constipation.

When rigidity, tremor, bradykinesia, gait impairment, and postural instability predominate, when the course of the disease is progressive, and when there is no obvious cause for the disorder, then the condition is known as idiopathic PD. Patients with these five clinical features exhibit pathologic changes characterized by degeneration of the pigmented neurons of the substantia nigra and locus ceruleus. The histologic marker of PD is the Lewy body, an eosinophilic inclusion in the affected neurons of the substantia nigra. Paralleling the nigral degeneration is a loss of striatal dopamine.[2] These patients have a uniformly good response to levodopa, which corrects, in part, the striatal dopamine deficiency. In fact, a pharmacologic trial of levodopa is one way of confirming a clinical impression of PD.

When there is an admixture of the five main clinical features with other features, when the course of the condition is atypical, when a cause of the disorder is known, or when there is a poor response of the condition to levodopa, the condition is referred to as Parkinson syndrome (PS). PS encompasses a number of disorders—some are of unknown etiology but have clinical features of PD. These disorders include progressive supranuclear palsy, striatonigral degeneration, the Shy-Drager syndrome, olivopontocerebellar atrophy with nigral degeneration, spinocerebellar atrophy with nigral degeneration, and multiple system atrophy disease. This latter condition includes parkinsonian features that may be associated with cerebellar dysfunction, autonomic insufficiency, and amyotrophy. All of these disorders differ from PD in that their main pathologic features are outside the substantia nigra and involve the striatum, pallidum, brainstem, or cerebellum. Lewy bodies are not usually found in these disorders, and the response of these disorders to levodopa is poor. Also classified under PS are a number of disorders of known etiology that result in parkinsonian symptoms, but the main pathologic features of these disorders are outside the substantia nigra. These disorders include multiple basal ganglia infarcts; manganese poisoning[3]; carbon monoxide poisoning[4]; methanol poisoning[5]; and cyanide poisoning.[6] Lewy bodies do not occur in any of these conditions. Some of these conditions are progressive. PS also includes pugilistic parkinsonism.[7] Recent newspaper accounts about Muhammed Ali have brought pugilistic parkinsonism to everyone's attention and have renewed interest in the effects of boxing on the brain. Professional boxers, who receive multiple severe blows to the head, may experience a number of neurologic problems including parkinsonism. The parkinsonism may progress even though the fighter stops boxing.

All of the above Parkinson syndromes are irreversible. In addition, there are several Parkinson syndromes that are induced by drugs; these syndromes are reversible on cessation of the offending drug. Drugs that can lead to PS include central dopamine receptor blocking agents such as phenothiazines, butyrophenones, and metoclopramide; and dopamine depleting agents such as reserpine, tetrabenazine and alpha-methyl paratyrosine. These drug-induced Parkinson syndromes are unassociated with any pathologic changes in the brain.

Etiology

Given the easy recognition of PD and the number of famous people with the disease, it is curious that the first description of PD did not appear until 1817 in the report

---

From the Department of Neurology, New York University School of Medicine, NY Infirmary—Beekman Downtown Medical Center, and the American Parkinson Disease Association.

Address correspondence to Dr Lieberman, Chairman, Medical Advisory Board, the American Parkinson Disease Association, 650 First Ave, 4th Floor, New York, NY 10016.

This work was supported, in part, by a grant from the American Parkinson Disease Association and the Leon Lowenstein Foundation.

by James Parkinson. The lack of references to PD before 1817 suggests that PD was uncommon before the industrial revolution. This, in turn, suggests that the increased number of patients with PD today may be related to an environmental agent, perhaps a toxic product of the industrial revolution. It should be noted, however, that PD also occurs in nonindustrialized countries. Alternatively, the increased number of patients with PD may be related to the increase in life span of all people since the industrial revolution, allowing more patients a chance to live long enough for PD to develop.

If PD is related to aging, because of an inherent tendency of a specific population of neurons (the pigmented neurons in the substantia nigra) to die prematurely, then this abiotrophy should be inherited. In a study of 43 monozygotic and 19 dizygotic twin pairs, in which one of the twins had PD, it was found that only one monozygotic twin pair was concordant for PD.[8] Thus, the frequency of PD in monozygotic twins was similar to what would be expected in an unrelated control group matched for age. Other genetic studies have also failed to reveal an increased incidence of PD in families with one exception—a study involving a subset of patients with early-onset PD (before age 40); this finding is not typical of idiopathic PD. It is believed at present that whatever the role of genetics may be, it is a subtle and as yet undefined one.

One environmental agent that has been considered as a cause of idiopathic PD is a virus, specifically the virus responsible for von Economo's encephalitis, a pandemic of which occurred between 1919 and 1926. Patients with von Economo's encephalitis had high fevers, mental changes, and altered levels of consciousness. Parkinsonian features developed in some of these patients at the time of the encephalitis or shortly after the encephalitis. The clinical features of these patients with postencephalitic parkinsonism differed somewhat from those of patients with idiopathic PD. Patients with postencephalitic parkinsonism had oculogyric crises, unusual body postures, and dystonic features. In addition, the disease did not progress as rapidly as did idiopathic PD. With the exception of these features, postencephalitic parkinsonism resembled idiopathic PD both clinically and pathologically.

Patients with documented postencephalitic parkinsonism account for less than 5% of the patients with PD. The possibility has been raised that idiopathic PD develops because the patients had at one time had contact with the von Economo virus. It has been postulated that PD developed in these patients as a late complication of the viral infection without previous encephalitis. However, this theory was rejected as describing a cause of idiopathic PD.[9] In addition, numerous clinical studies and laboratory investigations have failed to uncover a conventional virus or even a slow virus (similar to those associated with kuru or Creutzfeldt-Jakob disease) as a cause of PD.

Recent interest has centered on environmental toxins as a cause of PD. One epidemiologic finding that links environmental toxins to PD is a lower incidence of cigarette smoking among patients with PD. This suggests that there may be a substance in cigarette smoke that can protect against either an environmental toxin or an endogenously produced chemical that causes PD. A link between pesticides and PD has been suspected, based on the similarity of the pesticide Paraquat™ to the neurotoxin N-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP). Thus, in one part of Quebec where pesticides are in common use, the incidence of PD is seven times that in other parts of Quebec where pesticides are rarely used. If environmental toxins are responsible for PD, then there should be variations in the incidence of PD in different countries, and there should be changes in the incidence of PD with time. Although most investigators have found the incidence of PD to be similar in different countries and in all decades of the 20th century, rates of PD do differ in the Mediterranean, Japan, and China, and in some communities, such as those in Quebec.

A major epidemiologic finding linking PD to environmental toxins is the Parkinson disease–amyotrophic lateral sclerosis complex (PD-ALS) of Guam.[10] This disorder had affected up to 10% of native Guamians (Chamorros). PD-ALS is progressive, with death resulting within a few years of its onset. The clinical features of PD-ALS consist of a mixture of parkinsonism, dementia, amyotrophy, and pyramidal tract dysfunction. In some patients, parkinsonian features predominate, whereas in other patients, ALS features predominate clinically. Most patients have pathologic features of both PD and ALS. The initial discovery of PD-ALS among the Guamian Chamorros led to the theory that the disorder was genetic. However, later discoveries of PD-ALS in other parts of the Pacific—specifically on the Kii Peninsula of Japan and in western New Guinea—and the lack of PD-ALS among the Chamorros living outside of Guam, led to the search for an environmental cause. A number of potential agents were evaluated. Patients with PD-ALS were found to have subtle abnormalities in calcium and magnesium metabolism and to have an increased deposition of calcium and aluminum in their brains. Although a specific environmental toxin has not been identified, changes in the soil and water content of calcium and magnesium on Guam, in Japan, and in New Guinea have resulted in a marked decline in the incidence of PD-ALS.[10] However, there have also been major changes in dietary habits and in the location of the population on Guam, which may be as important in the epidemiology of PD-ALS as the changes in the soil and water content of calcium and magnesium.

MPTP

The greatest finding linking PD to environmental toxins is the identification of the pyridine derivative MPTP as a cause of parkinsonism that is similar clinically to idiopathic PD.[11] The role of MPTP in PD surfaced in 1977, when PD developed in a young drug abuser. Although no cause for the PD could be found, drugs were suspected. The patient continued to abuse drugs and eventually died of an overdose. His autopsy revealed degeneration of the substantia nigra similar to that seen in idiopathic PD, but without Lewy bodies. Subsequently, Langston[11] identified several patients with parkinsonian symptoms who had also been using illicit drugs that contained large quantities of MPTP. This seminal discovery, and subsequent discoveries by Burns et al,[12] were the first definitive evidence that MPTP could result in parkinsonism.[13]

Although at least 400 individuals are known to have injected themselves with MPTP, parkinsonism has developed in only about 20 of them. To ascertain whether the asymptomatic individuals may be at risk for PD, Calne et al[14] used positron emission tomography (PET) with 6-fluoro-levodopa in four patients. The authors found that there was a decline in the formation and storage of 6-fluoro-dopamine in the corpus striata of these patients.

Before a causative agent for PD can be identified and strategies for halting its effects devised, a better understanding of how this agent interacts with the pigmented neurons in the substantia nigra is needed. How does the agent specifically target the pigmented neurons in the substantia nigra? How does the agent cause the neurons to die? Such an understanding may come from studying single neurons with modern electrophysiologic techniques[15] and integrating this information with research using animal models of PD.

The localization of MPTP to dopamine-containing neurons is striking. Why is MPTP selective for these dopamine neurons? Are there specific receptor sites for MPTP on these neurons that attract the compound? Are these receptor sites similar in all animals?[12,13,16] When monkeys are given MPTP, even at low doses, an acute decrease in striatal dopamine develops that is associated with destruction of the monkeys' nigral neurons. Whereas monkeys are sensitive to low doses of MPTP, rodents are less sensitive. Rodents require a higher dose of MPTP than monkeys to cause a similar decrease in their striatal dopamine levels. A major difference between the primate substantia nigra and the rodent substantia nigra is that, in the rodent, the dopamine neurons in the substantia nigra do not contain neuromelanin. Thus, the presence of neuromelanin may be an important factor in determining the neurotoxicity of MPTP.

The neurotoxicity of MPTP was further characterized by Chiba et al.[16] They showed that the enzyme monoamine oxidase "B" (MAO "B"), found in the glia that surround the nigral neurons, converts MPTP to 1-methyl-4-phenylpyridine (MPP+). This catalytic activity of MAO "B" is crucial for the development of MPTP neurotoxicity.[17] MPP+ is selective for the dopamine neurons because, unlike MPTP, MPP+ is accumulated by the catecholamine uptake "pump" of these neurons.[18] The potencies of drugs that block the accumulation of MPP+ by the catecholamine uptake "pump" are identical to the potencies of the drugs in blocking the accumulation of dopamine.

Studies have shown that dopamine neurons accumulate and then concentrate dopamine from the extracellular space by 1,000 to 10,000 times. This ability to accumulate and concentrate dopamine within cells means that these neurons can also accumulate MPP+. Once MPP+ enters the dopamine neuron, it can bind with the intracellular neuromelanin, permitting the retention of large amounts of MPP+ which can then be released gradually. After MPP+ is released from neuromelanin, the mechanism by which MPP+ then damages the neuron may depend on the formation of free radicals. These free radicals, in the form of oxygen or hydrogen peroxide, then attack the cell membranes and start a chain reaction of lipid peroxidation. This chain reaction occurs only after certain protective, fat soluble antioxidants (such as vitamin E) have been depleted. Lipid peroxidation results in extensive damage to the cell membrane and to other intraneuronal molecules.[13]

Whether MPTP, MPP+, or other pyridines (which are abundant in nature) play a role in idiopathic PD is unclear at this time. The study of MPTP has also led to the formulation of new strategies for halting the progression of PD. These strategies include using drugs such as deprenyl, a MAO "B" inhibitor, to prevent the formulation of MPP+, benztropine or mazindol to prevent the uptake of MPP+, and vitamin E to restore intracellular levels of fat soluble anti-oxidants.

TREATMENT OF EARLY PARKINSON DISEASE

While attention is being directed toward an understanding of the basic cause of PD, the problem of treating PD remains. Current medical treatment for PD addresses the symptoms; there is no cure. Drug treatment must be tailored to each patient, to the stage of the disease, and to the presence of complicating factors such as depression and dementia.

The variable clinical expression of PD suggests that there may be subgroups of the disease. One subgroup, an exclusively motor disorder, is characterized by resting tremor, rigidity, and bradykinesia. This subgroup occurs in younger patients, runs a longer and more benign course, and responds better to levodopa.[19] The other subgroup is characterized by bradykinesia, gait impairment, and postural instability without tremor. This subgroup occurs in older patients, may be associated with dementia, runs a more rapid course, and has a poorer response to levodopa.[19] The clinical and pathologic features of the subgroups overlap, which makes it difficult to specify which subgroup affects a given patient.

The subgroups of PD may reflect differences in neurochemistry. In addition to the striatal dopamine deficiency in PD, there is also a dopamine deficiency in the mesolimbic area and in the mesocortex. It is believed that the striatal dopamine deficiency is responsible for the motor symptoms of PD, and the mesolimbic and mesocortical dopamine deficiencies are responsible for the affective and cognitive symptoms of PD. In addition to the dopamine deficiency in PD, there is also a norepinephrine deficiency in the locus ceruleus, nucleus accumbens, and hypothalamus. The norepinephrine deficiency may contribute to the symptoms of the autonomic nervous system and to some of the motor difficulties.

The first decision to be made when seeing a new patient with PD is whether the patient should be treated. If the symptoms are sufficiently disabling as to require treatment, the next question is which drug to use. Levodopa combined with an extracerebral decarboxylase inhibitor is the standard treatment for PD. However, there is controversy about whether levodopa should be the first drug used.[20] The controversy over the use of levodopa centers on whether the proven benefits of levodopa therapy are worth the adverse effects that develop later, including dyskinesias and diurnal fluctuations in performance. The dyskinesias, consisting of a combination of dystonia, chorea, and athetosis, occur in approximately one third to one half of treated patients. The diurnal fluctuations in performance consist of the "wearing off" phenomenon, a gradual loss of efficacy that is related to the timing of the last dose of levodopa, and, less commonly, the "on-off" phenomenon, an abrupt loss of efficacy that is not related to the timing of the last dose of levodopa.[21,22] Both the "wearing-off" and "on-off" phenomena may occur in the same patient. These phenomena may be associated with dyskinesias to varying degrees. Diurnal fluctuations in performance occur in half to two thirds of treated patients. In patients with PD that receive antiparkinsonian drugs such as anticholinergics, amantadine, or dopamine agonists without levodopa, dyskinesias or diurnal fluctuations in performance seldom develop. Therefore, it is assumed that dyskinesias and diurnal fluctuations are related to chronic levodopa treatment and to the dose of levodopa that is used. However, the severity of the underlying disease may be more important than levodopa in the development of the dyskinesias and diurnal fluctuations. Thus, when levodopa is given to patients without PD, dyskinesias or diurnal fluctuations do not develop.

Additional evidence relating the development of dyskinesias and diurnal fluctuations to PD comes from studies of patients with MPTP parkinsonism. In these patients with severe parkinsonism, dyskinesias and diurnal fluctuations in performance develop soon after levodopa therapy is instituted.[23] This suggests that patients with early PD should not be denied the benefits of levodopa treatment.[24,25] An additional reason for beginning levodopa early in PD relates to the biochemical changes associated with PD. Before parkinsonian symptoms appear, the level of striatal dopamine is reduced by at least 80%.[2] This implies that early clinical disease appears after advanced biochemical disease. This relationship has recently been supported with data from PET scanning.[14] Because PD is already advanced by the time the first symptoms appear, little time will be gained by using drugs such as anticholinergic agents and amantadine—which have more adverse effects than levodopa—in place of levodopa. These adverse effects include confusion, difficulty urinating, and orthostatic hypotension.

Levodopa should be the first drug used in treating PD, but the dose should be kept as low as possible. The dose of levodopa that is effective in early PD is between 500 and 750 mg, combined with at least 75 mg of carbidopa.

Levodopa is usually combined with an extracerebral dopa decarboxylase inhibitor. The latter drug blocks the conversion of levodopa to dopamine outside the brain, which enables more levodopa to enter the brain. In addition, extracerebral dopamine is responsible for the nausea and vomiting that is associated with levodopa treatment. As the extracerebral dopa decarboxylase inhibitors do not enter the brain, they do not block the intracerebral conversion of levodopa to dopamine.

There are two extracerebral dopa decarboxylase inhibitors, carbidopa and benserazide, but only carbidopa is available in the United States. For most patients, 75–200 mg of carbidopa is needed to block the extracerebral conversion of levodopa to dopamine.[26] Carbidopa reduces the plasma clearance of levodopa by 50% and doubles the bioavailability of levodopa.[27] The use of carbidopa permits an 80% reduction in the required dose of levodopa. Carbidopa is combined with levodopa (Sinemet™) in a ratio of 1 mg of carbidopa to 4 mg of levodopa (25/100 mg tablet), or a ratio of 1 mg of carbidopa to 10 mg of levodopa (25/250 mg tablet). The 25/100 mg tablet of Sinemet contains the same amount of carbidopa as the 25/250 mg tablet of Sinemet and blocks extracerebral dopa decarboxylase as well as the 25/250 mg tablet. However, since the 25/100 mg tablet contains less levodopa than the 25/250 mg tablet, the 25/100 mg tablet delivers a smaller amount of levodopa to the brain. Using three to four 25/100 mg tablets of Sinemet, extracerebral dopa decarboxylase can be inhibited, and the minimum amount of levodopa needed to treat patients can be used.

Some foods may block levodopa's absorption from the gut or its passage from the blood into the brain. These foods contain large amounts of the amino acids phenylalanine, leucine, and isoleucine that compete with levodopa for transport through the gut and across the blood-brain barrier.[28] Foods such as milk, milk products, lentils, meats, peas, and nuts should be used sparingly during levodopa treatment, especially among patients with diurnal fluctuations.

The tremor seen in PD mainly involves the hands and is present at rest; it usually decreases when the hands are stretched or moved. In a few patients, the tremor may increase when the hands are stretched (sustention or postural tremor), or when the hands are moved (action tremor).[1] The presence of a sustention or action tremor usually reflects a more widespread pathologic disorder. The sustention and action tremors respond less well, if at all, to antiparkinsonian drugs. Occasionally, these tremors may be helped by beta-blockers or by barbituates.

Some patients with resting tremor can also be difficult to treat. In these patients, levodopa has to be combined with an anticholinergic drug, with the phenothiazine ethopropazine (Parsidol™), or with bromocriptine (Parlodel™). Anticholinergic drugs and ethopropazine are less useful than levodopa or bromocriptine for treating the other symptoms of PD. Tremor may not respond to levodopa alone, because tremor may arise from factors other than the striatal dopamine deficiency. These factors can include a striatal acetylcholine imbalance or a defect in thalamic integration. Studies with a neuromagnetometer, a noninvasive magnetic technique that measures the magnetic field around neurons, can be used to clarify which structures in the brain give rise to tremor and what the relationship of resting tremor is to postural and action tremor.[29] Recent work using stereotaxic surgery has reawakened interest in this procedure as a means of controlling refractory tremor.

About half of all patients with PD suffer from depression. In some patients, the depression may be an appropriate psychologic reaction to the disease, while in other patients the depression may be an intrinsic part of the disease—it may be related to a dopamine deficiency in the mesolimbic or mesocortical systems. Clinically, the two kinds of depression cannot be distinguished. In many depressed PD patients, treatment with both an antidepressant and levodopa is necessary. Which antidepressant drug is administered depends on the individual. Occasionally, the depression in PD is so severe as to require electric shock treatment.

In many patients, the response to levodopa begins to decline within six months to one year after starting the drug. In the past, the response to this decline in efficacy had been to increase the dose of levodopa. Inevitably, this leads to the need for further increases in the dose of levodopa. Ultimately, within a year or two, the patient experiences dyskinesias, diurnal fluctuations in performance, and a further decline in response to levodopa.

Several mechanisms may be responsible for the decline in levodopa's efficacy. These include decreased absorption of levodopa from the gut, decreased transportation of levodopa across the blood-brain barrier, decreased ability of the nigral neurons to change levodopa to dopamine or to store dopamine, and decreased sensitivity of the postsynaptic dopamine receptors. As the exact mechanism is not known, treatment remains empirical. Treatment may consist of increasing the dose of levodopa, changing the ratio of levodopa to carbidopa, or adding amantadine. Amantadine may be effective in up to half of patients, but these benefits are temporary.

The best approach, at present, for patients whose response to levodopa is declining, is to leave them on a relatively low dose of levodopa (500–1,000 mg/day) and then to add a dopamine agonist such as bromocriptine. Bromocriptine, in low doses (less than 30 mg/day), combined with levodopa is effective early in PD.[30] Many neurologists will even add bromocriptine to levodopa before the response to levodopa declines. Bromocriptine is begun at a dose of 1.25–2.5 mg/day and is built up gradually in increments of 1.25–2.5 mg/day each week, to a maximum of 15–30 mg/day. Combination therapy is important in PD, because it maximizes the antiparkinsonian effects of each drug, while minimizing the drugs' adverse effects. Indeed, recent work suggests that there may be a distinct biochemical basis for combining bromocriptine with levodopa.[31] It has been shown that the activity of bromocriptine is enhanced by the presence of striatal dopamine, such as that formed from levodopa. The striatal dopamine may induce a conformational change in the dopamine receptor, making the receptor more responsive to bromocriptine.

In patients with mild to moderate PD, a good therapeutic response with few adverse effects may be achieved by a low dose of bromocriptine, less than 30 mg/day.[32] Patients with advanced PD usually require more than 30 mg/day. After two years, bromocriptine's efficacy, like levodopa's, may decline.[33] This decline in efficacy is more pronounced in patients with advanced PD.

Bromocriptine may also be used as the only treatment for PD. In patients treated with bromocriptine alone, dyskinesias or diurnal fluctuations in performance do not develop. Because an antiparkinsonian effect takes longer to achieve with bromocriptine than with levodopa, and because, eventually, most patients on bromocriptine also require levodopa, bromocriptine is seldom used as the only treatment for PD.

Among patients whose response to levodopa has declined and who are experiencing dyskinesias and diurnal fluctuations in performance, the question of doing a levodopa drug holiday arises. The basis of the levodopa drug holiday is the assumption that chronic levodopa treatment leads to the accumulation of toxic metabolites in the brain, or that levodopa desensitizes the postsynaptic dopamine receptors. Indeed, some patients do experience temporary improvement after completing the 3–14-day drug holiday. However, the improvement usually lasts only a few days, seldom longer, and many patients do not benefit at all. Furthermore, the levodopa drug holiday is not as benign as it sounds. During the holiday, the patient experiences a baseline PD state—the state the patient would have been in were it not for the levodopa. The patient may become bed-bound or severely depressed, have difficulty swallowing, aspirate, and suffer from pneumonia or phlebitis. Levodopa drug holidays, if they are used, should be done only in a hospital.[34]

For patients with advanced PD who are doing poorly and are already on levodopa and low-dose bromocriptine therapy, several treatment strategies can be considered. One strategy is to increase the dose of bromocriptine. In high doses (31–75 mg/day), bromocriptine often improves the condition of those patients whose response to levodopa and low-dose bromocriptine has declined. This dose of bromocriptine permits a 10% to 25% reduction in levodopa.[32,35] The reduction in levodopa often results in fewer dyskinesias. Another strategy is to use a different dopamine agonist. Two experimental dopamine agonists are now undergoing trials. Pergolide is a semisynthetic ergot, which, when combined with levodopa, reduces disability and increases the number of hours patients are "on."[36] The daily dose of pergolide is 2.5 mg (range, 0.2–10 mg/day). As with other antiparkinsonian drugs, pergolide's antiparkinsonian effect may decline within six months to one year.[37] The adverse effects of pergolide are similar to those of bromocriptine. Lisuride, like pergolide and bromocriptine, reduces disability and increases the number of hours patients are "on." However, tolerance to the antiparkinsonian effect of lisuride also develops.[38]

Improving the methods of using dopamine agonists and developing new agonists will require a better understanding of dopamine receptors. There are dopamine receptors in the striatum, in the hypothalamus, in the mesolimbic area, and in the mesocortex. In the striatum there are D1 and D2 receptors.[39] The D1 receptors are linked to the enzyme adenyl cyclase; the D2 receptors are not linked to this enzyme. The D1 and D2 receptors may exist in both a low or a high affinity binding state. The status of the receptors' binding state can determine the efficacy of the dopamine agonist. In addition to the postsynaptic striatal receptors, there are also dopamine receptors on the presynaptic nigrostriatal axons (D3 receptors). Stimulation of or blockade of these presynaptic dopamine receptors results in either feedback inhibition or feedback augmentation of the nigral dopamine neurons. The specific function of the striatal D1 and D2 dopamine receptors in mediating the antiparkinsonian effect of a particular dopamine agonist is not known. The availability of selective agonists and antagonists of D1 and D2 receptors will result in more selective drugs with fewer adverse effects.

Recent work has focused on a class of drugs called partial agonists. These drugs are weak agonists that exert their effects at the intact, nondenervated dopamine receptors. Thus, when these drugs stimulate the presynaptic dopamine receptors (D3 receptors) they result in feedback inhibition of dopamine synthesis. These drugs may be useful in disorders associated with a relative excess of dopamine, such as schizophrenia. However, the drugs are also powerful agonists at the denervated, postsynaptic dopamine receptors and hence may be especially useful in patients with advanced PD.[40,41] Partial agonists may also result in fewer mental changes and dyskinesias. Two such partial dopamine agonists, terguride and BHT 920, are now undergoing clinical trials.

Another approach toward augmenting the effect of levodopa is to combine levodopa with a monoamine oxidase (MAO) inhibitor.[42,43] There are two kinds of MAO: type A, which exists outside the brain, and type B, which exists inside the brain. The MAO inhibitors now available in the United States are nonselective and inhibit both MAO A and MAO B. As a result of inhibition of MAO A, the dietary amine tyramine is able to gain access to the circulation. Because an average serving of some foods (especially cheeses) contains enough tyramine to markedly increase blood pressure, ingestion of such food by patients receiving nonselective MAO inhibitors can lead to hypertensive crisis. In addition, the use of nonselective monoamine inhibitors is contraindicated in patients receiving drugs such as meperedine.

Deprenyl is a selective inhibitor of MAO B. Inhibition of MAO B does not result in hypertensive effects following the ingestion of foods rich in tyramine. Deprenyl, in combination with levodopa, has been shown to be effective in PD. It may even delay the progression of PD.[43] In one large retrospective study, patients with PD who received deprenyl and levodopa lived longer than patients who received levodopa without deprenyl. Several prospective studies are now under way to test whether or not deprenyl slows the progression of PD.

Another experimental approach toward treating patients with advanced PD and diurnal fluctuations in performance is to use a continuous intravenous infusion of levodopa, a controlled-release Sinemet™ preparation, or an infusion of a soluble dopamine agonist such as lisuride.[44,45] However, not all patients with diurnal fluctuations respond to such infusions or the controlled-release preparations. This suggests that the level of striatal dopamine is not always related to the level of plasma levodopa, and that the striatal dopamine level cannot be corrected.

Brain transplants, seemingly the stuff of science fiction, are now practical tools for studying PD.[46] Recent experiments have shown that pieces of fetal substantia nigra can be transplanted into the brains of recipient animals under certain conditions. The fetal nigral transplants form appropriate connections with their target cells in the striatum and can reverse parkinsonian symptoms. Interest has also centered on grafting autologous tissue from the body of the host into the host's brain. Thus, in the parkinsonian rat, chromaffin cells from the adrenal medulla—which have the capacity to synthesize catecholamines—have been transplanted into the rat's brain. The transplant partially reverses the rat's parkinsonism. Autologous transplantation of adrenal medulla tissue into the brain has also been accomplished in four patients with advanced PD (A. Bjorkland, personal communication, June 1986). In these patients, approximately half of one adrenal medulla was grafted onto the patient's caudate nucleus using stereotaxic techniques. The operations were without complications; improvement, however, has ranged from none to mild.

REFERENCES

1. Lieberman AN: Parkinson's disease: A clinical review. *Am J Med Sci* 1974; 267:66–80.
2. Bernheimer H, Birkmayer W, Hornykiewicz O, et al: Brain dopamine and the syndromes of Parkinson and Huntington. Clinical, morphological and neurochemical correlations. *J Neurol Sci* 1973; 20:415–455.
3. Banta RG, Markesbery WR: Elevated manganese levels associated with dementia and extrapyramidal signs. *Neurology* 1977; 27:213–216.
4. Klawans HL, Stein RW, Tanner CM, et al: A pure parkinsonian syndrome following acute carbon monoxide intoxification. *Arch Neurol* 1982; 39:302–304.
5. McLean DR, Jacobs H, Mielke BW: Methanol poisoning: A clinical and pathological study. *Ann Neurol* 1980; 8:161–167.
6. Uitti RJ, Rajput AH, Ashenhurst EM, et al: Cyanide-induced parkinsonism: A clinicopathologic report. *Neurology* 1985; 35:921–925.
7. Casson IR: Neurologic syndromes in boxers. *Neuroview* 1985; 1:1–4.
8. Ward CD, Duvoisin RC, Ince SE, et al: Parkinson's disease in 65 pairs of twins and in a set of quadruplets. *Neurology* 1983; 33:815–824.
9. Kurland L, Hausen W, Okazaki H: Epidemiologic studies of parkinsonism with special reference to the cohort hypothesis. Proceedings of the Third Symposium on Parkinsonism. Edinburgh, Livingtone Ltd, 1969, pp 12–16.
10. Gannuto RM, Yanagihara R, Gajdusek DC: Disappearance of high-incidence amyotrophic lateral sclerosis and parkinsonism-dementia on Guam. *Neurology* 1985; 35:193–198.
11. Langston JR, Ballard P, Tetrud JW: I. Chronic parkinsonism in humans due to a product of meperidine-analog synthesis. *Science* 1983; 219:979–980.
12. Burns RS, Chiueh CC, Markey SP, et al: A primate model of parkinsonism: Selective destruction of dopaminergic neurons in the pars compacta of the substantia nigra by N-methyl-4-phenyl-1,2,3,6-tetrahydropyridine. *Proc Natl Acad Sci USA* 1983; 80:4546–4550.
13. Snyder SH, D'Amito RJ: MPTP: A neurotoxin relevant to the pathophysiology of Parkinson's disease. The 1985 George C. Cotzias Lecture. *Neurology* 1986; 36:250–258.
14. Calne DB, Langston JW, Martin WRW: Positron emission tomography after MPTP: Observations relating to the cause of Parkinson's disease. *Nature* 1983; 317:246–248.
15. Llinas R, Greenfield SA, Jahnsen H: Electrophysiology of pars compacta cells in vitro substantia nigra—a possible mechanism for dendritic release. *Brain Research* 1984; 294:127–132.
16. Chiba K, Trevor A, Castagnoli N Jr: Metabolism of the neurotoxic tertiary amine, MPTP, by brain monoamine oxidase. *Biochem Biophys Res Commun* 1984; 120:574–578.
17. Heikkila RE, Manzino L, Cabbat FS, et al: Protection against the dopaminergic neurotoxicity of 1-methyl-3-phenyl-1,2,5,6-tetrahydropyridine by monoamine oxidase inhibitors. *Nature* 1984; 311:467–469.
18. Javitch JA, D'Amato RJ, Strittmatter SM, et al: Parkinsonism-inducing neurotoxin, N-methyl-4-phenyl-1,2,3,6-tetrahydropyridine: Uptake of the metabolite, N-methyl-4-phenylpyridine by dopamine neurons explains selective toxicity. *Proc Natl Acad Sci USA* 1985; 82:2173–2177.
19. Lieberman A, Dziatolowski M, Kupersmith M, et al: Dementia in Parkinson disease. *Ann Neurol* 1979; 6:355–359.
20. Lesser RP, Fahn S, Snider SR, et al: Analysis of the clinical problems in parkinsonism and the complications of long-term levodopa therapy. *Neurology* 1979; 29:1253–1260.
21. Marsden CD, Parkes JD: Success and problems of long-term levodopa therapy in Parkinson's disease. *Lancet* 1977; 1:345–349.
22. Marsden CD, Parkes JD: "On-off" effects in patients with Parkinson's disease of chronic levodopa therapy. *Lancet* 1976; 1:292–296.
23. Burns RS, Lewitt PA, Ebert MH, et al: The clinical syndrome of striatal dopamine deficiency. Parkinsonism induced by 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP). *N Engl J Med* 1985; 312:1418–1421.
24. Markham CH, Diamond SG: Evidence to support early levodopa therapy in Parkinson disease. *Neurology* 1981; 31:125–131.
25. Muenster MD: Should levodopa therapy be started early or late? *Can J Neurol Sci* 1984; 11:195–199.
26. Jaffe ME: Clinical studies of carbidopa and L-dopa in the treatment of Parkinson's disease. *Adv in Neurology* 1973; 2:161–172.
27. Nutt JG, Woodward WR, Anderson JL: The effect of carbidopa on the pharmacokinetics of intravenously administered levodopa: The mechanism of action in the treatment of parkinsonism. *Ann Neurol* 1985; 18:537–543.
28. Nutt JG, Woodward WR, Hammerstad JP, et al: The "on-off" phenomenon in Parkinson's disease. *N Engl J Med* 1984; 310:483–488.
29. Okada YC, Tannenbaum R, Williamson SJ, et al: Somatotopic organization of the human somatosensory cortex revealed by neuromagnetic measurements. *Exp Brain Res* 1984; 56:197–205.
30. Rinne UK: Combined bromocriptine-levodopa therapy early in Parkinson's disease. *Neurology* 1985; 35:1196–1198.
31. Goldstein M, Lieberman A, Meller E: A possible molecular mechanism for the antiparkinson action of bromocriptine in combination with levodopa. *Trends in Pharmacological Sciences* 1985; 6:436–437.
32. Lieberman AN, Goldstein M: Bromocriptine in Parkinson disease. *Pharmacol Rev* 1985; 37:217–227.
33. Lieberman A, Kupersmith M, Neophytides A, et al: Long-term efficacy of bromocriptine in Parkinson disease. *Neurology* 1980; 30:518–523.
34. Mayeux R, Stern Y, Mulvey K, et al: Reappraisal of temporary levodopa withdrawal ('drug holiday') in Parkinson's disease. *N Engl J Med* 1985; 313:724–728.
35. Lieberman A, Kupersmith M, Estey E, et al: Treatment of Parkinson's disease with bromocriptine. *N Engl J Med* 1976; 295:1400–1404.
36. Lieberman A, Goldstein M, Leibowitz M, et al: Treatment of advanced Parkinson disease with pergolide. *Neurology* 1981; 31:675–682.

37. Lieberman A, Goldstein M, Leibowitz M, et al: Long-term treatment with pergolide: Decreased efficacy with time. *Neurology* 1984; 34:223–226.
38. Lieberman A, Goldstein M, Gopinathan G, et al: Further studies with lisuride in Parkinson's disease. *Eur Neurol* 1983; 22:119–123.
39. Kebabian JW, Calne DB: Multiple receptors for dopamine. *Nature* (Lond) 1979; 277:93–96.
40. Fuxe K, Goldstein M, Agnoti LF: Interactions of a partial ergoline with dopamine receptors in vivo and in vitro. *Acta Physiol Scan* 1983; 117:303-305.
41. Corsini GU, Bonuccelli U, Rainer E, et al: Therapeutic efficacy of a partial dopamine agonist in drug-free parkinsonian patients. *J Neural Transm* 1985; 64:105–111.
42. Lieberman AN, Gopinathan G, Neophytides A: Deprenyl in the treatment of Parkinson's disease. *NY State J Med* 1984; 84:13–16.
43. Birkmayer W, Knoll J, Riederer P, et al: Increased life expectancy resulting from addition of L-deprenyl to Madopar treatment in Parkinson's disease: A long term study. *J Neural Transm* 1985; 64:113–127.
44. Quinn N, Parkes JD, Marsden CD: Control of on/off phenomenon by continous intravenous infusion of levodopa. *Neurology* 1984; 34:1131–1136.
45. Obeso JA, Luguin MR, Martinez Lage JM: Intravenous lisuride corrects oscillations of motor performance in Parkinson's disease. *Ann Neurol* 1986; 19:31–35.
46. Freed WJ, Perlow MJ, Karoun F, et al: Restoration of dopaminergic function by grafting of fetal rat substantia nigra to the caudate nucleus: Long-term behavioral, biochemical, and histochemical studies. *Ann Neurol* 1980; 8:510–519.

See also pp 139–140.

FROM THE LIBRARY

PARKINSONISM—IS IT A SURGICAL PROBLEM?

Medicinal and physical therapeutic measures for the alleviation of tremor and rigidity of Parkinson's disease have resulted in moderate, but usually transitory, improvement in some patients. In others, little benefit has been derived from these therapies. Failure to effect a cessation of the tremors, in particular, by these so-called conservative means has stimulated surgeons to explore the possibility of favorably modifying this distressing symptom by operation . . . .

From the reports available in the literature and my own observations it seems reasonably well established that the tremor as well as the rigidity of the disease under consideration *may* be modified by (*a*) excision of a part of the premotor cortex, (*b*) undercutting of the premotor area, (*c*) division of the anterior limb of the internal capsule, (*d*) section of the pallidofugal fibers, or (*e*) interruption of the lateral pyramidal tract at the second cervical segmental level. Whether or not any of these surgical procedures result in sufficient symptomatic improvement to justify their employment has been questioned by many. In truth, even neurologic surgeons frequently have raised just this issue and, it seems to me, rightly so. Therefore, it becomes necessary that those actively interested in surgical therapy for parkinsonism must report their results with a more objective attitude lest this therapy fall into disrepute.

Certainly one should not consider, except in the most unusual circumstances, paralyzing a limb in order to deprive it of a tremor. Nor should one subject a patient to a major surgical procedure without reasonable possibility of rehabilitation. Furthermore, the operation proposed should be one that can be executed successfully by anyone familiar with cerebral surgery.

I have had the opportunity to observe 33 patients with parkinsonism upon whom either a spinal or cerebral operation had been performed. Four of these had had a part of the premotor cortex removed by Klemme, 4 had a section of the lateral pyramidal tract carried out by Putnam, 12 had been operated upon by Meyers while he was associated with me, using a variety of procedures, that is, undercutting of the premotor cortex and removal of a part of the caudate head in 2 cases; extirpation of the head of the caudate nucleus and section of the oral half of the anterior limb of the internal capsule, 4 cases; extirpation of the head of the caudate nucleus and oral thirds of the putamen and globus pallidus and interruption of the fibers running in the oral fourth of the anterior limb of the internal capsule, 1 case; and section of the pallidofugal fibers, 5 cases.

The remaining 13 patients were operated upon by me. In each of these 13, the dorsal half of the head of the caudate nucleus was first removed and the fibers of the anterior limb of the internal capsule sectioned up to a few millimeters of the genu. Only unilateral operations were performed in this group.

Although this series of 33 cases is admittedly relatively small . . . , yet the results as I have observed them will be reviewed briefly but pointedly. Of the 4 operated upon by Klemme, 3 had bilateral tremor and rigidity prior to operation. Following a unilateral operation in each there resulted a marked spastic hemiparesis without rhythmical tremor in 2 cases and, in the third, a spastic hemiparesis with occasional outbursts of irregular tremor on the paretic side. The remaining patient had what may be termed unilateral parkinsonism. He was rehabilitated completely at the time I saw him, eighteen months after operation . . . .

JEFFERSON BROWDER, MD
(*NY State J Med* 1947; 47:2589–2592)

Evaluation and management of chronic aspiration

ANDREW BLITZER, DDS, MD

Intermittent or persistent aspiration or soilage of the airway may produce life threatening pulmonary disease. Slight soilage of the airway occurs in most people during sleep, but aspirated bacteria and foreign material are cleared from the lungs. Viral infections, ethanol ingestion, and altered states of consciousness can increase the quantity of material aspirated, decrease the clearing response, and therefore increase the severity of the consequences. As the amount of damage and the mechanical impairment (including tracheostomy) or neurologic disorder increases, the quantity and frequency of aspiration increases.[1-4]

The effects of aspiration depend on the quantity and pH of the aspirate and on the status of the pulmonary system. Aspiration of a large quantity of low pH material produces an intense bronchospasm, leading to hypotension, hypoxia, and hypercarbia secondary to the pulmonary edema caused by direct capillary damage. A smaller quantity of aspirate results in fever, bronchopneumonia, and/or atelectasis.

Chronic aspiration is due to a neuromuscular or mechanical inability to swallow feedings or secretions while the airway is being protected. Chronic intermittent soilage of the airway produces pulmonary disease. Evaluation of patients with this disorder should be expeditious, with clarification of the cause of aspiration and establishment of a treatment plan.[5-7]

HYPERSALIVATION

True hyperfunction of the salivary glands is uncommon and rarely produces aspiration, but rather an increased swallowing effort and occasional drooling or choking. The swallowing mechanism is totally intact, but the quantity of saliva is increased. The normal basal flow rate of secretion from a major salivary gland is approximately 0.03 mL/min, with a stimulated flow rate of 0.7 mL/min. True hypersalivation can be attributed to stomatitis, heavy metal intoxication, iodine or ammonium salts, anticholinesterases, or neurologic disease. Patients with altered endocrine states—including pregnancy or menopause—malnutrition, or alcoholism may also exhibit sialorrhea. Some psychiatric patients will also display symptoms of this condition. Diagnostic evaluation should include cannulation of the major salivary ducts with measurement of flow rates and salivary analysis. Pharmacotherapy is most effective in normalizing the levels of saliva. Anticholinergic agents such as glycopyrrolate or atropine in divided doses can control symptoms. Some patients may even require low-dose radiation therapy to decrease the quantity of secretion without medication.[8,9]

RELATIVE HYPERSALIVATION

Patients with relative hypersalivation have normal flow rates of saliva, but have diminished ability to handle their secretions due to mechanical or neurologic disorders that produce drooling and/or aspiration. This group includes patients with cerebral palsy, myasthenia gravis, amyotrophic lateral sclerosis (ALS), myopathies, cerebrovascular accidents, mechanical disorders associated with tumors (benign or malignant), fibro-osseous disease, status-post surgical resections of the head and neck for cancer, trauma, status-post caustic ingestion, or congenital malformations.

Treatment of relative hypersalivation should be geared toward trying to reverse the underlying cause of the mechanical disability. Drying agents rarely help, although as an adjunct therapy they may decrease symptoms. Low-dose radiation has also been used as an adjunct with limited success in some patients. Surgeons have attempted salivary duct repositioning in an attempt to make swallowing of secretions easier. This has had limited success. Sectioning of the parasympathetic nerve supply through a tympanic neurectomy and/or chorda tympanic neurectomy—a relatively simple procedure—is most effective in decreasing salivary production while the patient's mechanical function is returning.[8-11]

THE SWALLOWING MECHANISM

The swallowing mechanism depends on a series of events that must occur in a precisely coordinated fashion. If the events do not occur in a synchronized fashion, there will be nasal reflux, choking, aspiration, or regurgitation.

Phase I. During the oral phase of swallowing, the tongue prepares the bolus for swallowing. The tongue mixes the food with saliva and sorts out the large particles to be chewed again. It then forms the bolus against the palate and coats it with mucus. The tongue is pressed

---

Address correspondence to Dr Blitzer, Director, Division of Head and Neck Surgery, Department of Otolaryngology, College of Physicians and Surgeons, Columbia University, 630 West 168th St, New York, NY 10032.

against the palate, squeezing the bolus into the oropharynx. The soft palate is elevated, which seals the nasopharynx and prevents nasal reflux. When the bolus reaches the vallecula, elevation of the larynx is initiated.[12]

Phase II. The pharyngeal phase occurs after the bolus has passed the faucial pillars, and is a completely involuntary action. Respiration is inhibited during this phase. As the base of the tongue has moved posteriorly, the epiglottis has tipped postero-inferior at the same time that the larynx has been raised by the supraglottic musculature. The bolus is then deflected in a posterior and lateral direction away from the larynx by the epiglottis. As the bolus descends toward the hypopharynx, the airway is protected by the epiglottis and the aryepiglottic folds and, more importantly, by the closure of the glottis.[5,12-14]

Failures in these processes can be overcome by addressing the underlying defect. Patients with unilateral hypoglossal nerve paralysis usually do not have any noticeable functional deficit in the swallowing effort. Damage to both hypoglossal nerves, a myopathic process, and central nervous system disorders such as cerebrovascular accident or ALS can all severely limit swallowing and speech. Swallowing therapy can sometimes improve the situation, or patients can be taught to feed by gavage, bypassing the oral phase. Treatments for velopharyngeal insufficiency with nasal reflux include augmentation of the soft palate or posterior pharyngeal wall, prosthetic devices, or a pharyngeal flap. The epiglottis is helpful in swallowing but is not crucial, since many patients can be taught to swallow again after epiglottectomy or supraglottic resection. Treatment of swallowing disorders involving phases I and II can include feeding by gavage (with a bulb syringe and rubber tube) or nasogastric tube. However, patients with nasogastric tubes will often experience reflux around the tube or continue to aspirate saliva.[15,16]

Phase III. The esophageal phase of swallowing may fail because of an inability to open the upper esophageal sphincter or to contract the esophagus itself. Kirchner[17] studied the upper esophageal sphincter, or cricopharyngeal muscle, in dogs, using endoscopy, roentgenographic studies, and pressure recordings. He found that the cricopharyngeal muscle was in the state of contraction during the resting phase. During swallowing, the muscle relaxes, and then contracts to a pressure equal to or greater than the resting pressure. Bilateral vagal section abolishes the relaxation phase, which returns with electrical stimulation of the vagus. Sympathetic stimulation causes a marked increase in pressure. Kirchner therefore concluded that relaxation was a parasympathetic function, and that contraction was a sympathetic function. Conditions that damage vagal function, such as basilar artery thrombosis, will interfere with relaxation of the cricopharyngeal muscle, causing cricopharyngeal achalasia (Fig 1) and dysphagia with aspiration. Neuropathic and myopathic conditions can produce relative cricopharnygeal achalasia or a dysynchrony. A pharyngeal pressure of 15-23 mm Hg is normally exerted by the cricopharyngeal muscle, and must be overcome by pharyngeal pressure to induce opening of the sphincter.

Evaluation of patients should include fiberoptic laryngoscopy, since this technique offers a better functional assessment of the swallowing effort than other types of laryngoscopy. A cine-esophagram is important in assessing a disability in phase I, II or III of swallowing. In anteroposterior and lateral projections, the swallowing effort can be analyzed frame by frame for adynamic areas, relative obstructions, and dysynchrony. Manometric studies are also useful in evaluating functional disabilities in swallowing, including relative cricopharyngeal achalasia.[18]

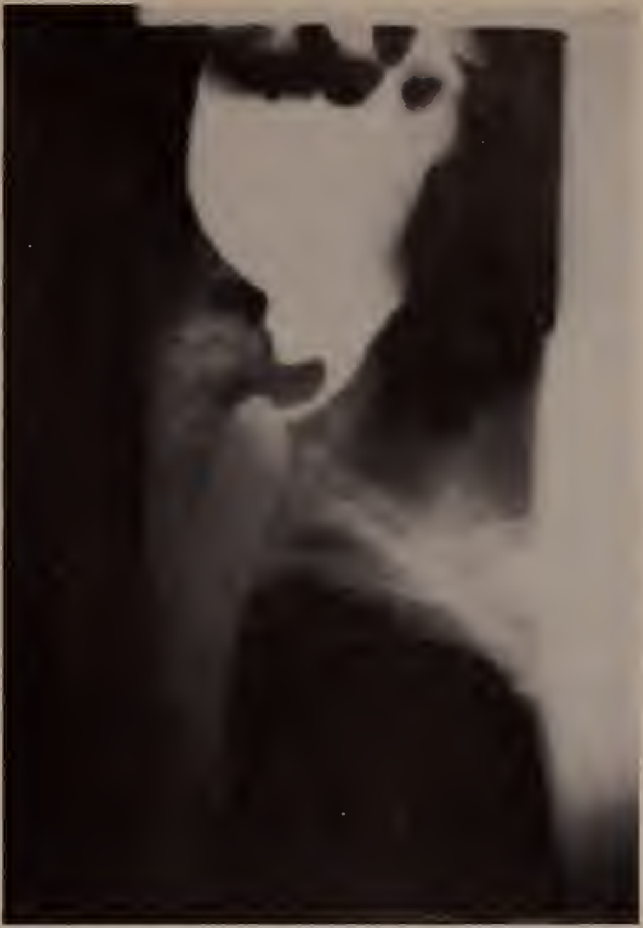


FIGURE 1. Esophagram showing cricopharyngeal achalasia.

If there is true cricopharyngeal achalasia or relative cricopharyngeal achalasia, a cricopharyngeal myotomy will often help correct the dysphagia or aspiration. Some authors have cited cricopharyngeal myotomy as the treatment of choice for dysphagia caused by various neurologic conditions. Cricopharyngeal myotomy is only a treatment for true and relative cricopharyngeal achalasia. Those patients with more generalized mechanical failure, such as occurs in ALS, brainstem stroke, or myopathies, and who have failure in all the phases of swallowing, will probably not benefit from such a procedure.[19-28]

Primary peristalsis of esophageal striated muscles is mediated by vagal cholinergics. The peristaltic action can be reduced or abolished by curare and succinyl choline in the proximal esophagus and atropine in the distal esophagus. Pharmacologic agents can be used to modify esophageal motility. Most of the research with pharmacologic agents has been done in patients with gastroesophageal reflux and has been aimed at increasing pressures at the lower esophageal sphincter and increasing peristaltic contractions. The two most common agents are bethanecol (which increases esophageal contractility, but also increases the gastric acid secretions) and metoclopramide hydrochloride (a potent anti-emetic, which has been found to increase contractility without an increase in gastric acidity).[29,30]

Patients with Parkinson disease can have a tremulous voice pattern and difficulty swallowing. These patients have a lengthened phase I due to lingual motility disorders, a lengthened phase II with vallecular stasis, and a lengthened phase III. These findings are not consistent with those of benign essential tremor. Patients with diabetes have also been found to have weak peristaltic action and poor sphincter pressure.

Phase III of swallowing can also be disrupted in patients with acute and chronic changes from caustic ingestion. These changes often lead to aspiration due to a decrease in sensation, fibrosis, and a dysynchronous swallowing effort. The effects of caustic ingestion are difficult to treat and may be irreversible. In severe forms, esophageal bypass using mobilized stomach, or small or large bowel, may be the only way of enabling swallowing and preventing aspiration.

ESOPHAGEAL DISORDERS

Reflux. The presence of a large hiatus hernia or a nasogastric feeding tube may allow reflux of gastric contents and possible aspiration. Reflux of gastric acid has also been associated with cricopharyngeal spasm. This presents as a "globus" or sensation of a ball in the throat at the level of the cricoid cartilage, and, occasionally, hoarseness due to inflammation of the posterior part of the larynx. Patients with this condition should be treated in a manner similar to patients with reflux esophagitis. Awareness of this entity and early diagnosis will avoid aspiration.

Zenker's or pulsion diverticulum can also present with recurrent aspiration. Patients often have little difficulty swallowing, but have reflux of much of the material seconds to minutes after the swallowing effort. Diagnosis is easily made based on barium swallow studies. Patients are best treated by excision of the diverticula as well as a cricopharyngeal myotomy.[2,30]

Obstruction. Partial or total esophageal obstruction often presents with dysphagia and intermittent aspiration. Acute onset is usually related to foreign bodies. A more chronic onset is found in patients with esophageal webs, strictures, achalasia, or narrowing related to collagen-vascular diseases. Esophageal carcinomas also present with progressive dysphagia and intermittent aspiration (Fig 2).

Diagnosis is usually made with radiographic and endoscopic techniques. Treatment is aimed at attempting to regain and maintain esophageal patency. Gastrostomy alone is not enough, since patients will continue to aspirate saliva. Techniques described for treating relative hypersalivation can be attempted in addition to gastrostomy. At times, an intraluminal bypass tube can safely be placed. In cases unresponsive to treatment, esophageal bypass can be performed using a gastric advancement with an anastomosis to the hypopharynx, or a colon or jejunal interposition from hypopharynx to stomach. This may allow the maintenance of oral feedings and may prevent aspiration.[31,32]

Tracheoesophageal Fistula. Tracheoesophageal fistulas, excluding the congenital variety, are most commonly encountered in patients with esophageal carcinoma. These fistulas can be life threatening due to the resultant constant soilage of the airway.

Tracheoesophageal (TE) fistula is often considered in patients who cough food material through their tracheotomy tube while eating. The incidence of TE fistula after tracheotomy was found to be 0.01% in a large series by LeBrigand and Roy.[33] Most of these cases were related to overinflated cuffs in patients on ventilatory support, rather than to direct surgical trauma.

The majority of patients who cough food out of their tracheotomy tubes have altered laryngeal function, allowing food to pass through the vocal cords into the trachea and then provoke a cough. The tracheotomy tube often prevents or compromises elevation of the larynx during swallowing; because of this relatively fixed inferior position, the epiglottis and aryepiglottic folds cannot provide adequate glottic closure. Food collects over the vocal cords and, on glottic opening, soils the airway. Swallowing is also more difficult since pharyngeal pressures may be diminished due to a leak through the tracheostomy. Food material may then pool in the hypopharynx and spill into the airway intermittently. The adductor reflex closure of the glottis is significantly altered after tracheostomy, with a nearly doubled threshold along with a delay in conduc-

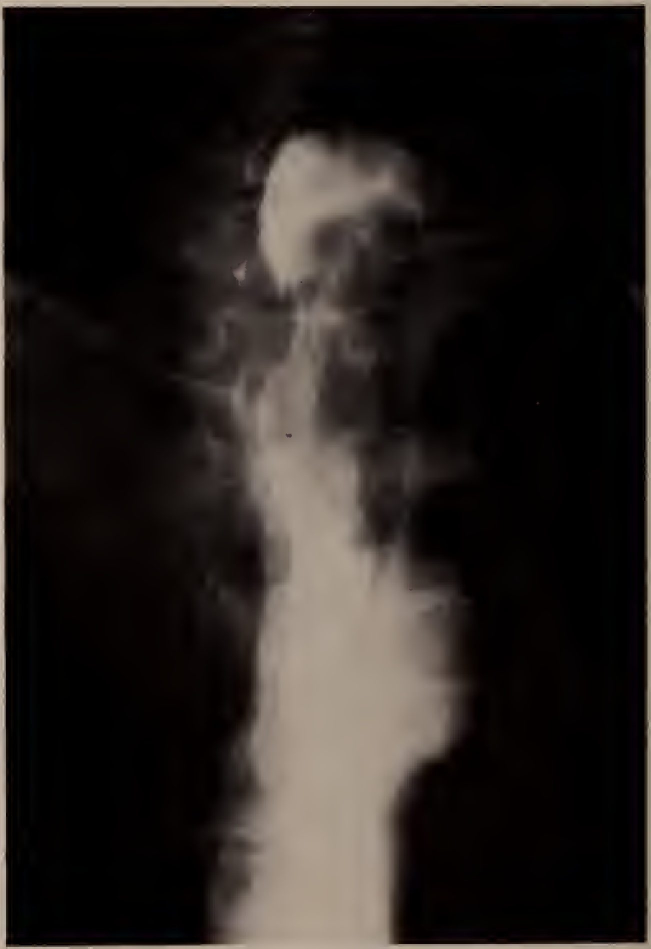


FIGURE 2. Esophagram showing esophageal obstruction due to an esophageal carcinoma.

tion. The change in reflex closure causes uncoordinated movements of the larynx, making soilage of the airway common. Normal function often returns after decannulation.[34]

If a TE fistula is documented, it must be dealt with as an emergency. The airway must be secured, the food passage must be diverted or repaired, and the mediastinum must be drained or kept unsoiled.

In a patient with esophageal carcinoma and a low TE fistula, there is often no solution, since providing a distal airway may be impossible. A higher linkage may be feasible with surgery. A tracheotomy will allow a cuffed anode tube to be inserted to secure the airway below the fistula. An esophageal bypass tube may give temporary relief. If this is not possible, a more definitive gastric pull-up or colonic bypass may be necessary as a palliative effort.

In all procedures aimed at correcting a TE fistula, the primary goal should be to separate the trachea and the esophagus, which share a common wall. Small holes will allow the separation and closure of both mucosal defects with the interposition of a muscle pedicle between the areas of repair. If the hole is larger, or there is evidence of infection, the airway and the esophagus should first be separated. The proximal end of the esophagus can be diverted through the neck skin as an esophagostomy and drained into a stoma bag. The distal esophagus can be oversewn. The trachea can then be repaired using primary closure, or local and regional flaps. If the patient's condition improves, a second operation can be performed to either reconstruct the esophagus with regional tissue or bypass it.[35,36]

LARYNGEAL INCOMPETENCE

The larynx is the portal of the airway and must be relatively competent as a sphincter to prevent aspiration. Closure of the larynx is initiated voluntarily when phonating or swallowing, or during a Valsalva's maneuver. It occurs involuntarily when material comes in contact with the supraglottic structures. The sensory afferent supply is through the superior laryngeal nerve, triggering a reflex adduction of the vocal cords. Unilateral stimulation causes a bilateral response. Conditions that interfere with the sensory afferents of the superior laryngeal nerve may prevent detection of foreign material in the supraglottic and glottic areas, resulting in failure to trigger laryngeal closure. The material will then proceed through the glottis and will not be detected until it reaches the sensory fibers of the recurrent laryngeal nerve which innervate the undersurface of the vocal cord and the subglottis. This will trigger a cough response. If these fibers are also not functional, a cough will not be produced until the vagal fibers of the trachea are stimulated, where there is considerable crossover.

Motor dysfunction of the larynx will also allow aspiration, and may be related to neuromuscular or mechanical impairment. Conditions producing unilateral laryngeal paralysis may produce incomplete glottic closure, thereby allowing aspiration. Recurrent laryngeal nerve damage from tumor, trauma, or infection can produce an open glottic chink. Many of these patients compensate for the dysfunction in that the paralyzed cord assumes a more medial position and the contralateral cord becomes adducted beyond the midline. The most important part of the closure is in the posterior region, since many patients can have part or all of a vocal cord resected for cancer, producing an open glottic chink in the anterior region, and have little or no aspiration. If an arytenoid cartilage is removed, or if there is damage to the posterior commissure, aspiration may become overwhelming.

As a lesion of the vagal nerve moves proximally, the disability markedly increases. Lesions at the base of the skull and other vagal injuries that produce dysfunction of both the superior and the recurrent laryngeal nerves produce widespread damage. Central lesions such as brain stem strokes produce disabilities far worse than vagal lesions alone.

Aspiration can also be due to mechanical disability of the larynx, leaving an open glottic chink. Congenital, traumatic, or surgical deficiencies in vocal cord volume, either uniform or segmental, can allow for aspiration. Traumatic dislocation of the cricoarytenoid joint can leave the vocal cord fixed in a relatively open position.[37,38]

If inactivity of a vocal cord results in an open glottic chink, hoarseness, and intermittent aspiration, the cause of the disorder should be evaluated. Mechanical versus neurologic cause can be assessed by visualizing the larynx and performing a laryngeal electromyogram.

TREATING LARYNGEAL INCOMPETENCE

Vocal Cord Augmentation. A symptomatic open glottic chink due to mechanical impairment, anatomic deficiency, or neuromuscular disability that is not compensated for by the contralateral cord should be corrected. Bruning[39] in 1911 reported using paraffin injection to move a paralyzed vocal cord to a more medial position. Due to the reactivity of paraffin, other materials were tested, including glycerine, cartilage, bone dust, and tantalum. Arnold[40] reported the use of tantalum and glycerine in 1962, and then Teflon and glycerine for medializing the vocal cord. Lewy[41] and others[42,43] have since corroborated Arnold's results using Teflon.

The Teflon is injected into the vocal cord via direct laryngoscopy. The injecting needle should be placed in the midcord area, as far laterally as possible. The procedure is best performed under local anesthesia, enabling constant monitoring of the airway. Small quantities of Teflon can be injected, medially displacing the paralyzed cord. If too much Teflon is injected, the airway may be diminished, producing stridor or obstruction. The Teflon is difficult to remove from the larynx, so judicious use of the material is imperative. Teflon should not be placed in the vocalis muscle, since this will produce an irregularly shaped vocal cord and will change the resonant characteristics of the cord. There are situations in which anatomic deficiencies of the vocal cord can be augmented with small amounts of Teflon.[44-46]

If medialization is necessary, but there is some hope of return of function (such as in brainstem strokes, or recurrent laryngeal nerve trauma) glycerine or gelfoam injection can be used. This will produce medialization for a limited time, until the material is resorbed. Vocal cord injection can also be performed percutaneously in selected patients via the cricothyroid membrane, as recently reported by Ward et al.[47]

In larger defects, where a large quantity of Teflon would be necessary, augmentation with cartilage implants might be more appropriate. Autogenous costochondral, nasal septal, or thyroid cartilage is easily placed submucosally through a midline thyrotomy. This technique was described by Meurman[48] in 1952, and was later refined by others.[49-52]

Laryngeal Suspension. Anatomical alterations of the base of the tongue, pharynx, or supraglottis due to trauma or cancer surgery may prevent the larynx from elevating during the swallowing effort. The inferior-anterior position of the vocal cords relative to the base of the tongue, in spite of the vocal cord adduction, will make aspiration likely. Edgerton et al,[53] Calcaterra,[54] and Goode[55] described a midline, anteriorly based laryngeal suspension to decrease aspiration. In this technique, a suture from the mandible or another anterior structure is sewn to the thyroid lamina, tilting anteriorly and raising the larynx under the base of the tongue to prevent aspiration. Hillel and Goode[56] described a modification of this technique, in which a lateral suspension was used to increase the pharyngeal opening and to change the position of the larynx to decrease aspiration.

Tracheotomy. Copious, chronic aspiration will develop in some patients due to a combination of laryngeal and/or pharyngeal disabilities. These patients warrant a separation of the airway and food passages. The most common method of dealing with this situation is tracheotomy. Using a cuffed tube, the airway can be secured through an independent external portal. Secretions or food substances that pass through the larynx will be kept above the tracheotomy tube cuff. Access for good pulmonary activity is also provided through the tracheotomy tube. Careful suctioning of secretions above the cuff is necessary, since the cuff should be periodically deflated to prevent acute injuries to the tracheal mucosa. At the time of deflation, all the material above the cuff will run into the trachea. Tracheotomy is only a short-term solution, since the pressure from the cuff will eventually produce tracheomalacia and an inability to obtain an effective seal, allowing aspiration to occur. Therefore, for long-term disability, another solution should be sought.[57-60]

Glottic Prosthesis. In an attempt to obtain a separation of the food and air passages without the use of an inflated cuffed tracheotomy tube, a glottic stent can be used. This can be a prefabricated or custom-made prosthesis which can be placed in the larynx through endoscopy, and is usually held in place with percutaneous sutures. The insertion technique is simple, and the procedure is easily reversible; the patient has a tracheotomy for an airway. The problems are related to local inflammation, discomfort, scarring at the glottic level, and the leak of fluid around the prosthesis due to an inefficient seal.[61]

Laryngectomy. For the better part of this century, total laryngectomy was the recommended treatment for patients with chronic aspiration. This provides a permanent cutaneous tracheostoma and a permanent separation of the airway and food passages. It eliminates phonation and the possibility of reversal in patients who might recover some or all of their function. It also involves the morbidity associated with an extensive surgical procedure.[62]

Glottic and Supraglottic Laryngeal Closure. In order to provide adequate separation of the food and air passages, avoid the tracheal damage from a constantly inflated, cuffed tracheotomy tube, and create a potentially reversible situation, several surgical procedures have been designed to close the larynx. These elective procedures can be done after tracheotomy and stabilization of the patient.

A glottic closure procedure was described by Montgomery[63] in 1975. His technique was performed through a median thyrotomy. The mucosa of the vocal cords was stripped bilaterally, and a figure of eight suture was placed to close the glottis and allow fibrous union of the vocal cords. This technique allows adequate separation of the air and food passages with a permanent tracheotomy. The patient cannot phonate. The procedure is potentially reversible, but laryngeal webs are difficult to remove, and the vocal cords themselves may not function well due to scarring. If the vocal cords are functional before surgery, the seal occasionally pulls apart before healing and leaks posteriorly.

For this reason, the Montgomery procedure was modified by Sasaki et al.[64] They added a sternocleidomastoid muscle flap interposed into the subglottic region to provide a better seal. When the larynx opens and leaks, however, the muscle flap also leaks. This added flap also makes reversal more difficult.

Habal and Murray[65] described a two-layered horizontal closure of the supraglottis. Through a pharyngotomy, an incision is made around the perimeter of the epiglottis, aryepiglottic folds, arytenoids, and interarytenoid area. The glossoepiglottic ligaments are severed, the tip of the epiglottis is then folded over the arytenoids, and the mucosa is closed. Mucosa from the piriform sinus is rotated over the epiglottis as a second layer. Patients breathe through a noncuffed tracheotomy tube. The patients cannot phonate, but the procedure is potentially reversible. In some cases, perhaps due to the flexibility of the epiglottic cartilage, the suture line opens posteriorly, allowing recurrent aspiration.

Biller et al[66] described a vertical supraglottic closure that leaves a small opening at the epiglottic tip. With an opening at the level of the tip of the epiglottis, aspiration does not occur, and phonation is possible in some patients. The technique consists of pharyngotomy—an incision along the edge of the epiglottis, aryepiglottic folds, arytenoids, and interarytenoid area. The epiglottic cartilage can be scored in a vertical direction to reduce its mobility. The supraglottis is then closed in two layers as a vertical tube, with an opening of several millimeters at the tip of the epiglottis to allow potential phonation. A permanent tracheotomy is necessary. The procedure, which is potentially reversible, was originally described as an alternative to total laryngectomy in patients undergoing total glossectomy. This author has used this closure successfully in several patients with intractable aspiration, with excellent results. Patients can often eat again, and some can speak.

Laryngotracheal Diversion. In 1975, Lindeman[67] described a procedure to separate the air and food passages, in a reversible fashion, without damage to the laryngeal structures. In this procedure, the subglottic trachea is severed, with the distal portion brought out and sewn to the neck skin. A small opening is then made in the esophagus at the level of the proximal tracheal stump and larynx.

The edges of the esophageal opening are then sewn to the proximal trachea. This system enables all material entering the larynx to exit into the esophagus, yielding complete separation of the airway. Phonation cannot occur following this treatment. If the patient recovers function, the procedure is reversible. Closure is achieved by separating the proximal trachea from the esophagus, and the esophageal opening is closed primarily. The distal trachea is separated from the skin, and an end-to-end anastomosis is performed. This will again allow phonation, respiration, and swallowing in a normal fashion. Many surgeons have used this technique many times with success.[68,69]

Partial Cricoid Resection. Krespi et al[70] recently described a technique for use in patients who have had resections of head and neck cancer, and in whom postoperative aspiration has developed. At the time of surgery, a submucosal dissection of the posterior aspect of the cricoid is performed. A large segment of the center of the posterior cricoid lamina is removed. This, in conjunction with a cricopharyngeal myotomy, leaves a large hypopharyngeal portal for secretions and food, and decreases the anteroposterior diameter of the larynx. Aspiration is therefore abolished or markedly decreased. Phonatory capability remains, but patients require a permanent tracheotomy. This procedure may be useful in other patients with aspiration, although the collapse of the subglottic airway will not be reversible. More studies are necessary to assess the future role of this innovative procedure.

CONCLUSION

Chronic recurrent aspiration can occur in patients suffering from a diverse group of pathologic processes. It is imperative to first elucidate these through a careful workup. There are then a variety of therapeutic modalities available to alter the often fatal course of continued aspiration. Many innovative surgical procedures are available to compensate for severe functional deficits. Some of these procedures can be reversed if the patient recovers from his or her underlying disability. An open-minded, eclectic approach is the best way of preventing the disastrous sequellae of chronic aspiration.

Acknowledgment. This work was supported in part by Teacher-Investigator Development Award #1 KO-7-NS-00392 from the National Institute of Neurological and Communicative Disorders and Stroke, Bethesda, Md.

REFERENCES

1. Bonanno PC: Swallowing dysfunction after tracheostomy. *Ann Surg* 1971; 174:29-33.
2. Nahum AM, Harris JP, Davidson TM: The patient who aspirates—Diagnosis and management. *J Otolaryngol* 1981; 10:10-16.
3. Bartlett JG, Gorbach SL: The triple threat of aspiration pneumonia. *Chest* 1975; 68:560-566.
4. Huxley EJ, Viroslav J, Gray WR, et al: Pharyngeal aspiration in normal adults and patients with depressed consciousness. *Am J Med* 1978; 64:564-568.
5. Ardran GM, Kemp FH: The protection of laryngeal airway during swallowing. *Brit J Radiol* 1952; 25:406-416.
6. Cameron JL, Reynolds J, Zuidema GD: Aspiration in patients with tracheostomies. *Surg Gynecol Obstet* 1973; 136:68-70.
7. Awe WC, Fletcher WS, Jacob SW: The pathophysiology of aspiration pneumonitis. *Surgery* 1966; 60:232-239.
8. Stuchell RN, Blitzer A, Mandel ID: Medical management of non-neoplastic salivary gland disease. *Otolaryngol Clin North Am* 1984; 17:697-703.
9. Parisier SC, Blitzer A, Binder WJ, et al: Tympanic neurectomy and chorda tympanectomy for the control of drooling. *Arch Otolaryngol* 1978; 104:273-277.
10. Parisier SC, Blitzer A, Binder WJ, et al: Evaluation of tympanic neurectomy and chorda tympanectomy surgery. *Otolaryngology* 1978; 86:308-321.
11. Parisier SC, Blitzer A, Binder WJ, et al: Tympanic neurectomy and chorda tympanectomy, in Silverstein H, Norrell H (eds): *Neurological Surgery of the Ear*. Birmingham, Aesculapius Press, 1977, pp 4-18.
12. Didio LJA, Anderson MC: *The Sphincters of the Digestive System*. Baltimore, Williams & Wilkins Co, 1968.
13. Atkinson M, Kramer P, Wyman SM, et al: The dynamics of swallowing. I. Normal pharyngeal mechanisms. *J Clin Invest* 1957; 36:581-588.
14. Negus JE: The second stage of swallowing. *Acta Otolaryng* (suppl) 1949; 78:78-82.
15. Logemann JA, Bytell DE: Swallowing disorders in three types of head and neck surgical patients. *Cancer* 1979; 44:1095-1105.
16. Shedd DP, Scatliff JA, Kirchner JA: A cineradiographic study of postresectional alterations in oropharyngeal physiology. *Surg Gynec Obstet* 1960; 110:69-89.
17. Kirchner JA: The motor activity of the cricopharyngeus muscle. *Laryngoscope* 1958; 68:1119-1159.
18. Blonsky ER, Logeman JA, Boshes B, et al: Comparison of speech and swallowing function in patients with tremor disorders and in normal geriatric patients: A cinefluorographic study. *J Gerontol* 1975; 30:299-303.
19. Stevens KM, Newell RC: Cricopharyngeal myotomy in dysphagia. *Laryngoscope* 1971; 81:1616-1620.
20. Mills CP: Dysphagia in pharyngeal paralysis treated by cricopharyngeal sphincterotomy. *Lancet* 1973; 1:455-457.
21. Lebo CP, U KS, Norris FH Jr: Cricopharyngeal myotomy in amyotrophic lateral sclerosis. *Laryngoscope* 1976; 86:862-868.
22. Calcaterra TC, Kadell BM, Ward PH: Dysphagia secondary to cricopharyngeal muscle dysfunction: Surgical management. *Arch Otolaryng* 1975; 101:726-729.
23. Seaman WB: Cineroentgenographic observations of the cricopharyngeus. *Amer J Roentgen* 1966; 96:922-931.
24. Montgomery WW, Lynch JP: Oculopharyngeal muscular dystrophy treated by inferior constrictor myotomy (trans). *Am Acad Ophthalmol Otolaryngol* 1971; 75:986-993.
25. Wilkins SA Jr: Indications for section of the cricopharyngeus muscle. *Amer J Surg* 1964; 108:533-538.
26. Begley MD: Crico pharyngeal achalasia. *J Coll Radiol* (*Aust*) 1962; 6:138-141.
27. Ross ER, Green R, Auslander MO, et al: Cricopharyngeal myotomy: Management of cervical dysphagia. *Otolaryngol Head Neck Surg* 1982; 90:434-441.
28. Asherson N: Achalasia of the cricopharyngeal sphincter: Record of cases with profile pharyngograms. *J Laryng Otol* 1950; 64:747-758.
29. Schulze-Delrieu K: Esophageal pharmacology, in Choen S, Soloway RD (eds): *Diseases of the Esophagus*. New York, Churchill Livingstone Inc, 1982, pp 35-49.
30. Diamant NE: Normal esophageal physiology, in Cohen S, Soloway RD (eds): *Diseases of the Esophagus*. New York, Churchill Livingstone Inc, 1982, pp 1-33.
31. Duvoisin GE, Ellis FH, Payne WS: The value of palliative prosthesis in malignant lesions of the esophagus. *Surg Clin North Am* 1967; 47:827-831.
32. Skinner DB: Esophageal malignancies: Experience with 110 cases. *Surg Clin North Am* 1976; 56:137-147.
33. Le Brigand H, Roy B: Fistules trachéo-oesophagiennes après trachéotomie. A propos de quatre observations. *Mem Acad Chir* (*Paris*) 1966; 92:405-416.
34. Sasaki CT, Suzuki M, Horiuchi M, et al: The effect of tracheostomy on the laryngeal closure reflex. *Laryngoscope*. 1977; 87:1428-1433.
35. Grillo HC, Moncure AC, McEnany MT: Repair of inflammatory tracheoesophageal fistula. *Ann Thorac Surg* 1976; 22:112-119.
36. Thomas AN: The diagnosis and treatment of tracheoesophageal fistula caused by cuffed tracheal tubes. *J Thorac Cardiovasc Surg* 1973; 65:612-619.
37. Sessions DG, Ogura JH, Ciralsky RH: Late glottic insufficiency. *Laryngoscope* 1975; 85:950-959.
38. Litton WB, Leonard JR: Aspiration after partial laryngectomy: Cineradiographic studies. *Laryngoscope* 1969; 79:887-908.
39. Bruning W: Uber eine neue behandlungsmethode. *Verh Deutche Laryngol* 1911; 18:151.
40. Arnold GE: Vocal rehabilitation of paralytic dysphonia. VIII. Phoniatric methods of vocal compensation. *Arch Otolaryng* 1962; 76:76-83.
41. Lewy RB: Glottic rehabilitation with teflon injection—The return of voice, cough, and laughter. *Acta Otolaryng* (*Stockholm*) 1964; 58:214-220.
42. Rontal E, Rontal M, Morse G, et al: Vocal cord injection in the treatment of acute and chronic aspiration. *Laryngoscope* 1976; 86:625-634.
43. Schramm VL, May M, Lavorato AS: Gelfoam paste injection for vocal cord paralysis: Temporary rehabilitation of glottic incompetence. *Laryngoscope* 1978; 88:1268-1273.
44. Dedo HH, Urrea RD, Lawson L: Intracordial injection of Teflon in the treatment of 135 patients with dysphonia. *Ann Otol Rhinol Laryngol* 1973; 82:661-667.
45. Rubin HJ: Intracordal injection of silicone in selected dysphonias. *Arch Otolaryng* (*Chicago*) 1965; 81:604-607.
46. Rubin HJ: Misadventures with injectable polytef (Teflon). *Arch Otolaryngol* 1975; 101:114-116.
47. Ward PH, Hanson DG, Abemayor E: Transcutaneous Teflon injection of the paralyzed vocal cord: A new technique. *Laryngoscope* 1985; 95:644-649.
48. Meurman Y: Operative mediofixation of vocal cord in complete unilateral paralysis. *Arch Otolaryngol* 1952; 55:544-553.
49. Opheim O: Unilateral paralysis of vocal cord operative treatment. *Acta Otolaryng* 1955; 45:226-230.
50. Waltner JG: Surgical rehabilitation of voice following laryngofissure. *Arch Otolaryngol* 1958; 67:99-101.
51. Levine HL, Tucker HM: Surgical management of the paralyzed larynx, in

Bailey B, Biller HF (eds): *Surgery of the Larynx*. Philadelphia, WB Saunders Co, 1985; pp 117-147.

52. Smith GW: Aphonia due to vocal cord paralysis corrected by medial positioning of the affected vocal cord with a cartilage autograft. *Can J Otolaryngol* 1972; 1:295-298.

53. Edgerton MT, McKee DM: Reconstruction with loss of the hyomandibular complex in excision of large cancers. *Arch Surg* 1959; 78:425-436.

54. Calcaterra T: Laryngeal suspension after supraglottic laryngectomy. *Arch Otolaryngol* 1971; 94:306-309.

55. Goode RL: Laryngeal suspension in head and neck surgery. *Laryngoscope* 1976; 86:349-355.

56. Hillel AD, Goode RL: Lateral laryngeal suspension: A new procedure to minimize swallowing disorders following tongue base resection. *Laryngoscope* 1983; 93:26-31.

57. Bryant LR, Tinkle JK, Dubiler L: Reappraisal of tracheal injury from cuffed tracheostomy tubes. Experiments in dogs. *JAMA* 1971; 215:625-628.

58. Thilenius OG, Vial CB: Chronic tracheostomy in dogs. *J Appl Physiol* 1963; 18:439-440.

59. Fee WE, Ward PH: Permanent tracheostomy: A new surgical technique. *Ann Otol Rhinol Laryngol* 1977; 86:635-638.

60. McDonald RE, Smith C, Mitchell D: Airway problems in children following prolonged endotracheal intubation. *Ann Otol* 1966; 75:975-986.

61. Weisberger EC, Huebsch SA: Endoscopic treatment of aspiration using a laryngeal stent. *Otolaryngol Head Neck Surg* 1982; 90:215-222.

62. Montgomery WW: Total laryngectomy, in Montgomery WW (ed): *Surgery of the Upper Respiratory System*. Philadelphia, Lea & Febiger, 1973, pp 484-496.

63. Montgomery WW: Surgical laryngeal closure to eliminate chronic aspiration. *N Engl J Med* 1975; 292:1390-1391.

64. Sasaki CT, Milmoe G, Yanagisawa EJ, et al: Surgical closure of the larynx for intractable aspiration. *Arch Otolaryngol* 1980; 106:422-423.

65. Habal MB, Murray JE: Surgical treatment of life-endangering chronic aspiration pneumonia. Use of an epiglottic flap to the arytenoids. *Plast Reconst Surg* 1972; 49:305-311.

66. Biller HF, Lawson W, Baek SM: Total glossectomy: A technique of reconstruction eliminating laryngectomy. *Arch Otolaryngol* 1983; 109:69-73.

67. Lindeman RC: Diverting the paralyzed larynx: A reversible procedure for intractable aspiration. *Laryngoscope* 1975; 85:157-180.

68. Yarington CT, Sutton D: Clinical experience with the tracheoesophageal anastomosis for intractable aspiration. *Ann Otol Rhinol Laryngol* 1976; 85:609-612.

69. Baron BC, Dedo HH: Separation of the larynx and trachea for intractable aspiration. *Laryngoscope* 1980; 90:1927-1932.

70. Krespi YP, Pelzer HJ, Sisson G: Management of chronic aspiration by subtotal and submucosal cricoid resection. *Ann Otol Rhinol Laryngol* 1985; 94:580-583.

FROM THE LIBRARY

VOICE AND BREATHING DISABILITIES FOLLOWING THYROID SURGERY

No clinic and no surgeon have been immune from voice and breathing disabilities following thyroid surgery. Injury to the breathing and vocal functions of the larynx following thyroid surgery varies from slight, unrecognized, and spontaneously ameliorated conditions to the tragic and devastating results of bilateral abductor paralysis.

It has long been recognized that a patient, after a thyroid operation, may have a unilateral abductor paralysis and exhibit no symptoms or disability thereafter. The maintenance of abductor function on the other side, with subsequent compensation for phonation, is sufficient to insure normal voice, normal breathing, and preservation of the sensory mechanism of the larynx. It is a matter of surprise to most thyroid surgeons to learn, during the course of their experience, of the number of individuals with goiter who have unilateral vocal cord paralysis and who have never been operated upon for their goiter . . .

The larynx has three functions, all closely interdependent and each necessary for normal voice and respiration. First, and foremost in importance, is the preservation of a normal respiratory airway. This presupposes a normal innervation and a normal muscular apparatus of the larynx. Second is the maintenance of the inspiratory reflex. This function is necessary to protect the larynx from the entrance of foreign bodies, such as food particles and fluids. Third is the preservation of voice. In order for normal voice function to be maintained it is necessary that all of the intrinsic muscles of the larynx function in perfect unison, and with normal innervation.

The disabilities of the voice and breathing in connection with thyroid surgery vary from slight to tragic. We may list them in order of severity somewhat as follows: (1) spasmodic episodes of painful coughing with no expectoration; (2) mucus retention with inability to clear the throat; (3) pain over the region of the molar teeth—the thyromandibular reflex—and the occasional earache; (4) the spasmodic spilling over of fluids into the larynx; (5) the normal voice but with early fatigue and hoarseness; (6) change in the pitch of the voice; (7) the hoarse voice at all times; (8) the inspirational stridor; (9) the stridor and crooning noise when asleep; (10) the whispered voice at all times; (11) dyspnea on exertion; (12) dyspnea on the slightest exertion; and (13) dyspnea at rest.

It is a formidable array, and the interpretation of many of these complications is still doubtful . . . .

CHAS. GORDON HEYD, MD
(*NY State J Med* 1944; 44:1905-1909)

Arthur Donaldson Smith, MD

Physician, explorer, naturalist, and diplomat—Part II*

PASCAL JAMES IMPERATO, MD

In the fall of 1886, Arthur Donaldson Smith began his first year of study at Harvard Medical School. On the other side of the Atlantic in Europe, two Austro-Hungarians, Count Samuel Teleki and Lieutenant Ludwig von Höhnel, were preparing to leave for East Africa in pursuit of Lake Samburu.

DISCOVERERS FROM VIENNA

Count Samuel Teleki von Szek, a count of the Holy Roman Empire, was born at Saromberke in Transylvania (now in Romania) on November 1, 1845, into a noble family that traced its roots to the early 15th century[1,2] (Fig 1). In 1685 one of his ancestors, Mihaly Teleki, who was chancellor of Mihaly Apafi, Prince of Transylvania, had been made a count for his diplomatic efforts on behalf of the latter before King Leopold of Hungary.[3] Beginning in the 17th century the Teleki family produced a number of renowned scientists, scholars, statesmen, soldiers, and politicians.

Samuel Teleki, who came from the Calvinist branch of the family, studied at Gottingen and Berlin universities and later entered the armed forces.[1] He eventually rose to the rank of lieutenant colonel in the Hussars.[4] Through the efforts of one of his brothers-in-law he met Crown Prince Rudolf, with whom he became close friends.[3] He was elected to the Hungarian Parliament from the circumscription of Gernyezegi in 1881.[1,4] Teleki never married and was from all accounts a rather happy-go-lucky sort of individual.[5] He enjoyed good food (he weighed close to 238 pounds at the outset of his African expedition), hunting, and climbing.

Crown Prince Rudolf had been the patron of both the Austrian and Hungarian geographical societies since 1874,[3] and took a keen interest in exploration. Thomson's journey came to his attention and he then decided to induce someone to undertake an expedition in order to discover Lake Samburu and to climb both Mount Kilimanjaro and Mount Kenya, two peaks that had been visited by others but which had not yet been successfully scaled to their summits. Teleki was already preparing a hunting expedition to another area of East Africa and thus it was fairly easy for Rudolf to entice him to lead an expedition of discovery into what is now northern Kenya. Teleki was no Carl Peters and clearly had no interest in serving as a surrogate for Austro-Hungarian colonial ambitions.[3] It is highly probable that Rudolf had some thoughts of acquiring territory for Austria. However, these ambitions were never realized because of the rapid chain of political

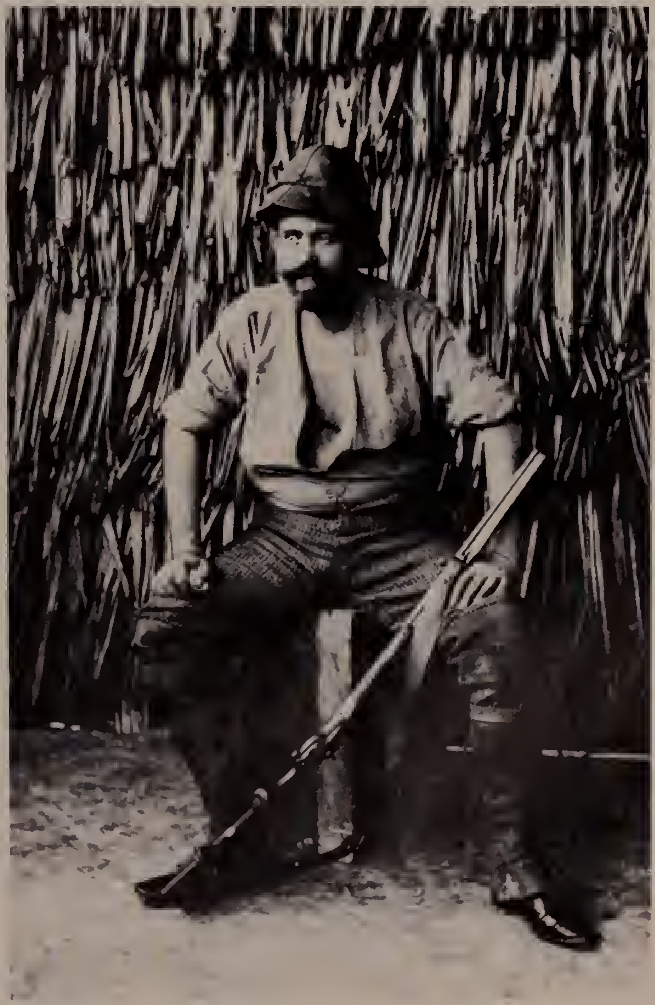


FIGURE 1. Count Samuel Teleki at the start of his expedition. The Africans called him *bwana tumbo* (stomach) (courtesy of Erdelyi Lajos).

Part I of this article was published in the February 1987 issue of the *New York State Journal of Medicine*; parts III and IV will be published in the first half of 1987. Author's acknowledgments will appear at the end of part IV.

Address correspondence to Dr Imperato, Editor, *New York State Journal of Medicine*, 420 Lakeville Rd, Lake Success, NY 11042.

events that sped up partition following Teleki's journey and Rudolf's own suicide on January 30, 1889.

Teleki's companion on this journey was Lieutenant Ludwig von Höhnel (Fig 2), who was a graduate of the Marine Academy at Fiume (1876)[6] and who in 1886 was serving as the navigator aboard the emperor's yacht, *Greif*, which had been put at the disposal of Crown Prince Rudolf.[7(v1,p2)]

The *Greif* was anchored off the island of Lacroma on the Dalmatian Coast. Crown Prince Rudolf and his wife the Archduchess Stephanie were staying in an unused monastery on the island.[8(ch4,p41)] During the cruise they came to know von Höhnel and liked him. The captain of the *Greif*, who von Höhnel says "... was the dayly guest at the princely table ...," [8(ch4,p41)] returned one evening from Lacroma and as was his habit told the watch officer what had happened that day. By luck the watch officer happened to be von Höhnel:

> ... he incidentally mentioned that a Hungarian count had arrived in order to take leave of the Crown Prince before starting on an African expedition. The captain had no idea that with his remark he had touched the most sensitive striving of my inner life, was therefore much surprised when I sympathetically said: "In this I must have a share."[8(ch4,p43)]

Von Höhnel relates that the captain, who liked him, volunteered to see Count Teleki and communicate his wish, which he did. Teleki came on board the *Greif* the next day at 3:00 PM. However, before he did the captain had told von Höhnel that Teleki was decidedly against associating himself with anyone else, much less with someone he did not know.[8(ch4,p44)] "... I had to thank the Crown Prince and the Archduchess who spoke strongly in favor of me." [8(ch4,p44)]

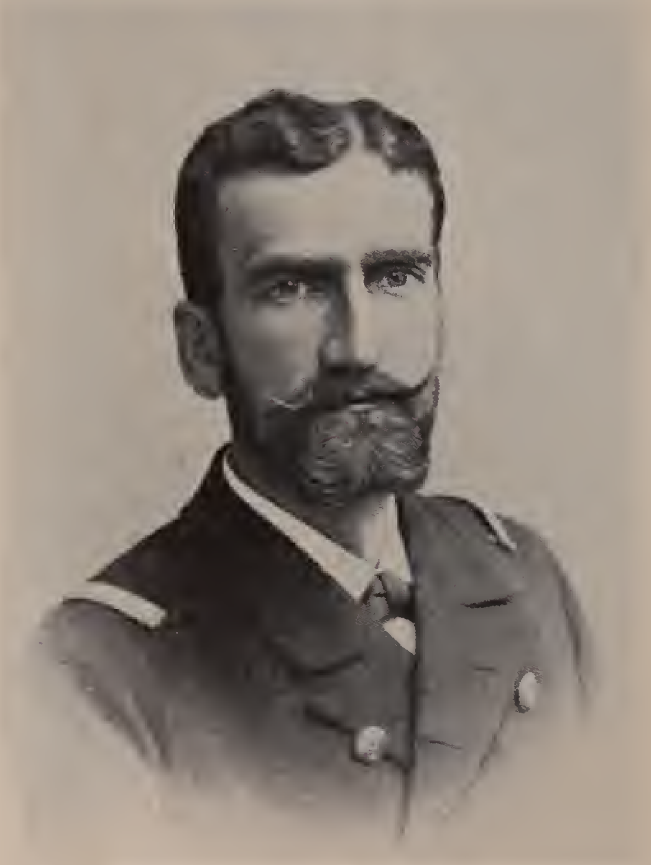


FIGURE 2. Lieutenant Ludwig von Höhnel (Rokeby Papers, courtesy of John Winthrop Aldrich).

Teleki stomped on board on schedule with a copy of Georg Schweinfurth's book, *The Heart of Africa*, under his arm. "In the deck salon he silently spread out the map of the book, pointed to a pencilled line on it and said, 'It is there where I want to go.' " [8(ch4,p44)] The line ran from Zanzibar to Lake Tanganyika and then up north towards Abyssinia. Given the imprecision of Schweinfurth's map it is small wonder that, as von Höhnel later says, "... the expedition from the outset had not been taken seriously ..." [8(ch6,p2)] This, coupled with Teleki's image as a portly bon vivant, gave rise to the anticipation that the expedition would be a colossal failure.

Von Höhnel agreed to everything Teleki said. "To me it was absolutely indifferent where the expedition was to go and how the Count looked if he only allowed me to have a share in it." [8(ch4,p44)] Teleki did agree to take von Höhnel along thanks to the intervention of as he says "powerful influence." [7(v1,p2)] The influence was that of the *Greif's* captain and the Crown Prince and his wife. Thus von Höhnel happened to be in the right place at the right time. Also, he was highly qualified to assume responsibility for the expedition's geographic and scientific activities. Born in Pressburg in what is now Czechoslovakia on August 6, 1857, he was fluent in Hungarian, which was an added asset as far as Teleki was concerned.[3,6] Teleki had been influenced to modify his hunting trip to Lake Tanganyika to explore the regions where the undiscovered Lake Samburu was supposedly located. He financed the journey himself.[9]

Teleki meticulously planned his expedition, and his leadership qualities and iron constitution, coupled with von Höhnel's organizational and scientific abilities, ultimately made it one of the most successful journeys of its kind. He had received advice on recruitment of men from Sir Richard Burton and was familiar enough with Thomson's work that he instructed von Höhnel, who preceded him to East Africa by a month, to enlist the services of James Martin and Jumbe Kimemeta.[7(v1,pp5-7)] They had been responsible to a large degree for the success of Thomson's trip and were clearly highly prized assets to any expedition going into the interior. Von Höhnel was unable to recruit Martin, who was taking a group of sportsmen into the interior, but he was successful in enlisting Jumbe Kimemeta as a guide.[7(v1,p7)] Teleki stopped off in Aden and then went to Somaliland to recruit Somalis on the advice of Sir Richard Burton, who wisely thought it best to have a strong personal escort of men from far away. He recruited a gun bearer there, Karsha, who later served with William Astor Chanler of New York on his expedition and with Arthur Donaldson Smith. Teleki also recruited Qualla Idris, a Somali, who had spent six years with Stanley and who had visited Europe.[7(v1,p11)] The success of expeditions of this kind hinged on the quality of the personnel, and Teleki and von Höhnel spared no effort in recruiting the best and the tried and tested.

Von Höhnel arrived in Zanzibar on October 31, 1886, and Teleki on November 29. There they were materially aided by Alfred Oswald, the Austro-Hungarian consul, General Lloyd William Mathews, the head of the Zanzibari army, Vice-Admiral Baron von Knorr, the head of the German naval squadron in East African waters, and Gustav Denhardt, the younger of the two brothers who had secured Witu for Germany. The fact that so much advice and help came from so many quarters and from British agents as well indicates that the expedition was not perceived by the British at least as having a political agenda.

Jumbe Kimemeta was put in charge of packing the trade goods, crucial for bartering in the interior. In the end, Teleki and von Höhnel sailed for Pangani from Zanzibar with 450 porters, 200 Zanzibaris, nine guides, 70 loads of iron wire, 200 muzzle loaders, 100 loads of glass beads, and 80 loads of cloth, making altogether 470 loads, including 22 that contained a canvas and an iron boat. By the standards of the day this was an enormous expedition. On February 4, 1887, the expedition started inland from Pangani where Teleki had picked up additional men[7(v1,p38)] (Fig 3). Von Höhnel's two-volume account of this expedition has long been a classic of African travel.[7] By April 30, 1887, they had reached Taveta, a caravan center previously visited by a number of European travelers. Two months later they were at Ngongo Bagas, near modern-day Nairobi, which was a stopping point for the coastal trading caravans where food was obtained from the Kikuyu through barter. Teleki set out for Kikuyuland in late August where he encountered frequent hostility and had to repel a number of armed attacks. Von Höhnel came down with dysentery at this time which incapacitated him for several months. Teleki then attempted to climb Mount Kenya, reaching an altitude of 16,200 feet according to his own reckoning,[9] and the Teleki Valley on the high slopes of the mountain commemorates this climb. The cold and damp weather forced Teleki to give up his attempt to reach the top, a task for which he was not equipped. He estimated that 5,000–6,000 feet remained to be climbed above the point that he reached, which given the actual height of the mountain (17,058 feet) may mean that he had not ascended as high as he estimated or else that he overestimated the remaining height. He also said that the mountain had four peaks,[8] but in reality there are only three, M'Batian (the summit), Nelion, and Lenana.

Of interest is the fact that in his book von Höhnel[7(v1,pp376-377)] quotes Teleki as saying, ". . . According to the reading of my aneroid I was now at a height of 15,355 feet above the sea level . . . ." This was at variance with the height of 16,200 feet Teleki told Crown Prince Rudolf he had reached in his letter of November 4, 1887, written within ten days of his returning from the climb.[9]

While von Höhnel described the difficulties they had with the Kikuyu in general terms, Teleki in his letter to Crown Prince Rudolf provided very graphic details not included in the published account of the expedition.[9] He says: ". . . The battle started over again and I took some prisoners; the ground was full of dead bodies. I sent 100 men out who burned 50 villages and captured nineteen men and 1400 sheep." [9] Von Höhnel's account of this is somewhat different. He says:

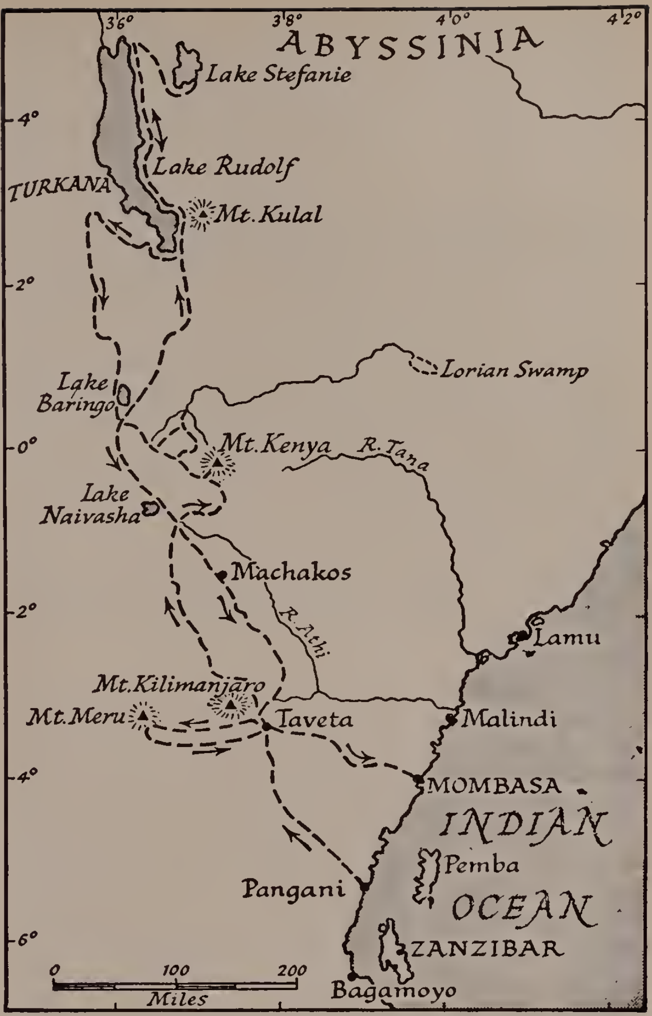


FIGURE 3. The East African interior and Count Teleki's route (from Richards C (ed): *Some Historic Journeys in East Africa*. Oxford, Oxford University Press, 1961).

> Qualla soon reappeared . . . and was told he might draw upon his next assailant. I heard this permission given as I lay on my bed, and then I fell into a refreshing sleep, from which I was aroused about an hour later by the noise of continuous firing. I jumped up, and, in spite of my weakness, ran out of my tent and fired again and again. Count Teleki was standing near doing the same thing.

Von Höhnel then goes on to say that he saw smoke rising from the direction of the villages and that parties of their men began to return with cattle, etc, and women and children as prisoners, ". . . and our men drunk with victory, smeared with their own blood and that of their victims, presented a horrible appearance . . ."[7(v1,pp339-340)]

To put the matter into perspective, however, it must be realized that the expedition had been threatened and provoked by the Kikuyu who were out to pillage it. Without a show of force on Teleki's part the expedition might have been decimated as had caravans been before. But it is of interest that von Höhnel's account implies that the men took matters into their own hands, whereas Teleki quite candidly states that he gave the order to burn the villages. Frederick Jackson, who met Teleki at Taveta, found him very amusing and outspoken, with ideas that were quaint, jarring, and even brutal. According to Jackson, Teleki had a low regard for the Africans.

Teleki also had some harsh words for Thomson in his letter to Crown Prince Rudolf. "Everything Thomson has written about the Masai are pure lies. The Masai are a peaceful people, honest, intelligent such that four to five people can travel in security among them . . . ." [9] In fairness to Thomson, many adverse conditions had befallen the Masai in the intervening years (see part I), which had made them more docile and cooperative, and Teleki may have been unaware of this. His acid comments about Thomson extended to the latter's description of Mount Kenya as well. "I had the agreeable task of justifying two dead travelers. Thomson accused and said of Krapf that he had seen two peaks and thus perhaps he had not really seen the mountain. But he saw it from the west where it appears as two peaks." [9] He continues by saying that "The description given by Thomson is so poor . . . that I was tempted to say that he had never seen Mount Kenya." [9] Teleki then goes on to defend the reputation of Dr Gustav Fischer who he claims was falsely accused by Thomson of buying large quantities of ivory.[9] None of these strong criticisms appear in von Höhnel's book, but they reveal the intensity of the negative feelings between rival explorers, particularly between the German-speaking ones and the British.

On December 7, 1887, the expedition reached Lake Baringo, the northernmost point seen by Thomson during his trip. It was here at the village of Nyemps that they learned that there might be two lakes to the north. On February 10, 1888, leaving men and supplies at their Nyemps base camp, Teleki and von Höhnel set out for the north with Kimemeta, 197 porters, six guides, eight Somali, and 15 Askari. They were now entering a region that had never before been seen by any European. By March 2 they reached Mount Nyiru, where they met a Burkeneji (Samburu) by the name of Lembasso. He confirmed the existence of two lakes to the north, the Basso Narok (black lake) and the Basso Ebor (white lake). The term Burkeneji is Masai and pejoratively means "people of the white goats." Samburu means "butterfly." Lembasso agreed to take them to visit the Basso Narok.[7(v2,pp75–76)] Marching up through the dry, parched landscape, they sighted the lake on March 5, 1888.[7(v2,p95)] Von Höhnel wrote the following of the discovery:

> We hurried as fast as we could to the top of our ridge, the scene gradually developing itself as we advanced until an entirely new world was spread out before our astonished eyes. The void down in the depths became filled as if by magic with picturesque mountains and ragged slopes, with a medley of ravines and valleys, which appeared to be closing up from every side to form a fitting frame for the dark blue gleaming surface of the lake stretching beyond as far as the eye could reach . . . we gazed in speechless delight, spell-bound by the beauty of the scene before us . . . full of enthusiasm and gratefully remembering the gracious interest taken in our plans from the first by his Royal and Imperial Highness, Prince Rudolf of Austria, Count Teleki named the sheet of water, set like a pearl of great price in the wonderful landscape beneath us, Lake Rudolf[7(v2,p95)] [Fig 4].

The following day they actually arrived on the southern shore of the lake and then continued their march up the eastern shore until they reached the northernmost end on April 3. They then set out for the Basso Ebor, the second lake that they had been told about that lay to the northeast, and on April 21 they reached its southern shore. Teleki named it Lake Stefanie in honor of Rudolf's wife, a name that has since been changed to Chew Bahir by the Ethiopian government. They then returned to the northern end of Lake Rudolf just as the rainy season was getting into full swing. Teleki and von Höhnel had discovered two lakes and the next geographic challenge was to determine the source of the larger of the two, Lake Rudolf. Because the rains had transformed the Omo delta at the northern end of the lake into an impenetrable marsh, von Höhnel's explorations of the area were greatly hindered. Based on his limited observations, he concluded that two rivers flowed into the lake, the Nianamm (Omo), which he saw,[7(v2,pp202–204)] and to the west of it the Bass. The meticulous von Höhnel was making an educated guess about the Bass because he did not actually see it. The Omo breaks up into many channels and forms a delta before emptying into the lake, and it is possible that von Höhnel's informants were speaking of one of these channels as being another river, the Bass. In fact he found it almost impossible to obtain information from the local Reshiat people. He says: ". . . it was extremely difficult to get any reliable facts about the surrounding country . . . ." [7(v2,p163)] The Bass, which means "water," and to which von Höhnel's informants were referring, may have been the Kibish River, a secondary source west of the Omo which only reaches the lake during very heavy rainy seasons. Thus Teleki and von Höhnel would return without definitively resolving the question of the course of the Nianamm and the Bass, which, in turn, would entice other explorers to do so.

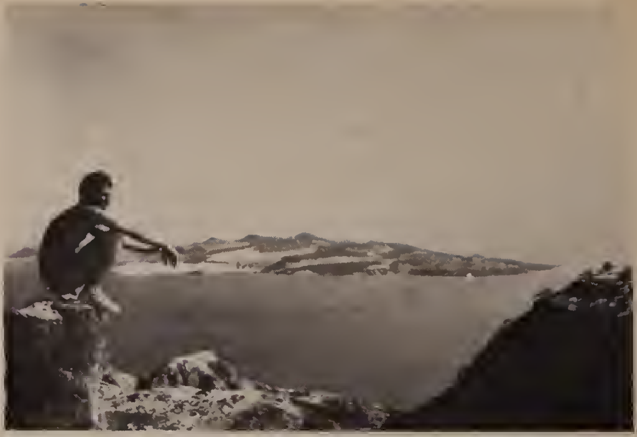


FIGURE 4. A view of the southern end of Lake Rudolf (Lake Turkana) (courtesy of Stephen Pern (in photo), who walked around Lake Rudolf in 1977–1978).

The expedition then started south again, where von Höhnel on a side trip discovered an active volcano at the southern end of the lake which he named Teleki in honor of the count. The gulf at the southern end of the lake near the volcano is still referred to as von Höhnel Gulf. However, the lake has receded since that time and the volcano is now quite a distance from the lake's southern shore and is dormant. From the southern end of the lake they made a detour to the western side into Suk and Turkana country before returning to Baringo, where they arrived on July 29, 1888. It would take them three months to reach the coast, they arriving at the mission station of Rabai on October 24, 1888. They had been in the interior for almost two years, and had discovered that last of the great African lakes. After a two-month stay in Zanzibar, in part necessitated by their both being very ill with malaria,[7(v2,pp318-319)] they traveled to Harar in Ethiopia before returning to Europe in the spring of 1889.[1] By this time Crown Prince Rudolf was dead, having committed suicide on January 30, 1889, at Mayerling. Thus, on his return, Teleki never got to see the man who had been the patron of his successful and acclaimed expedition.

During the time they were in the interior, Teleki corresponded with Crown Prince Rudolf, sending his letters down to the coast with passing caravans, one of which contained none other than the famous Sadi ben Ahedi, who had given Thomson so much grief.[7(v2,p284)] These letters were passed on to Alfred Oswald, the consul of Austria-Hungary in Zanzibar, who regularly reported on the expedition to Count Kalnocky, the Minister of the Imperial House and Foreign Affairs. Teleki's letters deal mainly with hunting experiences, which is not surprising, since von Höhnel's narrative is punctuated by accounts of the count either leaving or returning from the hunt.[10,11] Oswald reported in a letter dated June 6, 1887, that the expedition had visited the Mount Kilimanjaro area.[12] On February 14, 1888, he wrote Count Kalnocky that Teleki had reached Mount Kenya, and on the eighth of May he informed the Ministry of the Imperial House and Foreign Affairs that Teleki had arrived at Baringo in February.[13,14] In this last letter, Oswald dutifully reported on von Höhnel's dysentery as well. Teleki had previously told Crown Prince Rudolf about von Höhnel's dysentery in his letter of November 4, 1887. Poor von Höhnel. His dysentery must have been the talk of the Imperial court in Vienna. Oswald wrote to the Ministry of the Imperial House and Foreign Affairs on October 2, 1888, announcing the good news that the Teleki expedition had reached Taveta on October 22, 1888, 20 days before.[15] Thus the expedition was in sporadic contact with Oswald in Zanzibar throughout most of the time it was in the interior and, through him, with the Austrian government and with Crown Prince Rudolf. In some measure this reflects the relative safety and security of traveling in the interior and the regularity with which trading caravans were moving in and out from the coast.

Teleki remained in robust health throughout most of the trip and the loss of 97 pounds of weight may actually have been to his benefit. When he reappeared on the coast he weighed 141 pounds, in contrast to the 238 he had weighed when he started out.[7(v2,p284)] In fact, in his November 4, 1887, letter to Crown Prince Rudolf, Teleki says, ". . . I will tell you other things when I return home if you still remember the 'fat Teleki' who has had to shrink his trouser strap by three holes. I am in good health."[9]

On reaching Mombasa, Teleki sent a brief report of the expedition's findings to the *Times* in London which was published by the paper on December 25, 1888.[3] The Scottish Geographical Society adapted it for publication and it was subsequently published in Hungarian.[3] Von Höhnel made a verbal presentation of the expedition's findings to the Imperial and Royal Geographical Society of Vienna, much to the annoyance of its Hungarian counterpart.[3,5(p62)] Von Höhnel's full account of the journey was published in German in 1892[16] and in English in 1894.[7] Teleki gave his ethnological collections to the Hungarian National Museum in Budapest.[3] Neither von Höhnel nor Teleki received heroes' welcomes in Vienna when they returned even though they had given Austria-Hungary an honored place in the annals of African exploration. There were two reasons for this. Jealousy was clearly one, as von Höhnel describes:

> On the part of certain other personalities who liked to pretend that they were the only promoters of geographical researches, the expedition had from the outset not been taken seriously, now that they saw that they had been mistaken, they all the more buried it in silence.[8(ch6,p2)]

Even von Höhnel's fellow officers gave him a cool reception. "What did Africa matter to them," he wrote.[8(ch6,p75)] The other had to do with Crown Prince Rudolf's suicide. At the time Teleki and von Höhnel were enroute to Harar in Abyssinia ". . . to kill one more month of the prevailing European winter time . . . Our Qualla Idris suggested a trip to Harar. . . ."[8(ch5,p52)] On their fourth march they were overtaken by a messenger sent by the Austro-Hungarian consul in Aden, who handed them a dispatch that said "that our Crown Prince the Archduke Rudolf was dead." The news "filled us with bitter consternation."[8(ch5,pp54-55)] Von Höhnel explains that

> Count Teleki . . . did not reap the deserved reward for his enterprise. In consequence of the tragic death of the Crown Prince Rudolf one was on the look out for scapegoats whom one might hold responsible, the friends of the Crown Prince were of course the cause of the tragic development. My traveling companion had been one of these and though utterly innocent and though he had been a devoted friend to the Prince, abstentious in every way, he had to suffer for his friendship. His name was unpopular in high circles and this also was a reason not to speak of his travels and achievements.[8(ch6,p2)]

On their return to Vienna via Cairo, Teleki and von Höhnel learned how close they had come to being cheated of their prize. Jules Borelli, a French explorer, had left Egypt on September 16, 1885, and headed for Ethiopia in order to explore the Omo River.[17(p411)] He was delayed from moving into western and southwestern Ethiopia by the Emperor Menelik, who detained him at the capital of Antoto for part of the period from July 1886 to November 1887. Borelli started out in November 1887 for the headwaters of the Omo River and reached a point on the river 300 miles north of Lake Samburu on June 17, 1888, some three months after Teleki and von Höhnel had arrived on the lake's southern shore. Had Menelik not detained him and if he had not fallen ill, Borelli might have reached the lake well before Teleki and von Höhnel. He was forced to return north because of illness, which according to his description was probably malaria.[17(p413)] Borelli was given a good description of the lake by people in Ethiopia who locally called it Lake Schambara.

Borelli arrived in Cairo on November 15, 1888,[17(p414)] where he later met Teleki and von Höhnel. Borelli and the two Austro-Hungarians exchanged information and the former thanked von Höhnel in his book for supplying him with cartographic details which he later included in his own maps.[17(p484)] It is of interest that von Höhnel makes no mention of Borelli in his book. However, von Höhnel has a great deal to say about Borelli in his unpublished

English memoirs. Teleki and von Höhnel first met Borelli's older brother, who was a financier in Cairo and a collector of Egyptian antiquities.[8(ch5,p71)] On seeing the maps Jules Borelli had made Teleki and von Höhnel were "... generally amused, because according to this map, Mr. Borelli had penetrated south right across the country in which Lake Rudolf is lying without seeing it ... I advised him not to show this map to anybody and promised to make it harmonious with our discoveries and to set it right. This was no easy task ..." [8(ch5,p71)] Clearly Borelli was not a very good geographer nor cartographer. He had even connected Lake Baringo to Lake Victoria! Yet he doubted von Höhnel's assertion that two rivers flowed into Lake Rudolf. He had thoroughly explored the upper and middle reaches of the Omo and because of the curvature of the river's course thought that the Omo would have to join up with the alleged Bass river to the west on its way to the south, which subsequent explorers found was not the case.[17(p485)] He diplomatically expressed his conviction that the Bass did not exist by saying: "S'il en est ainsi, l'Omo ne pourrait couler vers l'ouest sans rencontrer ce nouveau fleuve ...." [17(p485)] No doubt the failure to define all the sources of the lake was a sore point with both Teleki and von Höhnel. Borelli deduced that the Omo alone flowed into the lake.[17(p485)] In this he was close to the truth as the Kibish River rarely did so and then only under unusual circumstances. He also correctly noted that "... sous le nom Niannam, et non d'Omo; mais cette difference de noms est sans interet, si l'on songe que les denominations de l'Omo varient suivant le pays qu'il traverse." [17(p484)] In other words the river had different local names and, as Borelli concluded, "... l'Omo est le Niannam." Borelli was the first to come very close to charting the correct course for the entire Omo River and the main source of the lake.[17(p443)] But as he had not traveled down its entire course, his views on the lower stretches were viewed by some as unconvincing. Borelli was correct in stating that the Omo was the real source of the lake and that the Nianamm and Omo were one and the same. However, he was incorrect in assuming that the westward curve in the Omo's course destined it to flow into a second river to the west if one existed. By saying this he was really denying the existence of the second river. Von Höhnel it seems made an intelligent guess that there was a second river to the west. It will never be known if von Höhnel's informants were referring to the Kibish River or simply to a channel of the Omo when they spoke of a running river; others said that the water was several miles wide and had no current at all; all of them simply called it Bass, which in general means "water." [18(p26)] Von Höhnel goes on to say that both groups of informants were correct, for "... later explorers ascertained that not only was there a river, the Kibish, but a long and narrow gulf of the lake as well." [18(p26)] When Teleki and von Höhnel returned to Vienna and Borelli to Paris the issue of whether the Omo and Nianamm were the same was an open one. Over the ensuing years some thought that the Omo eventually flowed into the Juba River to the east and that the Nianamm, the source of Lake Rudolf, flowed parallel to it in the west. Those who did so were in error, and Arthur Donaldson Smith was among them.

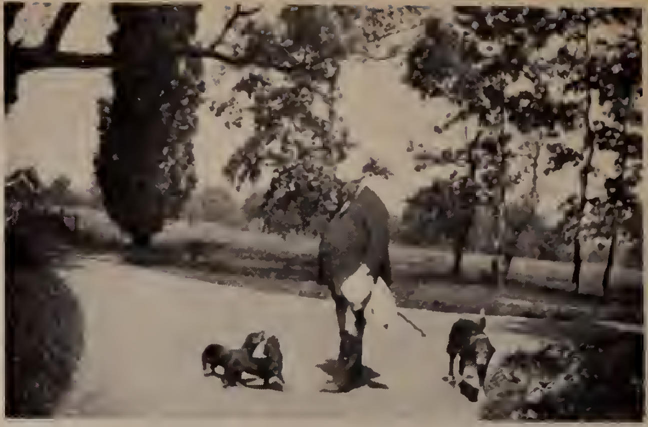


FIGURE 5. Count Samuel Teleki at Saromberke at the turn of the century (courtesy of Erdelyi Lajos).

Teleki never returned to Africa, but subsequently traveled to Indonesia in 1893 and to India.[1,3,5] However, he left no accounts of these later travels.[3] He was elected a member of the Hungarian Academy of Science in 1904. Von Höhnel visited Teleki at his country estate at Saromberke, a village that "had a purely Hungarian population" [8(ch6,p15)] (Fig 5). Von Höhnel wrote:

> Saromberke lies in Transylvania in the upper Maros Valley. Simple as the host himself, were the building which he inhabited as well as the people whom I first met there ... Simple and modest though Count Teleki had shown himself during the expedition he now proved he was a gourmet of no mean order. He imported his coffee directly from Mokka, his cigars from Havana and the same thing applied to everything in his household.[8(ch6,p15)]

Teleki had inherited a remarkable library from his great-grandfather, but his pride was his stud farm. "Saromberke bred half-bloods of special quality, tough horses with gazelle-like eyes and arab heads." [8(ch5,p15)] Von Höhnel was very fond of Teleki and a life-long friendship existed between them. Von Höhnel, looking back on his first meeting with Teleki on the *Greif*, said, "I then could not see what ... consequences would result from the first short interview, that many a year of this friendship would bind me together with this man." [8(ch4,p45)] Teleki died at his home at 4 Reviczky Street in the eighth arrondissement of Budapest after a long illness on March 10, 1916, at the age of 71.[1,4] Surviving him were his sister Elizabeth, Baroness Banffy Zoltan, and her children, and the children of his sister Agnes, Countess Joseph Zeyk, who had predeceased him. A Reformed service was held in his home on March 12, 1916, at 2 PM, and later his remains were transported to Saromberke where after a service in the local Reformed Church he was buried in the family vault[4] (Fig 6).

Teleki's former estate is now the Göngényszentimrei Forestry School and Saromberke is known as Dumbravioara.[19] The impressive Teleki library founded by Teleki's great-grandfather in 1802 is now housed in the nearby small city of Tirgu Mures.[19] In the early 19th century it had 40,000 volumes for Saromberke's 6,000 inhabitants.[19] Most of Teleki's African artifacts and hunting trophies disappeared during World War II.[5] A few trophies that were saved by the Mueramures hunters' committee are on exhibit at the Forestry School museum.[5] The Teleki mausoleum is preserved at Dumbravioara and was recently restored.[5,19]

In 1977, Erdelyi Lajos, a photographer from Tirgu Mures, published an account of Samuel Teleki's African expedition in Hungarian based on primary source documents.[5] It is illustrated with photographs taken by Teleki during the trip.[5] It is of note that many of these photographs were touched up and modified into illustrations for von Höhnel's published account of the expedition.[7] Erdelyi, however, presented them in their unedited form for the first time in his book.[5]

A REMARKABLE NEW YORKER

As Teleki and von Höhnel were leaving the East African coast for home, a young American from Dutchess County, New York, arrived enroute to Mount Kilimanjaro. William Astor Chanler was only 22 years old at the time. However, the previous year he had inherited a fortune on attaining his maturity and, like most of his seven siblings who lived to adulthood, struck out in life on a highly individualistic and unusual path, eventually leading a major expedition into Africa.[20] A great-great-grandson of John Jacob Astor, Chanler and his brothers and sisters were orphaned at a young age by the premature death of both their parents.[21(pp23,28)] They were raised by family guardians much older than themselves at Rokeby, the family's estate in Dutchess County that overlooks the Hudson River. The day-to-day rearing was left to servants at Rokeby or else to the authorities at boarding schools such as St John's in Ossining, where young Chanler was a student when his father, Congressman John Winthrop Chanler, died in 1877.[21(pp27)] Not surprisingly, a number of Chanlers became fairly self-reliant at a young age and William Astor Chanler was one of them. He was bright, daring, assertive, and opinionated, inclined to be impulsive and spurn advice. In a sense it was fitting that his first great endeavor in life was in the shadows of Mount Kilimanjaro, Africa's greatest volcano, for he himself was like a piece of molten rock shot out onto the African landscape without warning.

He arrived in Zanzibar in March 1889 with his servant, George E. Galvin, who at 18 was even younger than himself, and who also came from Dutchess County. Margaret Aldrich (Maddie) DeMott, Chanler's niece, records that he had obtained a commission in the German East African Ivory Patrol in Germany after dropping out of his sophomore year at Harvard, where studies bored him.[22]

FIGURE 6. The Teleki Family Mausoleum at Saromberke (Dumbravioara, Romania) (courtesy of Erdelyi Lajos).

Within a short time, Chanler was on his way to Mount Kilimanjaro, then a magnet for European sportsmen.[23(pp20,87)] It was also an area where British and German interests faced one another off in the tension-filled days before the Heligoland Treaty was signed on July 1, 1890. The Anglo-German boundary eventually passed just north of Kilimanjaro—not because Queen Victoria gave the mountain to Kaiser Wilhelm as a present as is often erroneously reported, but because the Germans had clearly established a presence on it. In 1889, German-British rivalry in East Africa was fierce and it was into this setting that young Chanler came marching with 180 porters and a large fluttering American flag!

The greatest tributes to him and his accomplishments during the six months he explored the mountain were made by the German scientist and explorer Dr Hans Meyer, who was the first to reach the top of Mount Kilimanjaro. He wrote,

> . . . The Kilimanjaro states have been visited by a number of missionaries and sportsmen of whom perhaps the most noteworthy is the young American, Mr. Chanler, who, proceeding to the region merely for the purpose of sport, has nevertheless distinguished himself by a thorough exploration of the lower slopes of the mountain.[23(p20)]

Meyer said later,

> . . . It proved to be Mr. Chanler, now on his way home from Moji to Taveta. He stayed with us overnight, and greatly delighted me with his spirited account of his achievements and plans, and with his keen insight into African affairs. Many of our colonial wise acres might envy the energy and tact which has enabled this youth of 23 with a still more youthful companion, to lead a caravan of 180 men into regions as yet totally unexplored.[23(p120)]

Meyer, who had made two previous trips to Kilimanjaro in 1887 and 1888 and who had met Teleki and von Höhnel on their return to the coast,[23(pvii)] pays even greater tribute to Chanler when he cites him as an authority on the flora of the mountain. "According to Mr. Chanler, the belt of tree-steppe at the bottom of the mountain stretches upward to a height of from 2460 to 2950 feet in the south, and to 4900 feet in the north, the trees gradually increasing in luxuriance as they ascend." [23(p322)] Meyer met Chanler again at Moji on the southern flank of the mountain at the station of the German East Africa Company, which was but 500 feet above an English mission.[23(p94)] "The American sportsman, Mr. Chanler, had arrived shortly before us with his followers, so that there were now about two hundred men encamped at this spot." [23(p94)]

Meyer's description of the flags flying on Kilimanjaro is interesting for he says, "My goal for the present, however, lay farther up the mountain where on the heights waved a solitary Union Jack and beyond it the flags of Germany and America." [23(pp93-94)] Meyer was received with warm hospitality by Mr Morris of the English mission station.[23(p94)] Despite the rivalry between the powers, individuals from either side managed to remain civil and hospitable.

It is of interest that Chanler was camped at the station of the German East Africa Company, where ". . . the decoration of the interior has been carried out in the national colors, black, white and red, and the earnest face of the Emperor Wilhelm II looks down from the place of honour on the wall." [23(p94)] This might have occurred because of Chanler's relationship with the German East Africa Company.[22] Yet Meyer's message is clear. The Union Jack stands alone against the stars and stripes and the German tri-color, which were flying side by side. This would not have endeared Chanler to the Imperial British East Africa Company which then held a charter from Britain, and may in part have contributed to the difficulties he encountered with British officials at the end of his expedition to the north of what is now Kenya in 1892–1894.[20]

Yet Chanler maintained cordial relations with the British, writing to Buchanan of the Imperial British East Africa Company about a well-armed Swahili caravan from Pangani that had just left Taveta for Kavirondo.[24(p160)] Such a report was significant because caravans of this kind often engaged in the outlawed slave trade.

Chanler maintained a compound in Taveta, southeast of Kilimanjaro. Taveta was at the time a trading caravan staging area and Meyer says of Chanler's compound: "Like the Englishman Johnston in 1886, a young American has now located himself here, in a charming little camp, comprising a dwelling-house, several huts, and a garden . . . ." [23(p87)] Frontera records that in 1889 Reverend Alexander Steggall of the Church Missionary Society, on hearing of Chanler's imminent departure from Taveta, urged the society to consider taking over the building and two acres of land. The society finally agreed and in March 1890, Steggall and a caravan of 200 went to Taveta to discuss the matter with the elders.[25(p22)]

Chanler spent six months exploring the Kilimanjaro region and seems to have determined either during that time or shortly thereafter to lead an expedition into the vast unexplored regions between Lake Rudolf and the Juba River. As he was formulating these plans the famous German colonizer Carl Peters was enroute along the southern fringes of this area, handing out German tri-colors and signing treaties all the way to Uganda. Britain's imperial advocates were in a state of alarm until the Heligoland Treaty of July 1, 1890, settled Anglo-German matters in East Africa. However, a claim without effective occupation was meaningless, as the officials of the Imperial British East Africa Company knew only too well. Although the purpose of Chanler's expedition was purely scientific, his traversing of unexplored territory claimed by Britain no doubt made officials of the Imperial East Africa Company uncomfortable on two counts. An alien flag, moving across the landscape of their unoccupied claim, was highly offensive in the charged, jingoistic environment of the time. Also, Chanler's proposed route would take him into the broad undemarcated regions to the north near Abyssinia where British claims were at best tenuous. There was always the possibility, however remote, that an American claim could be staked out. While the company could not forbid Chanler to enter country over which it had absolutely no effective control, it had no reason to wish him well.

As far as is known Chanler made only one trip to Mount Kilimanjaro. However, his son, William Astor Chanler, Jr, relates that he made two trips there, one in the company of German ivory hunters.[26] Documentation for this, however, is currently lacking.

CHANLER RETURNS TO AFRICA

Chanler knew East Africa well and set his sights on exploring the vast patch of unknown territory that lay north of the Tana River between Lake Rudolf and the Juba River. In this he was no mere dilettante out for sport and adventure. This is supported by the fact that he made meticulous preparations for this expedition in London by purchasing an impressive array of supplies and equipment and contacting respected scientists for guidance. Richard Harding Davis, the turn-of-the-century writer who as Thomas says was the "beau ideal of American youth . . . the glamourous foreign correspondent and man about town," [21(p155)] was mightily impressed with Chanler's preparations. He wrote the following of Chanler's preparations: "Part of his day was spent closeted with the officers of the East Africa Company, another part in Whitechapel buying Tommies' discarded redcoats as presents for African kings; and later he was testing smokeless powder and repeating rifles, or choosing canned meats and bottled medicine . . . ." [27] In March of 1892, Chanler wrote to von Höhnel out of the blue[26] asking him to accompany him on the expedition.[28(p1)] As De Mott observes, this also was an indication that he intended his expedition to be of a serious scientific nature.[22] The fact that he met with officials of the Imperial British East Africa Company clearly shows that he was sensitive to the political realities as well.

Von Höhnel had never heard of Chanler and had already turned down a number of offers to return to East Africa, including one from the Imperial British East Africa Company.[28(p1)] However, a visit to Vienna by Chanler in April made him change his mind. Chanler got the Austrian Admiralty to give von Höhnel a furlough.[28(p8)] Then Chanler left for London, where as Thomas relates he amused himself by riding in breakneck steeplechases against the best gentlemen jockeys.[21(p152)] Two months later, however, Chanler was ready to start off and invited von Höhnel to London for a final understanding.[28(p13)] They left Europe separately, met in Port Said, and then sailed to Aden, where von Höhnel remained behind to engage the Somali while Chanler went on to Zanzibar.[20(p7)] Von Höhnel also had to go over to Massowa in Eritrea to engage some Sudanese.[28(p14)] Chanler saw his main task at Zanzibar as the engagement of porters. However, as he says: "I could not have chosen a more inopportune time for the enlistment of porters at Zanzibar. The British East Africa Company, bent upon the retention of Uganda, had practically exhausted the supply of porters, and a missionary caravan was on the point of starting to the interior." [20(p7)]

Chanler, being the enterprising fellow he was, had no intentions of sitting in Zanzibar waiting for tried and tested porters to return from the interior at some vague period in the future, nor of following von Höhnel's plan of recruiting porters in nearby German East Africa. As Thomas observes, he was "tough, primitive, and single minded. His capacity for action was enormous and he despised dawdling and irresolution . . . but his inherent pugnacity was disguised by effortless manners and aristocratic ease." [21(p152)] True to form Chanler did what came naturally to him. He went out and hired men from the plantations, sending out word that an expedition was about to leave and that it had the approval of the government.[20(pp7-8)] When von Höhnel arrived in Zanzibar a few weeks later he was shocked by the news that Chanler had hired 135 porters "by hook and crook." [28(p15)] They were largely young and inexperienced men unaccustomed to the rigors of travel in the interior. The civil status of most of them was virtually that of slaves. Von Höhnel had arranged with the governor of German East Africa to hire Wanyamwezi porters who then and in later years were renowned as the best porters in East Africa. As von Höhnel says: "... we could have chosen the pick out of thousands of stalwart men. The Wanyamwezi, moreover, are cheerful and trustworthy men who never think of deserting." [28(p15)] Von Höhnel had made meticulous preparations with the Germans for hiring these porters and Chanler was well aware of these plans.[28(p16)] Chanler was impulsive and impatient in action as Thomas describes, even though he was cautious and far-seeing in his planning.[21(p161)] Von Höhnel was distressed and feared the worst consequences of hiring inexperienced youths. He later said of Chanler, "He at that time was not aware that he had made a grave mistake, but he admitted it some months later." [28(p16)]

Porters were the backbone of any expedition and illness and desertion among them could rapidly cause the collapse of even the best planned and supplied enterprises. Chanler had made meticulous preparations for this trip. George Galvin, his young servant, traveled separately to Lamu on the coast from Europe with the trading goods and other supplies. Von Höhnel had brought scientific and military equipment from Vienna and Somali and Sudanese escorts and now Chanler had impulsively hired the stuff of failures.

Sir Gerald Portal, who was the British consul-general in Zanzibar and a friend of Chanler's, gave every support possible, instructing his subaltern General Lloyd Mathews, the prime minister of Zanzibar, to assist Chanler in every way.[20(p7)] It was Chanler's intent to travel up the Tana River region from Lamu on the coast to Mount Kenya, climb the mountain, then go north to Lake Rudolf, and from there to move east across 600 miles of unexplored territory to the Juba River.[27] He then planned to follow the river to the ocean and then move southwest along the seashore to Lamu, covering 3,000 miles in the process.[27] This scope of the expedition was published by Davis and reflects Chanler's objectives before his departure. Chanler's own objectives as related in his book after the expedition are more vague.[20(p4)]

CHANLER'S EXPEDITION LEAVES THE COAST

The expedition left Lamu for the interior on September 18, 1892 (Fig 7). Chanler expected to return to Lamu in two years. The expedition consisted of three Europeans—Chanler, von Höhnel, and Galvin—and 60 men including the Zanzibari porters, seven Somali camel drivers, and 12 Sudanese guards who were armed with Mannlicher repeating rifles.[20(p21)] The Sudanese were provided with uniforms topped by a bright red fez. Also in the party were 15 camels, ten oxen, goats, sheep, donkeys, horses, and three dogs. Marching along the Tana River, they reached Hameye, some 250 miles inland, two months later. Hameye was an abandoned Imperial British East Africa Company post.[20(p69)] Enroute to it the expedition had encountered the usual perils of travel then—illness, hostile groups, desert wastes, and, as von Höhnel feared, more than the usual number of desertions. Chanler tried to mend the situation by sending back the most unreliable porters to the coast and calling for new ones. Since it would take several weeks for new porters to arrive, Chanler and von Höhnel set off to explore the area to the north, leaving Galvin at Hameye. They left on December 15, 1892, with 80 men.[20(p81)] Chanler became ill with fever, probably malaria, but would not consider stopping. Eventually they came to the Nyambeni Mountains that today fringe Meru National Park, where they encountered the Embu, an eastern extension of the Kikuyu. It was here that they picked up a guide, Motio, who was to take them to the Guaso Nyiro River and "Lake Lorian," which they eventually discovered was not a lake but a swamp into which the river emptied.[20(p140)]

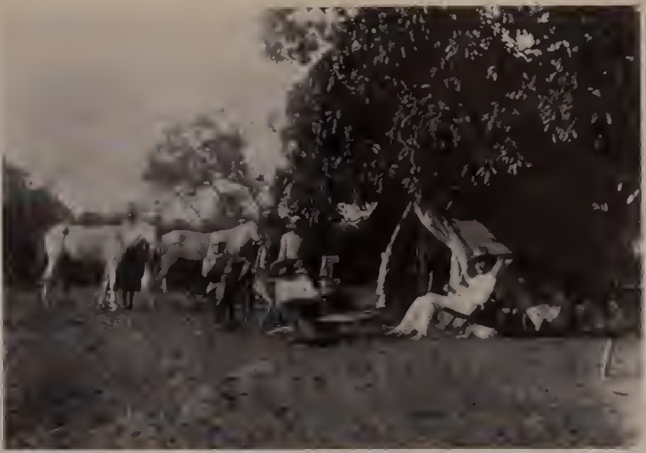


FIGURE 7. One of Chanler's camps on the East African coast, 1892. Seated from left to right: Chanler; Lieutenant Ludwig von Höhnel; and George E. Galvin. Standing behind von Höhnel in striped shirt is Sururu, Chanler's tent servant, who remained faithful to him throughout the expedition and who had traveled with Henry Morton Stanley (Rokeby Papers, courtesy of John Winthrop Aldrich).

Chanler had to be carried in a hammock for the first four days because he was delirious and semiconscious. It was the rainy season and malaria transmission was at its peak, unknown then as the parasite and the vector had yet to be linked.

On December 26, 1892, they came upon a waterfall on the Guaso Nyiro some 60 feet high which was named Chanler Falls, a name that is still retained today.[20(p119)] Marching ever eastward through the dry scrub they eventually reached the Merti Plateau by December 30. On January 12, 1893, they realized that Lake Lorian was really a swamp, swarming with mosquitoes. The members of the caravan were both dejected and angry, for instead of a fresh water lake where they could rest and recuperate, there was nothing but "an abode of pestilence and death," as Chanler recorded.[20(p139)] It took them a month to hobble back to Hameye. Short on rations and sick with malaria, they were attacked by the Wamsara (Meru). Three porters were killed and twelve wounded in the skirmishes that followed.[20(pp173-177)] They finally reached Embu country and on February 10, 1893, sighted Hameye where the stars and stripes were proudly waving over the camp.[20(p202)] The news that Galvin had was not good. The oxen had died, as well as some of the cattle, camels, and donkeys.[20(p202)]

Despite hardships and illness, Chanler and von Höhnel had traversed 600 miles of unexplored terrain and had mapped the middle and lower reaches of the Guaso Nyiro River, discovering Chanler Falls in the process. They had also laid to rest the myth of "Lake Lorain." Chanler was in sorry shape and had to rest for two weeks. Tenacious and determined, he set out again for the north on March 8, 1893, heading for the Mathews Range, which had never been fully explored. Enroute to the Nyambeni Mountains some 20 men deserted. Chanler sent his headman Hamidi down to the coast for replacements, which proved to be a mistake because he was not trustworthy, as von Höhnel had suspected all along.[20(p399)] Chanler, however, pushed ahead, exploring the southern Mathews Range and Lolokwi Mountain.[20(p277)] He then met with the Rendille people, a group of pastoral nomads who had had no previous contact with Europeans, a fact that Chanler communicated to the director of the Imperial British East Africa Company by letter on September 22, 1893.[22] Claim of first contact had disquieting political implications: for all the company directors knew Chanler could have signed a treaty of protection with them on behalf of the United States and handed them some flags, as the British and Germans had been doing all along elsewhere in Africa.

On August 24, 1893, von Höhnel was seriously gored by a rhinoceros and had to be carried in a litter back to the base camp at Daitcho.[20(p380)] Daitcho was reached on September 18 and from there von Höhnel was carried to Kibwezi, a mission station 250 miles to the south.[20(pp400-403)] Chanler sent George Galvin with 25 men as well as 18 unfit porters on this trip, which took six weeks. Von Höhnel later returned to the coast and home to Vienna. Meanwhile Chanler planned his trip to Lake Rudolf and beyond. It was never to take place.

MASS DESERTION

On October 7, 1893, Hamidi arrived with 80 unfit porters, who Chanler tried to train for the next two and a half months until Galvin returned.[20(pp417-423)] Galvin arrived on December 15 but on December 17, Hamidi and most of the porters disappeared. The next day the Sudanese left but then returned.[20(pp448-462)] This mass desertion effectively caused the collapse of Chanler's expedition. But why had it occurred? Chanler was dumbfounded at first because it came as a total surprise to him.[20(p450)] However, he set out to uncover the reasons for it. He wrote Hamidi, the headman, whom he suspected was at the bottom of it,[20(p453)] and sent the letter off. Hamidi appeared two days later[20(p155)] and said that the men felt that their enlistment time had expired and that they had no desire to go farther into unknown country filled with savage people like the Rendille. Chanler says: "I was firmly convinced that he was at the bottom of it." [20(p456)] Hamidi confessed that nothing Chanler had done should have caused the mass desertion and that he himself had no complaints to make. Yet on his revisit Hamidi tried to get the Sudanese to desert.[20(p462)] Chanler says:

> He told them that they should come to him at the river and follow him to the coast as General Mathews of Zanzibar had told him that if he succeeded in inducing the men to desert, he would see that they all received their pay upon reaching the coast.[20(p462)]

Chanler goes on:

> Taking all things into consideration, it looked as if Hamidi had been acting under orders received during his visit to the coast; but what possible reason the authorities at Zanzibar could have for breaking up my expedition could not appear clear to my mind. The ways of diplomacy are devious.[20(p462)]

The British authorities in Zanzibar, then the custodians of British interests on the mainland, had every reason at this time to see that Chanler was stopped. With seemingly endless personal financial means Chanler was capable of roaming all over the unexplored map with the American flag, and in so doing posed a potential political problem. An effective way of stopping him was to cause a mass desertion of his men. Speaking of Hamidi's role in this, Chanler cogently concluded "that, instead of twenty well-armed men and some donkeys, I had been furnished with a disorderly rabble of eighty unarmed and insubordinate men—that he must have received something stronger than a hint that it was the pleasure of the people in power at the coast." [20(p466)] Von Höhnel did not think much of Hamidi, observing, "In Count Teleki's Rudolf Lake Expedition he was a simple porter who after one year had been made an askari." [28(p19)] With von Höhnel the famous and well-respected explorer out of the picture it would have been easier for spoilers to cause the expedition's collapse.

Chanler and Galvin had no choice but to return to the coast, which they reached on February 10, 1894, at Mombasa (Fig 8). There Chanler found a letter from General

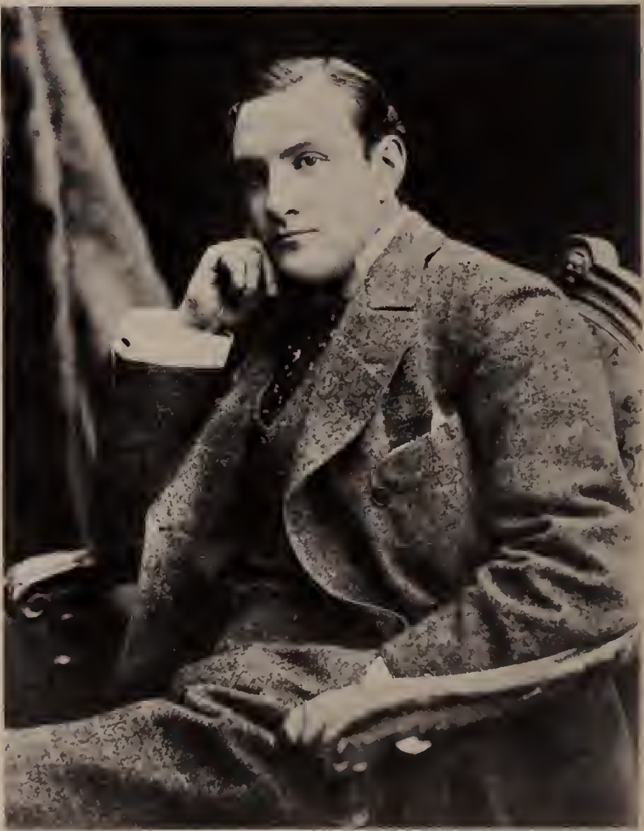


FIGURE 8. William Astor Chanler after his second African expedition (Rokeby Papers, courtesy of John Winthrop Aldrich).

Lloyd Mathews advising him not to come to Zanzibar but rather to leave on the sly at once and have his agents pay off his men.[28(p30)] Chanler also found that instead of arresting the deserters as was the established procedure, the British authorities in Mombasa had sent them on to Zanzibar at Chanler's expense.[20(p498)] There they had also been lodged at his expense.[20(p499)] Thus the deserters had been treated by the British authorities contrary to precedent, which lends some credence to Chanler's suspicion that Lloyd Mathews was behind the plot.

Matters were complicated by the fact that there was a new American acting consul general in Zanzibar with no prior experience in diplomacy or consular matters.[20(p499)] Sir Gerald Portal, the British agent and consul general, had died in London of typhoid fever and his replacement James Rennel Rodd was away because of illness. Mathews, who reported to the British agent in his capacity as the sultan's prime minister, was thus the supreme British authority.

Chanler met with Mathews and found that he had already concluded that the porters were justified in their desertion without having heard Chanler's side of the story.[20(p500)] Mathews had taken the porters to the acting US consul who took down their statements, which were conflicting. In general they accused Chanler of cruelty—of shooting down several and killing up to 30 through excessive flogging.[20(p502)] They also said that they had been engaged for 18 months which was not the case. On questioning the porters Allen, the US consul general, was unable to find a single instance of Chanler's having killed anyone by flogging or shooting. One porter had been accidentally shot by a Sudanese guard prior to March 1893. This seemed to warrant the desertion of the expedition in the minds of the British authorities.[20(p504)] Yet Hamidi had returned to the coast after this and had met with Mathews, who made no mention of the incident then to the US consul general.[20(p504)]

Chanler offered to have the complaints against him heard in the consular court of the US but Mathews objected, saying that Allen would be biased in Chanler's favor.[20(p505)] He insisted that Chanler appear before a British court, where of course the outcome would have been against him. Chanler got nowhere trying to vindicate his name. He remained in Zanzibar for several weeks, two of which were spent in a French hospital because of malaria.[20(p506)] Some 15 of the deserters attacked his house with clubs and were driven off by George Galvin.

> Such an attack upon a European, as far as I could learn, had not occurred in Zanzibar for more than twenty years; and it struck me as strangely coincident with the hostile attitude of the authorities at Zanzibar. A complaint by the US consul resulted in the confinement of a few of the men for a few days.[20(p507)]

Mathews wrote a letter to the US consul offering the opinion that the desertions had not been caused by cruelty on Chanler's part, but by plotting on the part of the Somali who were tired of the journey and wanted to return home.[20(p509)] This is at variance with his previous view of the cause of the desertion.

Chanler waited in vain for Mathews to bring suit in the US consular court[20(p507)] and then decided to submit the matter to arbitration. He proposed to appoint one arbitrator, have the government of Zanzibar appoint another; and the two to choose a third. Chanler chose Seth A. Pratt, the former US consul at Zanzibar, and the government of Zanzibar chose a Mr Wilson, then its legal adviser.[20(p508)] Pratt proposed eight candidates, German, Italian, and French, all of whom were fluent in Swahili, a necessity as much native testimony had to be heard in that language. Wilson refused all of these and instead proposed three men, one of whom had just been decorated by the government of Zanzibar for services rendered and two others who Pratt knew were hostile to Chanler's interests.[20(p508)] Thus, based on Chanler's account, General Mathews was intent on stacking the court against him. Pratt finally gave up, finding Wilson unreasonable and discourteous. Chanler steadfastly refused to pay the £1,000 in wages due the deserters and finally Mathews agreed to half that amount.[20(p509)]

THE REASONS FOR THE COLLAPSE OF CHANLER'S EXPEDITION

What is one to make of all the evidence? Clearly the porters were not the best. Desertions were common in expeditions such as Chanler's. Teleki and Meyer experienced them—they were handled with the flogging and chaining of the offenders.[7(v1,p55),23(pp59,61)] The level of desertion in Chanler's was higher because the men were young and inexperienced, a fact that von Höhnel underscores in his reminiscences.[28(p15)] The mass defection came suddenly, almost three months after von Höhnel's departure. Chanler seemed convinced that the British authorities in Zanzibar instigated it through Hamidi the headman. If they did then there must have been a motive. The late William Chamberlain Chanler, the explorer's nephew, once said, "The British didn't like the idea of an American marching around their unexplored territory carrying the American flag and decided to put a stop to it." [29] There is a great deal of credibility in this observation and much evidence supporting it. Thus it may be that Chanler was simultaneously victimized and vilified by British colonial interests that considered his travels a political thorn threatening to their then-precarious hold on the interior. Their aim would have been not only to stop his then-current expedition, but also to insure that he did not return to East Africa to launch another.

Chanler seemed convinced that had Sir Gerald Portal, who was the British agent and consul general in Zanzibar, been there in February 1894, matters would have gone differently. He also thought that had Portal's temporary replacement, James Rennell Rodd, been there the situation would not have gone the way it did under Mathews.[20(p500)] Portal had left Zanzibar for Britain on October 25, 1893, after completing his mission to Uganda. Rodd left with him as well, being extremely ill with malaria.[30(p339)] Portal had contracted typhoid fever and died in London on January 25, 1894.[31] Chanler said of Mathews who was now in charge:

> . . . affairs were in the hands of men who had been for many years inhabitants of the coast of East Africa, and as a natural result of long stays had become in large measure Arabized in character . . . instead of pursuing the simple and direct road to justice, the treatment of my affairs was made subservient to purely local, and I may also say private, ends.[20(p500)]

Chanler does not elaborate on what he means by "local and private ends," but these will become clearer further on.

Von Höhnel, who had left by the time of the mass desertion, saw it as a result of the poor quality of the men. In his reminiscences he says of the hiring of the porters:

> The blunder that had been made was the enlistment of the men in Zanzibar, who were unfit for such an expedition. When I saw the men on the morning following my arrival at Zanzibar, I found my worst apprehensions surpassed. With few exceptions, they were a rabble of untrained and too young men, who were not strong enough to carry a heavy load; fellows who for the most part, had never been in the interior and had no ideas of the fatigues that were in store for them, I was convinced that the expedition could not but be a failure.[28(p19)]

This statement made by von Höhnel in 1935 may explain why the porters deserted. However, it does not explain the subsequent course of events in which General Mathews played a central role. In order to comprehend what befell Chanler, one must have an overview of the dramatic events that unfolded in the British sphere of influence in East Africa between late 1892, when Chanler set out, and early 1894, when he returned.

THE BRITISH SPHERE, 1892–1894

Sir Gerald Portal became the British agent and consul general in Zanzibar on March 10, 1891. At this time the Imperial British East Africa Company (IBEA), which was primarily a trading company, was chartered to administer the British sphere, which included Uganda. By 1892, the IBEA had reached the end of its tether in East Africa and announced its intentions of withdrawing from Uganda by the end of the year. Undercapitalized and drained by the civil war between the Catholic and Protestant native forces in Buganda (January–March 1892), known as the Fransa-Ingliza War,[32(p145)] the company announced its plans to pull out by the end of 1892 and retreat to Dagoretti near modern-day Nairobi.[32(p145)] Frederick Lugard, the IBEA's man in Uganda, who had successfully negotiated a treaty between the French-led Catholics and English-led Protestants, was withdrawn by Sir William Mackinnon, the company's head, and reached London in October 1892.[33(p385)]

The British government was indifferent to this event and showed no interest in placing the area under direct British protection. In fact, at the time, the British were seriously considering withdrawing from Egypt as well.[33(p388)]

Lugard, an arch-proponent of British imperialism, lost no time lobbying for the establishment of a British protectorate, delivering speeches and writing articles and letters in the papers.[33(p388)] The spectre of Church Missionary Society (CMS) missionaries and their families stranded unprotected in the interior, coupled to the CMS's own campaign, temporarily swayed public opinion so that immediate abandonment was postponed. In addition, Bishop Tucker of the CMS, who was on leave in England, raised £15,000 from the CMS Gleaners Union, which coupled to the £25,000 raised by Sir William Mackinnon enabled the company to limp along until March 1893.[32(p148)] The foreign secretary, Lord Rosebery, then succeeded in getting the government to agree to send Gerald Portal on a visit to Uganda to make a report and recommendations.[33(p388)] Given Portal's support for imperial expansion it was almost a foregone conclusion as to what he would recommend.[32(p148),33(p388)] Portal left Zanzibar in January 1893,[30(p275)] and Rosebery asked James Rennell Rodd to go to Zanzibar and act in his absence. Thus at the time that Chanler arrived in Zanzibar in the summer of 1892, the IBEA was on the verge of collapsing, and to the expansionists like Portal and Mathews, the future of the British sphere seemed in peril of foreign takeover. However, Portal clearly did not see Chanler's presence as threatening at the time. If he had he would not have "offered me every assistance in his power . . . ." [20(p7)] Portal pulled down the IBEA flag in Uganda on April 1, 1893, and replaced it with the Union Jack.[32(p148)] He declared a provisional British protectorate which had no legal standing as it had not received the approval of the British government. In both Parliament and the cabinet many were opposed to establishing a protectorate in either Uganda or elsewhere on the East African mainland.[33(pp387–389)] There was a good possibility that the British sphere on the mainland would be abandoned. In April 1893, Chanler was moving into the middle of the sphere. To the imperialists like Mathews bent on retaining the mainland for Britain, he must now have become a threat, for if the IBEA pulled out then the sphere was up for grabs. And there was Chanler right in the middle of it with his American flag. In former times other lone travelers had succeeded in staking claims that were subsequently assumed by their governments. Thus the possibility of an American move into the vacuum could have been seen by some of the imperialists as being real.

In 1893, the full year that Chanler was in the interior, things otherwise went very badly for British interests in East Africa. In February, the IBEA's agent Todd in Kismayu was attacked by Somali.[30(p299)] Rodd had to rush from Zanzibar to defend the area as best he could. Just then Cecil Rhodes, the master empire builder, dropped in on Rodd in Zanzibar and urged the establishment of a protectorate on the mainland.[30(p300)] In Zanzibar itself matters sharply came to a crisis on March 5, 1893, when the Sultan Seyid Ali died and the succession was contested by Seyid Khalid, who secured the palace, and Seyid Hamed, whom the British favored.[30(p304)] An armed confrontation between Hamed and the forces of the challenger Khalid took place at the palace doors, Mathews threatening to blow them down with cannon if Khalid did not submit, which he eventually did.[30(p305)] While Rodd and Mathews eventually got the situation in hand and put the more cooperative Hamed on the throne, Khalid was not without his supporters.

Rodd and Mathews did not have much respite because in May 1893 the IBEA announced its withdrawal from Witu on the coast, whose sultan, Fumo Omari, now defied outside authority.[30(p317)] Eventually Mathews had to take a military expedition against Witu in July 1893. Fumo Omari and his bands were routed but not brought under administrative control. On July 31, 1893, Mathews hauled down the IBEA flag at Witu and flew the Zanzibari one, placing Witu under Zanzibar which itself was a British protectorate.[30(pp323–324)] This did not go over well with the British government, large segments of which were clearly leaning toward abandoning mainland holdings. Rodd was desperate as the IBEA was withdrawing its men and not replacing them, and he had but 150 Zanzibari troops in Uganda and 150 in Witu.[30(p337)]

Portal returned to the coast on October 22, 1893, and on October 25, 1893, left for England with Rodd who was severely ill with malaria.[30(p339)] Mathews was now in charge.

Possible Reasons for Desertion

Chanler's headman, Hamidi, had returned to Zanzibar for more porters after the military confrontation at Witu and the Somali rebellion at Kismayu early in the year. Hamidi would certainly have known about both events, which would have been threatening to both him and the porters as Chanler intended to traverse these areas later in his journey.

The fact that both Witu and Kismayu were insecure would not have deterred Chanler. However, it would certainly have frightened some inexperienced porters into cowardly defection. In addition, the IBEA was withdrawing from the areas south of the Tana River while Chanler was moving into the unexplored area to the north. This would have put the porters at even higher risk.

Chanler's men knew of the death of the sultan before von Höhnel left for Kibwezi, the news having been brought up by a trading caravan.[20(p402)] Chanler said of their reaction to this,

> . . . I could hear them discussing the probabilities of the successor attempting to free the Sultanate of Zanzibar from British influence . . . they seemed to think the time had come when the natives of Zanzibar should rise, and, throwing off the European yoke make Arab influence paramount along the coast. Had I been a stranger in Africa, it might have seemed odd to me that these men, for the most part slaves, should feel that their interests were in far greater degree with their masters than on the side of the British, who were ostensibly their friends and anxious to free them from servitude . . .[20(p402)]

Among the porters Hamidi brought from the coast were a group of 20 who ". . . had followed the fortunes of Bushiri, an Arab patriot, who had endeavored to prevent the Germans from taking that portion of East Africa which they had claimed. Many of them boasted of the numbers of Europeans they had slain. These men by their boastings quickly became heroes in my camp." [20(p423)]

Hamidi's group also brought distorted tales of Mathews' military campaign against Witu. Chanler said they gave ". . . highly colored accounts of victories achieved over the Europeans by the Arabs and outlaws in the neighborhood of Lamu. They said the time had at last come when the Arabs were about to reestablish themselves in Zanzibar on a firm footing and that messengers had come from Mecca advocating the 'Jehad' (holy war) against the infidel." [20(p419)] Chanler, true to form, called his men together and said that "not for a moment were lies of this sort to be tolerated in my camp; that any one of my men who would take the trouble to think for a moment should know the stories were absolutely untrue; and that I being a European, would not permit such rumours to circulate while I was there to stop them." [20(p419)] Chanler later learned what had really happened on the coast. However, his disparaging of the news brought up by Hamidi and the men from the coast did not and could not stop his porters from believing it. Thus, as some of the porters saw it, British rule was collapsing and the Arabs were about to rise again as an independent power on the coast. This was certainly no time to be away in the interior on the way to a distant lake, particularly if one were a battle scarred nationalist.

The porters had another reason to desert as well. Most of them had already gone up north to the Rendille country near the Mathews Range on Chanler's first visit there. They knew the road was rough and difficult and the Rendille unfriendly and dangerous. They had little stomach for such a difficult undertaking, especially after spending almost nine comfortable months in camp life at Daitcho. Galvin's return marked the point in time when easy camp life would end and the march north would begin. If defection were to occur it had to take place then, before the men were far from the well-trodden and relatively safe trade routes from the coast. Once they got up into Rendille country, defection would have proven suicidal because of the harshness of an environment devoid of food and plentiful water and because of the dangers posed by the Rendille and other groups. Defection out of fear of hardship or because of a simple desire to return to the coast would have deprived the porters of the monetary compensation due them according to the contracts made with them on Chanler's behalf by Smith, Mackenzie and Company in Zanzibar. Once the porters had decided on defection they had no choice but to claim cruelty as the reason so as to not jeopardize their wages.

At the time of defection in December 1893, the British sphere was essentially on the verge of being abandoned and as a consequence being put up for grabs. A protectorate over Uganda would not be confirmed in Parliament until August 1894.[32(p148)] Thus the sphere had entered a period of great vulnerability to foreign seizure as the imperialists saw it. What had been earlier viewed as an innocuous scientific expedition headed by Chanler would now have been seen as a major threat in the waning days of 1893. Thus Mathews would have had a strong motive in promoting the collapse of the expedition, as Chanler himself implies.[20(p462)] Whether he actually did is another matter.

Mathews' treatment of the deserters does not necessarily imply complicity in promoting the defection. However, it does represent a very pragmatic approach to a potentially serious local problem. Mathews could have imprisoned the deserters as was customary. Had he done so the government of Zanzibar would have run political risks imprisoning so large a number of men and significant costs housing and feeding them. The plantation owners to whom these men virtually belonged would have raised a fuss.

With a new, cooperative sultan uneasily seated on the throne, Mathews was clearly not going to antagonize power groups in Zanzibar in the interests of justice for an American explorer. Forces in Zanzibar unfriendly to Britain and desirous of throwing off the protectorate from the island were well aware of Britain's weakening hold on the mainland and would have made a move given the right opportunity. Their interest was to continue the banned slave trade and recoup lost local political power.[32(p162)] In 1895, when the mainland coast was transferred from the IBEA to the British government, the coastal Arabs who were closely integrated with Zanzibar did in fact rebel. Mathews obviously realized the delicacy of Britain's position in Zanzibar and sought to avoid unnecessary confrontations. Thus Chanler comes close to the truth when he says, "... the treatment of my affairs was made subservient to purely local, and I may also say private, ends." [20(p500)]

Rodd thought highly of Mathews, saying of him that he led "... a straight and beneficent life." [30(p348)] He characterized him as "... the most generous of men ... ." [30(p341)] Yet Mathews, who had by that time spent 17 years in Zanzibar, rising to the position of the sultan's prime minister, was clearly adept at statecraft.[33] Beneficent though he may have been, he could easily have sped Chanler on his way once the expedition collapsed, and although supporting documentation has not been uncovered it is not beyond the shadow of a doubt that he helped the desertion along. For as Rodd says, "The outlook was not a bright one on the coast in the latter months of 1893. The company was sending home officers and not replacing them." [30(p337)]

In summary, the available evidence suggests that there were a number of reasons why Chanler's porters would have wanted to desert. Von Höhnel was of the opinion that they were inexperienced, soft, unfit, and not up to the rigors of the required journey.[28(p19)] After nine months of easy camp life at Daitcho some of them would not have relished a return to the harsh semi-deserts of the north where they had previously encountered the unfriendly Rendille. Also, some would have been uneasy about going into the unknown north and through rebellion-wracked areas of the northeastern coast. Others, however, aware of the rapidly emerging political events on the coast, may have wanted to return home, believing that an Arab takeover was imminent. Still some neophytes at portering may have interpreted the usual and customary hardships and hazards of the job and Chanler's treatment of them according to then-current standards as unusual, harsh, and cruel. If they were inclined to think this, Hamidi, the experienced porter and headman, could have convinced them they were correct in this assessment. Thus there were several strong reasons for individual porters to desert. It appears that Hamidi, the headman, galvanized these to produce a mass desertion. He was able to do this by promising the men that they would receive their full pay on returning to Zanzibar.[20(p462)] Thus there would have been no material loss. If they were to receive full pay, clearly there would be no punishment either. The porters then would have seen desertion as free of both risks of punishment and loss of earnings. Whether Hamidi acted on his own or through prompting by Mathews out to achieve a limited political objective is an open question at present. However, Mathews' actions after the desertion lend support to the latter possibility.

CHANLER'S ACCOMPLISHMENTS

Chanler left Zanzibar for home after two months of fruitless effort. His antagonist, General Lloyd Mathews, continued to serve in Zanzibar until his death from cerebral malaria on October 11, 1901.[34] Chanler had much to be proud of. He had explored 2,500 square miles of new, unknown territory. He and von Höhnel fixed the exact geographical position of Mount Kenya's peak, discovered the Nyambeni Mountains, charted the course of the Guaso Nyiro, discovered the Chanler Falls, and found the Lorian Swamp. Chanler made the first European contact with the Rendille and discovered a new species of antelope, Chanler's reed buck, *Cervicapra chanlerii*.[28(p18)] He collected thousands of specimens of reptiles, beetles, butterflies, plants, and other specimens that he donated to the Smithsonian Institution.[20(p517)] A number of these were new to science, including two species of reptiles, *Mabuya chanleri* and *Simocephalus chanleri*; and two species of butterflies, *Iphthima chanleri* and *Charaxes chanleri*.[20(p518)] Chanler's collections were described in several scientific publications.[20(pp517-519)]

While Chanler's geographic explorations fell short of his original expectations, it seems he had kept his options open about this. In a letter written to von Höhnel on November 16, 1893, from Daitcho he said, "... If I get through Borana, I shall make for Ime and Berbera ... ." [35] Had he done so he would have gone through the country Arthur Donaldson Smith eventually explored, but only from the opposite direction. Even the day before his men deserted, he wrote to von Höhnel saying that he would wait a year for him to return if necessary. He then said that if he could not get through the Boran country he would return to Zanzibar.[36]

Chanler never returned to sub-Sahara Africa. He was elected to the New York State Assembly as a Democrat in 1896, campaigned in Cuba during the Spanish-American War, and served in congress for a term (1899–1901). After 1912 he lived in Paris breeding race horses. He married Beatrice Ashley, an accomplished sculptress and author, and they had two sons, William Astor and Sidney Ashley, both of whom are still alive. Chanler wrote two short novels in addition to his book on African exploration: *A Man's Game* (1921)[37] under the pseudonym John Brent and *The Sacrifice* (1925) under the pseudonym Robert Hart.[38] He suffered a leg amputation following an accident later in life and died at the age of 66 on March 4, 1934, in Mentone, France.[39,40] He is buried in the Chanler family vault in Trinity Cemetery in upper Manhattan.[41] Some of his African artifacts and trophies still adorn the main house at Rokeby, the Chanler family estate in Dutchess County, New York.

Ludwig von Höhnel maintained a life-long friendship with Chanler. He was at sea between 1894 and 1899, being promoted to Captain of Corvette in the latter year.[8] In the late 1890s his ship called at New York and Chanler took him in tow. He visited Rokeby and was taken by Chanler to Sagamore Hill in Oyster Bay, New York, where he met Theodore Roosevelt.[8] Between 1899 and 1903 he served as an aide de camp to Emperor Franz-Josef of Austria, and in 1903 was promoted to Frigate Captain. In 1905 he was sent by Austria on a diplomatic mission to Emperor Menelik of Ethiopia, and in 1909 was promoted to Rear Admiral.[8] Following World War I, von Höhnel and his wife, Laska, who were childless, fell on hard financial times. At Chanler's urging he wrote his memoirs, which were published in Germany in 1926 under the title *Mein Leben Zur See Auf Forfchunosreifen und* *beihofe.*[42] An English version of his memoirs written in his own hand was never published, nor was a book concerning his expedition to Africa with Chanler. Of this he said: "Several years ago I . . . composed my version of the expedition in German, in the hope I would find a publisher for the book. In this, however, I did not succeed . . . ." [28(p16)]

Von Höhnel did publish the survey results of the expedition which were supplemented with astronomical analyses by Dr J. Palisa.[43] This article contains a map drawn on a scale of 1:750,000 showing the features of the areas as well as profiles of mountain ranges.

Beginning in 1931 Chanler arranged to have 1,000 French francs ($40.00 US) sent to von Höhnel each month from his bank, Bankers Trust Company in Paris.[44] Chanler arranged that his name not appear on any of the correspondence and that the exchange costs be charged to his account.[43] Chanler had previously sent von Höhnel $400 in 1929 to pay his rent and to get his wife's jewels out of pawn.[45] Without Chanler's monthly check, von Höhnel and his wife would have been close to destitute, judging from the Chanler-von Höhnel correspondence in the Rokeby papers. Chanler also arranged that the stipend continue after his death, which it did.

Von Höhnel was deeply grieved by Chanler's death, writing about it to Chanler's sister, Margaret Aldrich,[46] "It is with a heavy and sorrowful heart that I console you . . . he was the truest and staunchest friend . . . he altogether was a hero, my hero . . . whom I always admired and dearly loved . . . ." [46] Von Höhnel died in Vienna on March 23, 1942, Chanler's widow Beatrice learning of it in January 1946.[47] At the time of his death the Nazi authorities in Vienna removed all of his papers from his house for safekeeping.[41] Their whereabouts, if they still exist, are unknown.

George E. Galvin faithfully remained with Chanler throughout the expedition (Fig 9). His devotion to Chanler's welfare constitutes a striking parallel with that earlier shown by James Martin to the British explorer Joseph Thomson (see part I). In 1905, Galvin married Helen Goldner in New York City and they had seven children. In the early part of the century he operated a horse farm in Leesburg, Virginia, and later ran a travel agency in both New York City and Paris. In New York City the agency was set up in the Vanderbilt Hotel, in which Chanler held a minority interest. Galvin lived for many years in Jamaica, New York, where he died in March 1951 at the age of 79.[48]

The British colonial authorities in Zanzibar may have taken some comfort in the fact that a potential threat to British interests in East Africa had been removed. Yet another determined American explorer was about to descend on them from the north. As Chanler was arriving in Trieste in May 1894 from Zanzibar,[20(p516)] Arthur Donaldson Smith, a physician from Philadelphia, was preparing to leave London for Somaliland. The authorities in Zanzibar would know about him before long.

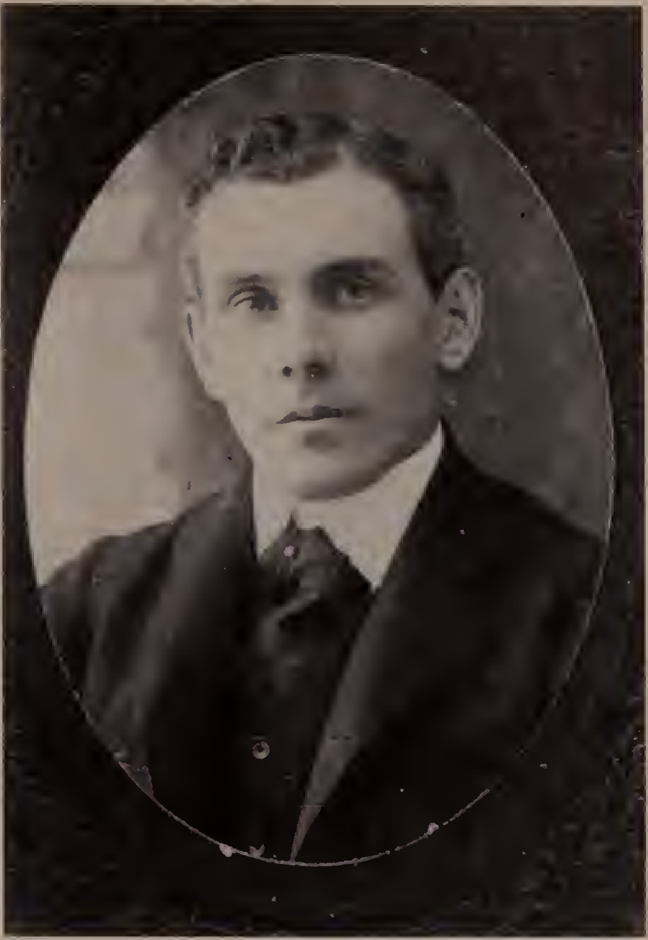


FIGURE 9. George E. Galvin in 1901 (courtesy of George E. Galvin, Jr).

REFERENCES

1. *Révai Nagy Lexikona Az Ismeretek Enciklopédiaya XVIII.* Kotet, Budapest, Révai Testverek Irodalmi Intézet Reszeny Tarsaság, 1925, pp 87–92.
2. Graf Samuel Teleki (obituary), *Wiener Abendpost*, March 11, 1916, p 3.
3. Kadar L: The Man Whose Name the African Teleki Volcano Bears. *Communications from the Geographical Institute of the University of Debrecen*, 1966–1967, pp/181–184.
4. Romai Szentbirodalmi Széki Gróf Teleki Samuel (death announcement), *Wiener Zeitung Jahrgang*, March 11, 1916.
5. Erdelyi L: *Teleki Samu Afrikaban.* Bucharest, Editura Kriterion, 1977.
6. Von Höhnel L: Admiral und Afrikaforscher, in *Osterreichisches Biographisches Lexikon 1815–1950.* Graz, Cologne, 1959, p 358.
7. Von Höhnel L: *Discovery of Lakes Rudolf and Stefanie: A Narrative of Count Samuel Teleki's Exploring and Hunting Expedition in Eastern Equatorial Africa in 1887 and 1888*, Bell N (D'Anvers) (trans). London, Longmans, Green & Co, 1894, vols 1, 2.
8. Von Höhnel L: *Over Land and Sea in Earlier Times and More Recent Days (1857–1909): Being an Austrian Rear Admiral's Own Narrative of His Life* (unpublished), written in 1926. William Astor Chanler Documents, Rokeby Papers (courtesy of Mr John Winthrop Aldrich).
9. Letter from Count Samuel Teleki to Crown Prince Rudolf, Leikipea, November 4, 1887. Crown Prince Rudolf Papers, Austrian State Archives, Vienna (in Hungarian).
10. Letter from Count Samuel Teleki to Crown Prince Rudolf, Baringo, December 16, 1887. Crown Prince Rudolf Papers, Austrian State Archives, Vienna (in Hungarian).
11. Letter from Count Samuel Teleki to Crown Prince Rudolf, Guasso Nyuki, December 22, 1887. Crown Prince Rudolf Papers, Austrian State Archives, Vienna (in Hungarian).
12. Letter from Alfred Oswald, consul of Austria-Hungary, to Count Kalnocky, Zanzibar, June 6, 1887. Austrian State Archives, Vienna (in German).
13. Letter from Alfred Oswald, consul of Austria-Hungary, to Count Kalnocky, Zanzibar, February 14, 1888. Austrian State Archives, Vienna (in German).
14. Letter from Alfred Oswald, consul of Austria-Hungary, to the Ministry of the Imperial House and Foreign Affairs, Zanzibar, May 8, 1888. Austrian State Archives, Vienna (in German).
15. Letter from Alfred Oswald, consul of Austria-Hungary, to the Ministry of the Imperial House and Foreign Affairs, Zanzibar, October 22, 1888. Austrian State Archives, Vienna (in German).
16. Von Höhnel LR: *Zum Rudolph-See und Stephanie-See. Die Forschungsreisse des Grafen Samuel Teleki in Ost-Aequatorial-Africa, 1887–1888.* Wien, A. Holder, 1892.
17. Borelli J: *Ethiopie Meridionale: Journal de mon voyage aux Pays Amhara, Oromo et Sidama Septembre 1885 à Novembre 1888.* Paris, Ancienne Maison Quantin, 1890.
18. Von Höhnel L: The Lake Rudolf Region. Its Discovery and Subsequent

Exploration 1888–1909. Part I. *J of the Royal African Society* 1938; 37:21–45.

19. Letter from Frank C. Strovas, Public Affairs Officer, US Embassy, Bucharest, Romania, to Pascal James Imperato, MD, October 24, 1986.

20. Chanler WA: *Through Jungle and Desert; Travels in Eastern Africa,* 1896, reprint ed. Detroit, Mich, Negro History Press, 1970.

21. Thomas L: *A Pride of Lions, The Astor Orphans. The Chanler Chronicle.* New York, William Morrow & Co, 1971.

22. DeMott M: William Chanler. *Africana* 1967; 3:15–19.

23. Meyer H: *Across East African Glaciers: An Account of the First Ascent of Kilimanjaro,* Calder EHS (trans). London, George Philip & Son, 1891.

24. Jackson F: *Early Days in East Africa.* London, Edward Arnold, 1930, p 160.

25. Frontera AE: *Persistence and Change: A History of Taveta.* Waltham, Mass, Crossroads Press, 1978, p 22.

26. Personal communication with William Astor Chanler, Jr, Camden, Maine, June 7, 1978.

27. Davis RH: An American in Africa. *Harper's New Monthly Magazine* 1893; LXXXVI:632–635.

28. Von Höhnel L: Reminiscences: An interview by William Astor Chanler, Jr, 1935. William Astor Chanler Papers, New York Historical Society.

29. Personal communication with William Chamberlain Chanler, New York, May 8, 1978.

30. Rodd JR: *Social and Diplomatic Memories 1884–1893.* London, Edward Arnold, 1922, vol 1.

31. Stephen L, Lee S (eds): Sir Gerald Herbert Portal, in *The Dictionary of National Biography.* Oxford, Oxford University Press, 1917, vol 16, pp 166–167.

32. Marsh Z, Kingsworth GW: *An Introduction to the History of East Africa.* Cambridge, England, Cambridge University Press, 1963.

33. Oliver R, Matthew G: *History of East Africa.* Oxford, England, Oxford University Press, 1963, vol 1.

34. Lee S (ed): Sir Lloyd William Mathews, in *The Dictionary of National Biography.* Oxford, England, Oxford University Press, 1912, vol 1, pp 591–592.

35. Letter from William Astor Chanler to Ludwig von Höhnel, Daitcho, East Africa, November 16, 1893. William Astor Chanler Correspondence, Rokeby Papers (courtesy of Mr John Winthrop Aldrich).

36. Letter from William Astor Chanler to Ludwig von Höhnel, Daitcho, East Africa, December 16, 1893. William Astor Chanler Correspondence, Rokeby Papers (courtesy of Mr John Winthrop Aldrich).

37. Brent J: *A Man's Game.* New York, The Century Co, 1921.

38. Hart R: *The Sacrifice.* London, Boswell, 1925.

39. Wm Astor Chanler is Dead in France. *NY Times,* March 5, 1934.

40. Chanler, William Astor, in *The National Cyclopedia of American Biography.* New York, James T. White & Co, 1935, vol 26, p 279.

41. Personal communication with John Winthrop Aldrich, Rokeby, November 2, 1986.

42. Von Höhnel L: *Mein Leben Zur See Auf Forfchunosreifen und beihofe.* Berlin, Verlag Von Reimar Hobbing, 1926.

43. Von Höhnel LR: Zur Karte des Nordostlechen Kenia—Gebrets. *Petermann's Mitteilungen* 1894; 40:193–199.

44. Letter from The Bankers Trust Company, Paris, France, to William Astor Chanler, October 21, 1931. William Astor Chanler Correspondence, Rokeby Papers (courtesy of Mr John Winthrop Aldrich).

45. Letter from William Astor Chanler to Ludwig von Höhnel, Paris, France, February 12, 1929. William Astor Chanler Correspondence, Rokeby Papers (courtesy of Mr John Winthrop Aldrich).

46. Letter from Ludwig von Höhnel, 61 Reisner Strasse, Vienna, Austria, to Margaret Aldrich, March 12, 1935. William Astor Chanler Correspondence, Rokeby Papers (courtesy of Mr John Winthrop Aldrich).

47. Letter from Laska von Höhnel to Beatrice Chanler, Vienna, January 5, 1946. Beatrice Chanler Correspondence, Rokeby Papers (courtesy of Mr John Winthrop Aldrich).

48. Letter from George E. Galvin, Jr, Albuquerque, NM, to John Winthrop Aldrich, November 2, 1986. Rokeby Papers (courtesy of Mr John Winthrop Aldrich).

Mesenteric cyst

VINCENT CALDAROLA, MD, JOHN LADUCA, MD

Mesenteric cysts are considered among the rarest of abdominal tumors and remain a medical curiosity. Despite their rarity, they still intrigue the general surgeon. During the past ten years, the observed incidence of these cysts has been one in 120,000 hospital admissions at the Erie County Medical Center.

CASE REPORT

A 59-year-old woman was admitted to the hospital on May 11, 1982, with a two-year history of an abdominal mass; the mass was increasing in size. The mass had originally been detected during a routine physical examination. At the time of admission, the patient was asymptomatic. The mass was palpable and very mobile, and there was no apparent abdominal wall fixation. The mass had grown slowly while the patient was followed with interval examinations every six months.

The patient denied any history of pain, fever, chills, anorexia, or weight loss. She had diabetes, which was being managed with diet and oral hypoglycemic agents. She had had four previous cesarian sections, the last having been 20 years ago. Her last menstrual period occurred five years prior to admission. A pelvic examination and Pap smear revealed no abnormalities.

During the physical examination, the patient was alert, well oriented, and in no acute distress. She was a well-developed, well-nourished woman. Her vital signs were stable: blood pressure, 120/60 mm Hg; pulse rate, 80/min; and oral temperature, 98.6°F. Her pulmonary and cardiovascular systems were normal. The abdomen was symmetrical, although mildly obese, with normal bowel sounds. A nontender, mobile, palpable mass was identified in the right flank. The mass was ovoid and measured approximately 4 × 4 cm in diameter. A rectal examination was noncontributory. A bimanual, internal gynecologic examination disclosed no masses.

Laboratory investigations revealed normal hemaglobin and white blood cell counts. Electrolyte levels and liver functions were within normal limits. An electrocardiogram and a chest radiograph revealed no abnormalities. A flat plate of the abdomen revealed a radio-opaque mass situated in the right flank. An upper gastrointestinal radiographic series demonstrated an extrinsic mass located in the mesentery of the small bowel (Fig 1). A barium enema was normal, but a sonogram of the abdomen identified areas of ecodensities related to possible calcification. A preoperative diagnosis of mesenteric cyst was made, and exploration was advised.

An exploratory laparotomy was performed on May 12, 1982, through a midline incision. A large mass was encountered in the distal jejunal mesentery (Fig 2). The mass was cystic and was enucleated without difficulty. The patient made an uneventful recovery and was discharged from the hospital on the sixth day after surgery.

The gross pathologic report of the surgical specimen disclosed a cyst measuring 7 × 6 × 6 cm in diameter (Fig 3). The central contents were a cheesy-yellow material. Microscopic analysis demonstrated lymphoid tissue and blood vessels, calcification, and a thick fibrous lining (Fig 4). Several slide preparations for tuberculosis and mycotic figures were performed and were negative. The final pathologic diagnosis was a mesenteric cyst.

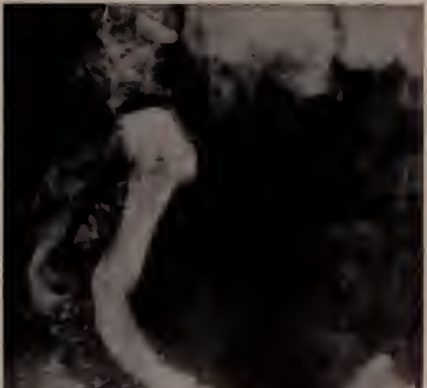


FIGURE 1. Upper gastrointestinal series demonstrating a mass effect in the right lower quadrant.

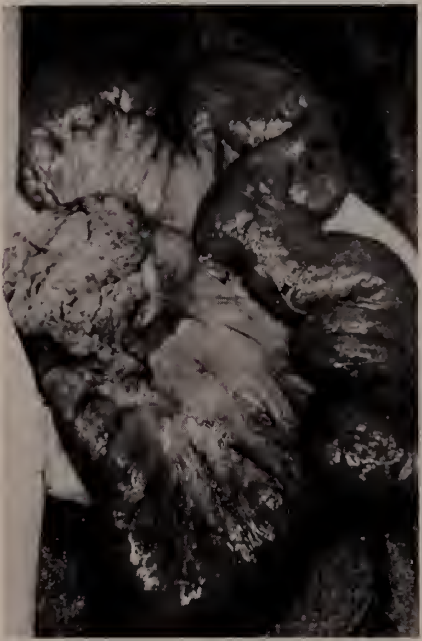


FIGURE 2. A large mass located in the mesentery of the distal jejunum and proximal ileum.

DISCUSSION

The etiology, pathology, classification, and incidence of mesenteric cysts have all been repeatedly disputed. Beneviene, a Florentine anatomist, first reported a mesenteric cyst discovered during an autopsy in 1507.[1] In 1948, Lee[2] proposed a theory that ruptured lymphatic vessels, fluid extravasation, and granulation tissue are responsible for cyst formation. In 1950, Gross[3] theorized that misplaced bits of lymph

---

From the Department of Surgery, State University of New York at Buffalo, Erie County Medical Center, Buffalo, NY.

Address correspondence to Dr LaDuca, Clinical Associate Professor, Department of Surgery, Erie County Medical Center, 462 Grider St, Buffalo, NY 14215.

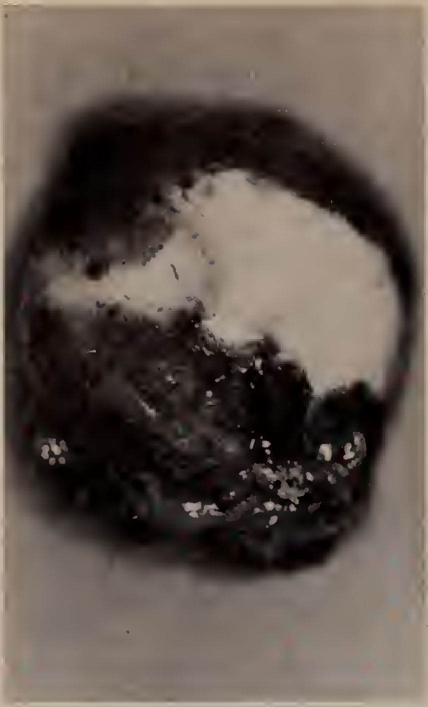


FIGURE 3. Enucleated specimen measured 7 × 6 × 6 cm and was encased in thick fibrous tissue.

tissue perforated, without access to drainage, thus creating a cyst. Beahrs et al,[4] in their 1948 review of nine cases, believed that the cysts are preformed or developmental abnormalities, possibly forming secondary to trauma or degeneration of lymph node tissue. Finally, Thompson and Chambers[5] and Hardin and Hardy[6] advanced the concept that a mesenteric cyst could result from an embryological failure of the leaves of the mesentery to fuse.

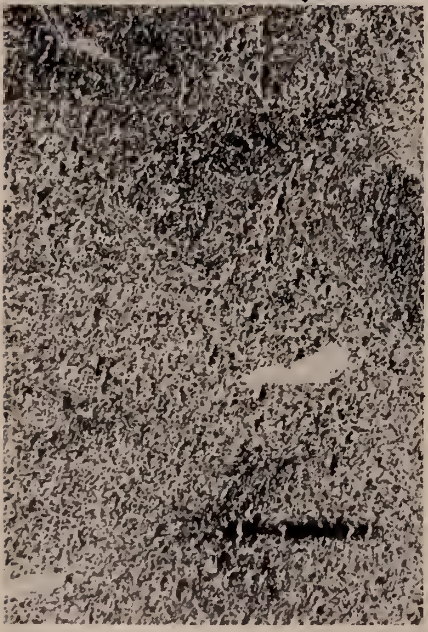


FIGURE 4. Photomicrograph demonstrates lymphoid tissue and blood vessels, calcification, and abundant fibrous tissue (hematoxylin-eosin stain; original magnification × 125).

Mesenteric cyst may occur at all ages, with a peak incidence in the fourth decade of life. The cystic lesions are uncommon, and the estimated incidence varies from one in 30,000 to one in 150,000 hospital admissions. A predominant 2:1 female-to-male ratio has been noted. There appears to be no predisposition in any ethnic or racial group.

A review of the medical literature revealed 57 reported new cases since 1950. The most commonly reported anatomical position of these cysts is the mesentery of the small bowel (47%), followed by the omentum (23%) (Table I). Sixty-one percent of the patients had a palpable mass, while the remaining 30% had complaints of pain (Table II). Eighty-seven percent of the patients had been treated by enucleation of the cyst; 10% of the cysts were resected. The mortality rate associated with surgical enucleation is 2% (Table III).

As identified by Simpson and Horsburgh,[7] most cysts tend to occur near or in the mesentery. They are not parasitic or dermoid, and are not derived from a normally placed retroperitoneal organ. A system for classifying the cysts was first proposed by Dowd[8] in the 1900s, and was modified by Hill[9] in the 1930s. The cysts can be of two types: simple and neoplastic. Beahrs and coworkers[4] reorganized the classification system into four main groups, based on the histopathological characteristics of the cyst wall: embryonic and developmental; traumatic or acquired; neoplastic; infected and degenerative.

It is apparent from the literature that cysts can be both multinodular and uninodular. The contents of the cyst may range from clear or gelatinous to turbid or creamy. The specific gravity ranges between 1.015 and 1.020, with biochemical components similar to plasma.

Although the cysts can occur in all possible locations, more than half of the 57 cysts reported in the literature since 1950 occurred in the mesentery of the small bowel, and one fourth occurred in the omentum (Fig 1). The original report[10] indicated that 46.5% of cysts occurred in the small bowel mesentery, while 13% were in the mesentery of the sigmoid colon, 11% in the mesocolon, 8% in the mesentery of the cecum, and only 2% were in the omentum. The microscopic features agreed with the 1950 criteria of Burnett et al.[10] The cells were of lymphatic origin, with endothelial lining, lymphatic vessels, and lymph tissue in the wall—with the presence of lymphocytes. Although this is one of the rarest types of abdominal tumors, malignant forms of this tumor have been reported. These are usually sarcomas, since all the cellular elements are of mesodermal origin.

TABLE I. Location of Mesenteric Cysts in 57 Cases Reported in the Medical Literature, 1951–1981

Location	Number	Percent
Small bowel mesentery	27	47%
Mesentery of sigmoid	4	7%
Mesocolon	7	12%
Mesentery of cecum	1	2%
Omentum	13	23%
Gastro-hepatic mesentery	1	2%
Retroperitoneum	4	7%

TABLE II. Medical History Obtained in 57 Patients with Mesenteric Cysts Reported in the Literature, 1951–1981

Symptom or Finding	Number	Percent
Palpable mass	35	61%
Pain	17	30%
Asymptomatic	13	23%
Diarrhea	9	16%

Patients commonly report a mass that appears or disappears. Prior to the availability of sonography and computed tomographic (CT) scanning, an accurate diagnosis was seldom made before an exploratory laparotomy, since there are no pathognomonic clinical features. The cyst may present in one of three ways: as an incidental finding at surgery; with no acute signs and symptoms; as an acute abdominal emergency. Symptoms can vary with patients. Although Caropreso[11] reported that 81% of patients had abdominal pain, 58% had a palpable mass, 45% had nausea and vomiting, 20% had constipation, and 6% presented with diarrhea, a review of the literature demonstrated a lower frequency of pain as a presenting sign (30%) or the absence of symptoms. This can be attributed to the availability of CT scanners and ultrasonography (Fig 2). The presenting symptoms are related to the size of the cyst and its anatomical relationship to adjacent organs. The clinical manifestations may be consistent with intestinal obstruction, volvulus, hemorrhage, rupture, or torsion. Physical examination will reveal a palpable, mobile mass that shifts from the right to the left flank easily because of its mesenteric attachment. This finding was described by Tillaux,[12]

TABLE III. Treatment and Outcome of 57 Patients with Mesenteric Cysts Reported in the Literature, 1951–1981

Procedure	Total Cases	Recovery	Deaths	Results Unknown
Enucleation (87%)	50	46	1 (2%)	3
Resection	6	4	1 (16%)	1
Drainage (2%)	1	1	0	0

who first successfully operated to remove a mesenteric cyst in 1880.

Laboratory data are essentially noncontributory to the preoperative diagnosis of mesenteric cyst. Examination of the abdomen by ultrasound, upper gastrointestinal series, barium enema, and CT scan has the most value in determining whether the tumor is cystic or solid, and whether or not it is calcified. The differential diagnosis includes ovarian cyst, pancreatic cyst, ascites, a pedunculated uterine fibroid, hydronephrosis, hydrops of the gallbladder, retroperitoneal tumor, mobile kidney, and an enlarged spleen.

The conventional treatment of mesenteric cyst is enucleation. Occasionally, marsupialization or resection of the intestine may be necessary due to involvement of the mesenteric vessels and adjacent gut. Mortality rates range from 0–8% with enucleation, to as high as 15% with bowel resection.

REFERENCES

1. Swartley WB: Mesenteric cyst. *Ann Surg* 1927; 85:886-896.
2. Lee FC: Large retroperitoneal chylous cyst; report of case, with experiments on lymphatic permeability. *Arch Surg* 1942; 44:61-71.
3. Gross RE: *The Surgery of Infancy and Childhood*. Philadelphia, WB Saunders, 1953, pp 377-383.
4. Beahrs OH, Judd ES Jr, Dockerty MB: Symposium on abdominal surgery; chylous cyst of abdomen. *S Clin North America* 1950; 30:1081-1096.
5. Thomson GCV, Chambers CH: Chylangioma of the mesentery with report of a case and a brief discussion of mesenteric cysts. *MJ Australia* 1946; 1:210-215.
6. Hardin WJ, Hardy JD: Mesenteric cysts. *Am J Surg* 1970; 119:640-645.
7. Simpson DA, Horsburgh AG: An unusual presentation of a mesenteric cyst of the sigmoid colon. *Postgrad Med* 1982; 58:586-587.
8. Dowd C: Mesenteric cysts. *Ann Surg* 1900; 32:515-542.
9. Hill LG: Lymphangioma of the abdomen. *Lancet* 1930; 2:897-898.
10. Burnett WE, Rosemond GP, Bucher RM: Mesenteric cyst. Report of 3 cases, in one of which calcified cyst was present. *Arch Surg* 1950; 60:699-706.
11. Caropreso PR: Mesenteric cysts: A review. *Arch Surg* 1974; 108:242-246.
12. Tillaux T-J: Lymphangioma of the mesentery (as translated by Crane). *Am J Surg* 1930; 9:441.

See also pp 140–141.

Ethylene glycol intoxication: A new stage in the clinical syndrome

STEWART A. FACTOR, DO, NEIL S. LAVA, MD

Ethylene glycol is a solvent best known for its use in antifreeze and its common misuse by alcoholics as a substitute for ethanol. The first cases of ethylene glycol intoxication were reported in 1930.[1] Since then, numerous others have been reported and a stereotyped clinical syndrome has been observed. Three stages are recognizable: Stage I refers to the central nervous system; stage II refers to the cardiopulmonary system; and stage III refers to the renal findings. All three will be discussed.

The patient described here, together with three recently reported cases,[2-4] represents a distinct new stage. This stage consists of facial diplegia and other neurologic sequelae not previously described in ethylene glycol intoxication.

CASE REPORT

A 46-year-old man with a history of alcohol abuse presented to a local community hospital three days after ingesting ethylene glycol in a suicide attempt. His initial complaints were headache, dizziness, and ataxia. Laboratory studies performed on admission showed a metabolic acidosis and an anion gap of 31. His blood urea nitrogen (BUN) level was 79 mg/dL on admission, and the creatinine level was 15.8 mg/dL. On the seventh day after ingestion, peritoneal dialysis was started, and the patient was transferred to Albany Medical College for hemodialysis.

A neurologic examination done on admission was within normal limits. All neurologic symptoms of which the patient originally complained were no longer present. On the 13th day after ingestion, the patient began suffering from intractable vomiting, which, after further investigation, was felt to be central in origin. On the 14th day, signs of facial diplegia developed; complete facial diplegia was noted by day 15. A computed tomographic (CT) scan and electroencephalogram (EEG) were done and were within normal limits. Studies of the cerebrospinal fluid (CSF) yielded a protein level of 333 mg/dL, a glucose count of 57 mg/dL, and no cells. Cytologic studies and cultures were negative. Electrodiagnostic studies done eight days later revealed bilateral facial neuropathy; the lower extremities were normal. Analysis of conduction velocities was repeated three weeks later and was improved. Brainstem auditory evoked potentials were measured and were found to be abnormal. The abnormalities suggested a left retrocochlear dysfunction including the brain stem. Visual evoked potentials were also abnormal. These abnormalities suggested the presence of bilateral optic nerve dysfunction or optic pathway dysfunction.

In addition, the patient underwent a renal biopsy. The biopsy demonstrated birefringent crystals in the tubular lumens consistent with calcium oxalate crystals. Focal tubular cell degeneration, atrophy, and tubular interstitial inflammation were also noted. During the next month, renal and neurologic sequelae improved, and the patient was discharged. He was kept on maintenance hemodialysis.

From the Department of Neurology, Albany Medical College, Albany, NY.

Address correspondence to Dr Lava, Associate Professor, Department of Neurology, Albany Medical College, Albany, NY 12208.

Discussion

Since 1930, many cases of ethylene glycol intoxication have been reported, and it has become clear that a fairly typical clinical syndrome is present. This syndrome consists of three stages,[5-7] which depend on the amount of ethylene glycol ingested.

Stage I occurs within 30 minutes to 12 hours after ingestion and involves the central nervous system (CNS). Clinically, the patient appears drunk but does not smell of alcohol. The CNS signs and symptoms can include dizziness, encephalopathy, ataxia, convulsions, coma, and, with large doses of ethylene glycol, death.[2] Other neurologic abnormalities have been reported.[2,8] Lactic acidosis, an anion gap greater than 20, and hypocalcemia are also associated with this stage.

Stage II occurs 12–24 hours after ingestion and involves the cardiopulmonary system. Typically, tachypnea, tachycardia, and hypertension develop. Pulmonary edema and congestive heart failure can cause death within 24–72 hours after ingestion.

Stage III occurs 24–72 hours after ingestion and involves the renal system. Clinical presentation varies from mild elevation in BUN levels to overt oliguric renal failure. The oliguria can last as long as 50 days[6]; a return to normal, or almost normal, is expected.

Since 1981, three cases of ethylene glycol toxicity similar to the case reported here have been described in the literature.[2-4] The clinical syndrome of these cases contains a distinct new stage of ethylene glycol intoxication. This stage, like stage I, features predominantly neurologic sequelae. The first case was reported in 1981 by Berger and Ayyar.[2] The second case was detailed in 1982 in a letter by Fellman,[3] and the third case was described by Mallya et al[4] in 1986.

All patients went through stage I of ethylene glycol intoxication to a mild extent, showing signs of agitation, irritability, lethargy, dizziness, or ataxia. (One patient did not seek medical treatment until 11 days after ingestion, so stage I is not described in the article.[4] Only one patient received acute care with ethanol, bicarbonate, and hemodialysis.[2]) Each patient went through stage III, requiring dialysis. None of them went through stage II, which is not atypical for this syndrome. In addition, all patients exhibited a fourth stage.

Stage IV involves the central nervous system, but occurs days after ingestion of the toxin—six days in the first case, and eight, 11, and 13 days for the other three cases, respectively. (The neurologic features of stage I typically occur within 12 hours.) Three patients each had facial diplegia and an elevated CSF protein level with no pleocytosis. (One patient did not undergo a spinal tap.[4]) More subtle sequelae included anisocoria, blurred vision, dysphagia, vomiting, hyperreflexia, and ataxia. The emergence of these new features in the clinical syndrome of ethylene glycol intoxication invites a few important questions. What is the pathophysiology of this fourth stage? The time factor alone would suggest a different mechanism from the other three stages. Why is bilateral facial nerve injury common to all patients in this stage? And why has this stage not been reported prior to 1981?

The mechanism of injury of stage I is unclear, as noted earlier. Because of the small number of patients and lack of pathologic studies in the four recent cases, the mechanism of stage IV is equally unclear.

A possible causative mechanism might be the Landry-Guillain-Barré syndrome. Facial diplegia is fairly common in this syndrome, and increased CSF protein levels with no cells is the rule. In ethylene glycol intoxication, the stimulus to the immune system might be the toxin(s) instead of, possibly, a virus, as in the typical Landry-Guillain-Barré syndrome. But some peripheral nerve involvement would be expected, and there has been none in patients with ethylene glycol intoxication.

A toxic metabolic mechanism of injury is another possibility. There are three metabolites of ethylene glycol that are known to accumulate in the central nervous system at high enough levels to be toxic. Oxalic acid causes cerebral oxalosis when precipitated as calcium oxalate crystals.[5,6,8] Glycoaldehyde in the CNS decreases cellular aerobic respiration and serotonin metabolism and alters amine levels.[6] It has been postulated that stage I correlates with the highest level of CNS glycoaldehyde production. This does not necessarily eliminate this mechanism as a cause of stage IV. Finally, a glycolic acid alters CNS metabolism and causes CNS depression.[6] Either individual metabolites or a combination of metabolites may be involved in the pathogenesis of stage IV.

Preferential injury to specific neural tissue in certain disease processes is not uncommon. In hypoxia, the hippocampus is preferentially injured, and in alcohol abuse, the Purkinje's cells of the cerebellar vermis are injured or destroyed. A disease process that more closely parallels the situation described here is superficial siderosis,[9] in which cranial nerve VIII is preferentially injured, and the patient presents with bilateral hearing loss along with other neurologic deficits. In stage IV of ethylene glycol intoxication, cranial nerve VII is damaged. The mechanism for specific susceptibility of some tissues to certain diseases has not yet been uncovered.

Before 1981, stage IV had not been seen in the clinical syndrome of ethylene glycol intoxication. We suggest that this is because, with the advent of better diagnosis and treatment—including dialysis—patients are surviving the first three stages and are living longer. Since more patients survive the initial poisoning, stage IV may develop in more patients. This fourth stage seems to have occurred with enough regularity that it should be recognized as a component of the clinical syndrome of ethylene glycol intoxication.

References

1. Brekke A: Two cases of poisoning by ethylene glycol; acute uremia cured by unilateral decapsulation of the kidney. *Norsk mag f laegevidensk* 1930; 91:381–388.

2. Berger JR, Ayyar DR: Neurological complications of ethylene glycol intoxication. Report of a case. *Arch Neurol* 1981; 38:724–726.

3. Fellman DM: Facial diplegia following ethylene glycol ingestion [letter]. *Arch Neurol* 1982; 39:739–740.

4. Mallya KB, Mendins T, Guberan A: Bilateral facial paralysis following ethylene glycol ingestion. *Can J Neurol Sci* 1986; 13:340–341.

5. Friedman EA, Greenberg JB, Merrill JP, et al: Consequences of ethylene glycol poisoning. Report of four cases and review of the literature. *Am J Med* 1962; 32:891–902.

6. Parry MF, Wallach R: Ethylene glycol poisoning. *Am J Med* 1974; 57:143–150.

7. Brown CG, Trumbull D, Klein-Schwartz W, et al: Ethylene glycol poisoning [clinical conference]. *Ann Emerg Med* 1983; 12:501–506.

8. Widman C: Few cases of ethylene glycol intoxication. *Acta Med Scand* 1946; 126:295–305.

9. Koeppen AH, Barron KD: Superficial siderosis of the central nervous system: A histological, histochemical, and chemical study. *J Neuropathol Exp Neurol* 1971; 30:448–469.

Acute disseminated intravascular coagulation as a complication of systemic lupus erythematosus

LESLIE DUBIN KERR, MD; HARRY SPIERA, MD; LOUIS N. ALEDORT, MD

Acute disseminated intravascular coagulation (DIC) is recognized as a complication of sepsis and malignancy. It is classically, but not invariably, characterized by hypofibrinogenemia, thrombocytopenia, elevated levels of fibrin degradation products (FDP), reduced factor V and VIII activity, and microangiopathic hemolytic anemia in association with excessive bleeding and/or thrombosis. Although the association between low-grade chronic intravascular coagulation and collagen vascular disease is widely accepted,[1] only two case reports mention the occurrence of acute DIC in a patient with systemic lupus erythematosus (SLE).[2,3]

Acute DIC may be a more prevalent complication of SLE than was previously thought. We describe four patients with SLE in whom life-threatening thromboses accompanied by laboratory evidence of acute DIC occurred, in each case causing significant morbidity. These thrombotic complications occurred despite the use of conventional therapy (high dose steroids and immunosuppressant agents) and rapidly resolved with intravenous heparin therapy.

CASE REPORTS

Case 1. A 57-year-old woman sought treatment for progressive weakness, arthralgias, pleuritic chest pain, increasing confusion, and skin lesions of three weeks' duration.

Physical examination was remarkable for bibasilar rales, an S3 gallop, scattered papular lesions on the upper back and trunk, and severe lethargy. The results of blood studies were as follows: white blood cell count, 2.4 × $10^3/\text{mm}^3$; hemoglobin, 7.1 g/dL; platelet count, 42 × $10^3/\text{mm}^3$; erythrocyte sedimentation rate, 55 mm/h; blood urea nitrogen, 35 mg%; and creatinine, 2.1 mg%. An electrocardiogram revealed a 1° atrioventricular block, with inverted T-waves in leads 2, 3, F, and V_4-V_6. A chest film showed cardiomegaly and bilateral lower lobe infiltrates. A Coombs' test was positive for immunoglobulins (Ig) G, IgM, C3, and C4. Laboratory tests were positive for antinuclear antibody (titer > 1:640), and negative for anti-DNA antibodies. The level of C3 was 54 mg/dL (normal > 90 mg/dL); the C4 level was 23 mg/dL (normal > 14 mg/dL). Urinalysis revealed 4–5 white blood cells per high power field, with no red blood cells or urinary casts. Cultures of cerebrospinal fluid (CSF) obtained via lumbar puncture grew no organisms. A skin biopsy revealed leukocytoclastic vasculitis. Tests for a circulating anticoagulant were negative.

During the first two days of her hospital stay, the patient became increasingly less alert, prompting the initiation of high dose steroid therapy (prednisone, 60 mg, every day). Her fibrinogen level dropped (Table 1), and the coagulation profile was consistent with DIC. She received heparin, 500 U/h, in addition to the steroids and broad spectrum antibiotics. Blood, urine, and CSF cultures were subsequently negative. After one week of treatment, the coagulation profile became normal, as did her mental status. In addition the infiltrates and vasculitic skin lesions disappeared.

Case 2. A 23-year-old woman with a one-year history of SLE manifested by arthralgias, myalgias, pleuropericarditis, and positive test results for ANA and anti-DNA antibodies, sought treatment for the acute onset of fever and arthralgias.

Physical examination on admission was remarkable for a fever of 103°F and vasculitic skin lesions. In addition, facial edema and non-pitting edema of all her extremities were noted and were attributed to protein-losing enteropathy associated with SLE. Neurologic examination revealed a Babinski sign on the left. Laboratory studies showed a white blood cell count of 13.0 × $10^3/\text{mm}^3$; platelet count, 100 × $10^3/\text{mm}^3$; and hemoglobin, 13 g/dL. Test results were positive for antinuclear antibody (titer, 1:80) and for anti-DNA antibodies, by *Crithidia lucillae* assay. The erythrocyte sedimentation rate was 1 mm/h. Tests for a circulating anticoagulant were negative. Urinalysis revealed 10–20 white blood cells per high power field. A chest film disclosed a left pleural effusion, and an electrocardiogram showed low voltage. A lumbar puncture showed the cerebrospinal fluid to contain no white blood cells and to have a protein level of 75 mg/dL and normal glucose levels.

The patient was treated with broad spectrum antibiotics and steroid therapy (prednisone, 60 mg/dL). Blood, urine, and CSF cultures were subsequently negative.

Eight days later, severe epigastric pain developed, and the patient had a serum amylase level of 1,365 mg/dL. The next day, erythema and edema of the right arm and thigh occurred, and deep venous thrombosis in both extremities was confirmed by Doppler study. Repeat coagulation studies confirmed the clinical diagnosis of DIC. In addition, she had a focal seizure thought to represent lupus cerebritis. High dose steroid therapy was continued. Intravenous heparin therapy (500 U/h) was added to the drug regimen and was associated with resolution of her symptoms and with a return to normal of the coagulation profile.

In the setting of active cutaneous vasculitis, protein-losing enteropathy, and cerebritis—which represent diverse manifestations of active SLE vasculitis—the development of pancreatitis was inferred to be secondary to the vasculitis as well. The fact that the pancreatitis resolved rather than worsened on high dose steroid therapy further suggested that it was a vasculitic phenomenon rather than a complication of steroid therapy.

Case 3. A 25-year-old woman who had had SLE for six years with evidence of a circulating anticoagulant was transferred to this hospital with sharp epigastric pain, headache, fever, and accelerated hypertension.

On admission, her temperature was 101°F and the blood pressure was 160/110 mm Hg. Physical examination was remark-

---

From the Divisions of Rheumatology (Drs Kerr and Spiera) and Hematology (Dr Aledort), Mount Sinai School of Medicine, New York.

Address correspondence to Dr Kerr, Instructor in Medicine, Division of Rheumatology, Mount Sinai Hospital, One Gustave L. Levy Place, New York, NY 10029.

TABLE I. Summary of Coagulation Profiles Before, During, and After the Thrombotic Event

	Case	Hemoglobin (g/dl)	Platelets × 10^3/mm^3	Prothrombin Time (sec)	Partial Thromboplastin Time (sec)	Fibrinogen (mg%)	Fibrin Degradation Products nl < 10 (μg/mL)	Erythrocyte Sedimentation Rate (mm/h)
I		7.1	124	13/12	50/39	130	10–40	55
II	1	11.8	85	15/12	75/57	50	>40	—
III		9.6	179	12/12	—	275	<10	—
I		13.9	94	11/11	41/control	—	<10	1
II	2	13.9	100	13.7/11	78/control	75	10–40	2
III		12.5	100	13/11	—	150	<10	2
I		8.4	100	15.6/11	68/control	330	—	92
II	3	9.1	81	15.6/11	68/control	330	>40	—
III		10.0	80	18/11	150/control	380	<10	43
I		7.7	57	11/11	—	565	—	3
II	4	7.9	105	15/12	—	380	<10	—
III		5.2	100	12/12	115/control	460	<10	—

I, on admission
II, during thrombotic event
III, clinical resolution of thrombosis (PTTs reflect heparin therapy)
—, data not obtained

able for malar erythema, epigastric tenderness without peritoneal signs and hepatosplenomegaly. Laboratory tests were strongly positive for antinuclear antibody (titer, 1:640) and were positive for anti-double stranded DNA antibodies. The clinical impression was intestinal vasculitis; steroid therapy—oral prednisone, 60 mg/day—was begun.

Two days later, the patient complained of intense headache with blurred vision. Examination revealed flaring of the malar rash, evidence of ischemic optic retinopathy, and new palmar vasculitic skin lesions. During the next 24 hours, she became blind and lapsed into a coma. A brain scan and computed tomographic scan of the brain revealed bilateral occipital lobe infarctions. She was transferred to the intensive care unit. The coagulation profile was consistent with DIC, and she was treated with intravenous heparin (500 U/h), pulse steroids (1 g methyl prednisolone), cyclophosphamide (100 mg/day), and broad spectrum antibiotics. Subsequently, all blood, urine, and CSF cultures were negative. After a prolonged stay in the intensive care unit, complicated by seizures and massive gastrointestinal bleeding, she regained normal mental status, her vision returned, all the coagulation abnormalities resolved, and she was discharged from the hospital after a four-month stay.

Case 4. A 33-year-old woman had a 14-year history of SLE with biopsy-proven, diffuse proliferative glomerulonephritis, and a strongly positive titer of antinuclear antibody. She was admitted to the hospital with uremia, profound anemia, and hypotension.

Physical examination was remarkable for a blood pressure of 98/64 mm Hg, scattered lower extremity ecchymoses, and guaiac-positive stools. Initial laboratory data were remarkable for the following: blood urea nitrogen, 292 mg%; creatinine, 6.8 mg%; carbon dioxide, 5 meq/L; creatine phosphokinase (CPK), 492 mg/mL; MB fraction of CPK negative; hemoglobin, 7.4 g/dL; platelet count, 57 × 10^3/mm^3; erythrocyte sedimentation rate, 3 mm/h. A Coombs' test was negative, and there was no evidence of a circulating anticoagulant.

Urinalysis revealed 2+ protein, many white blood cells and red blood cells per high power field, with no casts. An electrocardiogram showed new q waves in leads 2, 3, and F, consistent with a recent ischemic event. Blood and urine cultures were negative.

The patient was treated with antibiotics, hydration therapy, and daily high dose steroid therapy (8 mg dexamethasone on odd days alternating with 4 mg dexamethasone on even days), as well as with transfusions, which, despite persistent anemia, enabled normalization of her blood pressure and resolution of the acidosis. A bluish discoloration and edema of her lower extremities then developed. Doppler studies confirmed the presence of bilateral iliofemoral deep venous thrombosis.

The clinical impression, in light of the low fibrinogen level, thrombocytopenia, anemia, and multiple thromboses was DIC. Intravenous heparin therapy, 500 U/h, was started. Subsequently, the coagulation abnormalities resolved, and the deep venous thrombosis cleared, as confirmed by repeat Doppler studies four weeks later.

DISCUSSION

When an occlusive vascular event occurs in a patient with SLE, it is often difficult to determine on clinical grounds whether the thrombosis was initiated by vessel obstruction or by vasculitis. This distinction is crucial because of its therapeutic implications. These four cases demonstrate that even when the distinction between obstruction versus vasculitis appears to be clear, it is important to entertain the additional differential diagnostic possibility of DIC. If the coagulation profile supports the diagnosis of DIC, then full dose heparin therapy (500 U/h) should be instituted, in addition to aggressive therapy for vasculitis.

Although there is an association between the presence of circulating anticoagulants and thrombotic events, there is no known association between the presence of circulating anticoagulants and acute DIC. A circulating anticoagulant was detected in only one of these four patients (case 3). This patient had striking evidence of an acute consumptive coagulopathy at the onset of her thrombotic event. Both the thrombosis and the coagulopathy resolved coincident with the addition of heparin to the therapeutic regimen. Thus, we ascribe her thrombotic event to DIC rather than to the presence of the circulating anticoagulant.

All of the patients presented in this paper had active SLE with ongoing vasculitis as a major manifestation. Accordingly, they received aggressive treatment consisting of high dose steroid therapy (prednisone, 60 mg/day or its equivalent) alone or in combination with cyclophosphamide. In spite of this intervention, life-threatening vascular occlusive events occurred accompanied by laboratory evidence of acute DIC. In addition, strikingly low sedimentation rates reflected profound hypofibrinogenemia. Neither the thrombotic phenomena nor the coagulation abnormalities were reversed by immunosuppressive therapy alone.

A similar experience was recently reported by Chellingsworth et al.[2] That patient had persistent evidence of DIC and extensive thrombosis despite treatment with high dose prednisone and cyclophosphamide. In the cases described here, the addition of heparin was associated with clinical resolution of the thromboses as well as a reversal of the coagulopathy. Thus, heparinization appears to be an essential adjunct to immunosuppressive therapy in patients with SLE and DIC.

Athough the hypothesis that vasculitic endothelial injury initiates thrombosis is widely accepted, the exact mechanisms whereby the coagulation system is activated are unknown. Immune complex deposition,[3,13,17] procoagulant release from damaged endothelial cells, smooth muscle cells, or fibroblasts,[4] Hageman factor activation,[5,6,14] complement activation,[15,16] and platelet activating factor[6] have all been postulated as explanations for coagulation system activation in the setting of vasculitis. Clearly, further study is required to elucidate these mechanisms.

REFERENCES

1. Owen CA Jr, Bowie EJ: Chronic intravascular coagulation syndromes: A summary. *Mayo Clin Proc* 1974; 49:673-679.
2. Chellingsworth M, Scott DG: Case report: Acute systemic lupus erythematosus with fatal pneumonitis and disseminated intravascular coagulation. *Ann Rheum Dis* 1985; 44:67-69.
3. Whitaker AN, Emmerson BT: Acute disseminated intravascular coagulation in systemic lupus erythematosus. *Aust NZ J Med* 1972; 2:182-187.
4. Sergent JS, Sherman RL, Al-Mondhiry H: Fibrinogen catabolism in systemic lupus erythematosus. *Arthritis Rheum* 1976; 19:195-198.
5. Hardin JA, Cronlund M, Haber E, et al: Activation of blood clotting in patients with systemic lupus erythematosus. *Am J Med* 1978; 65:430-436.
6. Cochrane CG, Wuepper KD, Aiken BS, et al: The interaction of Hageman factor and immune complexes. *J Clin Invest* 1972; 51:2736-2745.
7. Conesi BA, Banfi F, Rossi AF, et al: Activation of the coagulation system in connective tissue diseases. *Scand J Rheumatol* 1980; 9:266-270.
8. Robbins J, Stetson CA Jr: An effect of antigen-antibody interaction on blood coagulation. *J Exp Med* 1959; 109:1-8.
9. Cooper HA, Bowie EJ, Owen CA Jr: Evaluation of patients with increased fibrinolytic split products (FSP) in their serum. *Mayo Clin Proc* 1974; 49:654-657.
10. Kanyerezi BR, Lwanga SK, Bloch KJ: Fibrinogen degradation products in serum and urine of patients with systemic lupus erythematosus. Relation to renal disease and pathogenetic mechanism. *Arthritis Rheum* 1971; 14:267-275.
11. Marchesi SL, Aptekar RG, Steinberg AD, et al: Urinary fibrin split products in lupus nephritis. Correlation with other parameters of renal disease. *Arthritis Rheum* 1974; 17:158-164.
12. Silverman ED, Miller JJ, Bernstein B, et al: Consumption coagulopathy associated with systemic juvenile rheumatoid arthritis. *J Pediatr* 1983; 103:872-876.
13. Colman RW, Robboy SJ, Minna JD: Disseminated intravascular coagulation (DIC): An approach. *Am J Med* 1972; 52:679-689.
14. Ratnoff OD: Some relationships among hemostasis, fibrinolytic phenomena, immunity, and the inflammatory response. *Advances Immun* 1969; 10:145-227.
15. Kaplan AP, Silverberg M, Dunn JT, et al: Mechanisms for Hageman factor activation and the role of HMW kininogen as a coagulation cofactor. *Ann NY Acad Sc* 1981; 370:253-260.
16. Zimmerman TS, Muller-Eberhard HJ: Blood coagulation initiation by a complement-mediated pathway. *J Exp Med* 1971; 134:1601-1607.
17. Tomar RH, Kolchins D: Complement and coagulation. Serum beta-lc-beta-la in disseminated intravascular coagulation. *Thromb Diath Haemorrh* 1972; 27:389-395.
18. Movat HZ, DiLorenzo NL: Activation of the plasma kinin system by antigen-antibody aggregates. I. Generation of permeability factor in guinea pig serum. *Lab Invest* 1968; 19:187-200.
19. Horowitz HI, Des Pres RM, Hook EW: Effects of bacterial endotoxin on rabbit platelets. II. Enhancement of platelet factor 3 activity in vitro and in vivo. *J Exp Med* 1962; 116:619-633.
20. Kaplan AP, Austen KF: Activation and control mechanisms of Hageman factor-dependent pathways of coagulation, fibrinolysis, and kinin generation and their contribution to the inflammatory response. *J Allergy Clin Immunol* 1975; 56:491-506.
21. Kaplan AP, Meier HL, Mandle R Jr: The Hageman factor dependent pathways of coagulation, fibrinolysis, and kinin-generation. *Semin Thromb Hemostas* 1976; 3:1-26.

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication. All letters are personally acknowledged by the Editor.*

Male to female transmission of AIDS

TO THE EDITOR: With regard to the case reported by Miller, Deluca, and Ringler, "AIDS in the wife of a bisexual man," [1] I would like to know if it was determined whether or not the patient had had rectal intercourse with her husband.

HERBERT BERGER, MD
7440 Amboy Rd
Staten Island, NY 10307

1. Miller SN, Deluca PS, Ringler RP: AIDS in the wife of a bisexual man. *NY State J Med* 1986; 86:158–159.

In reply. I appreciate the question raised by Dr Berger. I think his concern about anal receptive intercourse is valid as this is likely a more efficient way to transmit HIV (formerly called HTLV-III/LAV) than is vaginal intercourse. Unfortunately we did not specifically question the patient about this. She died on April 9, 1986, and her ex-husband could not be located. Her preterminal course was complicated by blindness secondary to cytomegalovirus (CMV) retinitis and a prolonged psychiatric hospitalization for an unsuccessful suicide attempt.

STEVEN N. MILLER, MD
Department of Medicine
Huntington Hospital
270 Park Ave
Huntington, NY 11743

Emergency medical services

TO THE EDITOR: I read with interest the excellent article, "Emergency medical services: Twenty years of growth and development," by Anthony C. Mustalish, MD, in the August 1986 issue of the *Journal.*[1] I notice that in the section on national professional organizations the contributions of the American College of Physicians were not covered. In 1971, while representing the College at a conference in Bethesda, Maryland,[2] I proposed that all able-bodied individuals in the United States over the age of ten be trained to perform cardiopulmonary resuscitation (CPR). The following April at the Annual Session in Atlantic City a resolution[3] was approved by the Regents and Governors urging the total membership of the American College of Physicians to join with the American Heart Association in a national effort to mass-train citizens in the procedure. The American Medical Association and *The New York Times*[4] reported the event, and later that year, again representing the American College of Physicians on a panel at the American Heart Association meeting in Dallas, I presented the plan.[5] Dr Lee Cobb of Seattle and Dr Eugene Nagel of Jacksonville, Florida, were also on the panel. Both had been training firemen and medical technicians in basic life support but to my knowledge no one had started to train all able-bodied citizens over the age of ten years to do CPR.

It may also be of interest that the poor quality of ambulance services during the 1950s and 1960s encouraged Dr Halloway and Dr Zein and myself to design the first mobile emergency room.[6,7] After its construction, the vehicle was donated to the City of New York. Based at Kings County Hospital in Brooklyn, it was used to serve people in the Bedford Stuyvesant section of Brooklyn. It became the prototype for several "baby Mervans" built in New York and elsewhere.

JULIUS E. STOLFI, MD
SUNY Health Science Center
at Brooklyn
450 Clarkson Ave
Brooklyn, NY 11203

1. Mustalish AC: Emergency medical services: Twenty years of growth and development. *NY State J Med* 1986; 86:414–420.
2. *Proceedings of the Second National Conference on Emergency Health Services,* Bethesda, Md, December 2–4, 1971. Rockville, Md, Department of Health, Education and Welfare, 1972, p 38.
3. Stolfi JE: Resolution: The Committee on Hospitals, presented at the American College of Physicians Annual Sessions, Atlantic City, NJ, April 1972.
4. Schmeck HM: Physicians propose training of public to aid heart cases. *The New York Times,* April 20, 1972.
5. American Heart Association Assembly Panel: VII Emergency Cardiac Care Delivery Systems, presented at the American Heart Association Meeting, Dallas, November 20, 1972.
6. Stolfi JE, Holloway RM, Zeins W: Design of a mobile emergency unit. *NY State J Med* 1972; 72:630–633.
7. Holloway R, Stolfi JE: Mobile vans as disaster scene emergency rooms. *Hospitals* 1972; 46:43–47.

In reply. Dr Julius E. Stolfi's letter correctly presents the pioneering efforts of the American College of Physicians in the growth and development of emergency medical services. Dr Stolfi and others contributed greatly to these efforts during those early years. Cardiopulmonary resuscitation (CPR) training in both first responder programs as well as in lay training programs was an important element leading to the acceptance of this training.

Dr Stolfi deserves special credit and recognition not only for his advocacy and support of CPR, but also for his efforts with early ambulance design. He deserves our thanks, as do all who labored in the vineyard in the earlier days and somehow are forgotten or inadvertently not acknowledged during the harvest period. I applaud their work and express appreciation for the many of us who enjoy the benefits of their efforts.

ANTHONY C. MUSTALISH, MD, MPH
Chief, Emergency Services
Lenox Hill Hospital
100 E 77th St
New York, NY 10021

A "pack of trouble" for smoking drivers

TO THE EDITOR: The health consequences of smoking on both smokers and nonsmokers are complex because the rights of each group are also at issue. There are many excellent reasons for smokers to abandon their habit, with toxic effects on the pulmonary and cardiovascular systems clearly documented, and passive exposure risks to nonsmokers rapidly being demonstrated.[1] An interesting connection between smoking and the incidence of motor vehicle accidents (MVAs) and traffic violations has recently been published in the *Journal*. DiFranza et al[2] attempt to show, in a group of smokers vs nonsmokers, that over a period of one year the relative risk of an accident on the road is 1.46:1.00. The interpretation of these results is not clear. Data must be precise and carefully analyzed before one reaches conclusions that may have future legal ramifications for smokers—in terms of their qualifications to obtain a driver's license, for example, if they are found to be impaired drivers.

Some possible bias may have been introduced into this study by the use of a group of drivers with only "minor infractions," excluding the drunken-driver violators. The authors state that smokers may have a heavier use of alcohol. Thus the absence of this group may be statistically significant. The actual amount of alcohol that constitutes a "drink" was never stated in the methods. Having had nine drinks or none is statistically equal here and the relative proportions of each are unstated. The second group of subjects, those applying for driver's license renewal, may have been a better group to look at in the smoker-to-nonsmoker ratio for traffic violations, although age and driving experience still seem to be predominant as risk factors. The drug use effect was intentionally, but unfortunately, omitted, and the actual percentage of drivers without violations excluded.

The specific effect of smoking on driving, if true, is speculative. The paper did not discuss whether or not smokers had actually been smoking at the time of their accident or violation. This would seem to be of great importance in looking at the question of personality versus some distractive or physiological problem. There is no indication if nonsmokers had accidents with smoking passengers in their cars exerting a passive effect. No observations were made of the incidence among smokers of accidents prior to the onset of their smoking habit, unless all smokers had started their habit before driving. It would have been useful to look at the night accident incidence rate given the concern for effects of carbon monoxide on night vision. That smokers may have personality traits different from nonsmokers, rendering them more accident-prone, is a clever speculation. If this is proven to be the case, as proposed but not yet documented, then the logical inference would be that psychological testing for these reckless traits may be needed and could even factor into insurance coverage.

Society restricts seizure patients, persons suffering from alcoholism, and the visually impaired from the road. Proper deliberation must be taken before accepting conclusions that, if valid, could lead to restrictions or penalties for smokers. A good population for restudy of this issue might be professional bus drivers, who spend roughly equal amounts of time on the road and who would have similar characteristics. Further study of the relationship between smoking and MVAs and violations needs to be conducted, as has been pioneered in the paper by DiFranza et al. In the meantime, however, there would seem to be other, more compelling reasons to quit smoking, as the effects of cigarettes "off the road" are incontestable.

KIMBERLY HALL FERNANDEZ, MD
FRANK M. TAMARIN, MD
ROBERT D. BRANDSTETTER, MD
Department of Medicine
New Rochelle Hospital Medical Center
16 Guion Pl
New Rochelle, NY 10802

1. Weiss ST: Passive smoking and lung cancer. What is the risk? *Am Rev Respir Dis* 1986; 133:1-3.
2. DiFranza JR, Winters TH, Goldberg RJ, et al: The relationship of smoking to motor vehicle accidents and traffic violations. *NY State J Med* 1986; 86:464-467.

In reply. The letter by Drs Fernandez, Tamarin, and Brandstetter addresses several issues including selection bias, the role of alcohol, and the need for caution in applying our findings to areas of public policy.

We agree with Dr Fernandez et al that our choice of a high-risk population to study the association of smoking and motor vehicle accidents may have influenced the magnitude of the association found. This population consisted of drivers attending traffic safety school because they had recently received a ticket for a minor traffic violation, not including drunken driving. Although these drivers were not drunk at the time they received the ticket which brought them to the school, they may have been arrested previously for drunken driving. The exclusion of drunken drivers may have magnified the association between smoking and accidents since we found the greatest effect of smoking among those who did not drink. On the other hand, the influence of smoking may have been underestimated if risk taking by smokers is an important factor in smoking-related accidents, since most of the drivers in this population, both smokers and nonsmokers, had taken some risk resulting in the violation. Although the use of this particular population might have had an effect on the magnitude of the association we found, it is unlikely that selection bias produced the association between smoking and accidents. Such an association has also been found by Adams and Williams[1] among male insurance applicants, by McGuire among male Air Force recruits,[2] by Waller[3] among accident fatalities, and, most importantly, by the Farmer's Insurance Company in the general driving population.

The strong association between smoking and drunken driving and between drunken driving and accidents clearly suggests a potential confounding role for alcohol in the association of smoking and accidents.[4] For the purposes of this study, drivers were asked to report anonymously the number of drinks (beer, wine, or hard liquor) they have in a typical week. Twenty three percent of drivers reported they do not drink, 17% reported having more than ten drinks in an average week. Because the self reports obtained in this study were not verified, it is reasonable to question whether alcohol can be ruled out as a confounding factor. The greatest difference in accident rates seen between smokers and nonsmokers was among drivers who stated that they average less than one drink per week. Although self reports of drinking are notorious for underreporting, it is unlikely that, in an anonymous setting, heavy drinkers would underreport to the degree of completely denying drinking. Thus in the population for which the data is probably most accurate (ie, nondrinkers), the greatest association between smoking and accidents independent of alcohol use was seen. Also, Waller,[3] in his study of driver fatalities using the "gold standard" of blood alcohol concentrations, has also shown an effect of smoking independent of that of alcohol.

Our findings have generated many questions regarding possible policy changes that might follow from the association of smoking and accidents. Should smoking while driving be prohibited, should ashtrays and lighters be eliminated from future vehicles, should smokers be prohibited from driving? We do not feel that the magnitude of the association between smoking and crashing is significant enough to warrant prohibiting smokers from driving, and not enough is known about the mechanisms involved to warrant prohibiting smoking while driving. Should smokers pay more for collision insurance and should nonsmokers be hired preferentially as drivers? We can now conclude with confidence that smokers as a group are more dangerous drivers and therefore both of these policy changes would be logical applications of that knowledge.

JOSEPH R. DiFRANZA
Department of Family
and Community Medicine

ROBERT J. GOLDBERG, PhD
Department of Behavioral and
Preventive Medicine
University of Massachusetts
Medical Center

THOMAS H. WINTERS
Department of Clinical Medicine
Boston University School of
Medicine

LEONARD CIRILLO, PhD
Department of Psychology
Clark University

TIMOTHY BILIOURIS
University of Massachusetts
Medical School

1. Adams JR, Williams EB: The association between smoking and accidents: Overdependency as an influencing variable. *Traffic Q* 1966; 20:583–588.
2. McGuire FL: Smoking, driver education and other correlates of accidents among young males. *J Safety Res* 1972; 4:5–11.
3. Waller JA: On smoking and drinking and crashing. *NY State J Med* 1986; 86:459–460.
4. DiFranza JR, Winters TH: Smoking and drunk driving. *N Engl J Med* 1985; 313:1422–1423.

Breast carcinoma in a man following local trauma

TO THE EDITOR: Carcinoma of the male breast is a rare malignant disease that accounts for 0.1–0.2% of all male cancer deaths and about 1% of all breast cancers. The annual incidence of this cancer is about 0.6–0.7 per 100,000 men in the US; about 300 deaths a year are reported as being caused by this form of cancer.[1-4]

The infrequency of breast carcinoma in men has resulted in a paucity of epidemiologic studies, and the etiologic factors involved are still obscure. It is known, however, that Klinefelter's syndrome has been associated with male breast cancer, and gynecomastia per se may be a predisposing factor.[2,3] Other risk factors appear to be x-ray exposure, infectious orchitis, hepatic schistosomiasis with hyperestrogenism, and exogenous estrogen therapy.[2,3,5-7] These observations suggest that there is probably a relationship between male breast carcinoma and the ratio of estrogen to androgen. An association between trauma and male breast cancer has been reported by some authors, but no detailed history or description of any pre-existing lesion was presented.[2,6,8]

We describe a man in whom breast cancer developed ten years after a wound to his left breast.

Case Report. A 67-year-old man was in good health until two months prior to admission, when he started to notice weakness, fatigue, exertional dyspnea, and weight loss (15 lbs in three months), as well as a left axillary mass. His medical history was remarkable only for mild hypertension; no history of x-ray exposure was obtained.

After six weeks he consulted a physician, who noted an indurated scar, which measured about 5 × 2 cm, involving the left areola and nipple. A small ulceration was present at the level of the remnant of the areola. The patient described a wound to the left breast incurred during a car accident ten years before. The nipple had been almost completely sectioned off, and healing took place slowly over a period of months, resulting in a deforming scar. A chest radiograph showed a nodule in the right lower lobe and other small nodular formations in the left lower lobe. A biopsy of the left axillary mass showed "anaplastic carcinoma with large cell features and suggestive glandular features infiltrating the lymphoid tissue."

After admission to the hospital, the patient underwent a number of diagnostic tests. A computed tomographic (CT) scan of the chest revealed a mass in the right lower lobe. A biopsy of the scar tissue in the left breast resulted in the diagnosis of infiltrating ductal carcinoma, scirrhous type (Fig 1). An assay for estrogen receptor proteins was negative. A bone scan showed increased uptake at the level of the thoracolumbar spine, ribs, femurs, and pelvis. A liver scan and a complete gastrointestinal work-up disclosed no abnormalities.

The patient received a combination chemotherapy regimen on a monthly basis. Regression of the presumed metastatic lesions in the lungs was noted within six months. Only minimal progression of the osseous metastasis was documented one year later.

Discussion. The relationship between trauma and breast carcinoma has not been clarified, and most authors are skeptical about trauma as a possible causative mechanism.[6,8-10] In 1955, Treves et al[9] reported that 12% of 146 male patients with breast carcinoma were able to give a history of antecedent trauma. The trauma varied with respect to type, frequency, and time of occurrence prior to breast symptoms (from one day to 44 years). The authors concluded that, in most cases, the traumatic accident merely called attention to a pre-existing lesion, thus casting doubt on any genuine etiologic relationship.[9]

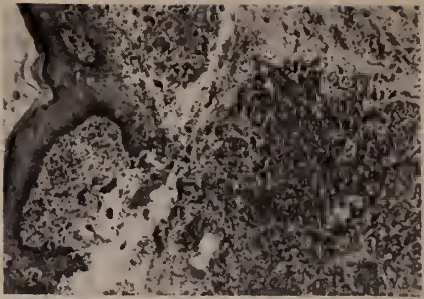


FIGURE 1. Microphotograph showing ductal carcinoma infiltrating the skin (hematoxylin-eosin stain; original magnification × 200).

In 1963, Schottenfeld et al[10] interviewed 53 male patients with breast carcinoma and observed that 11.3% recalled trauma to the breast within weeks or years prior to the time of diagnosis. These authors noticed that the traumatic episodes were isolated events rather than repeated insults associated with a specific occupational activity. The authors concluded, based on Treves's opinion, that the trauma caused a patient to notice an existing lesion.[10] In his review of breast cancer in men, Crichlow[4] is rather vague about the existence of this etiologic link, and he does not come to any specific conclusion.[4] Haagensen,[6] in his 1971 edition of *Diseases of the Breast*, also avoids precise comments on the subject and, in his review of the relationship between trauma and breast carcinoma in women, essentially denies the existence of such a causative factor.

In recent years, review articles published in major medical journals have almost completely ignored the possibility of an association between trauma and breast carcinoma in men.

In this patient, the time sequence of the trauma followed by cancer of the breast is of interest. Whether the previous trauma is causal or coincidental is not obvious. More extensive series of cases will be needed in order to clarify this issue.

MARIO C. RAVIGLIONE, MD
PETER E. GRAHAM, MD
Department of Medicine
Cabrini Medical Center
227 E 19th St
New York, NY 10003

Acknowledgment. The authors thank Dr Angelo Taranta, Director of Medicine, for his encouragement and constructive advice, and Dr Maria T. Sabatini, Associate Director of the Department of Pathology, for reviewing the histologic slides.

1. Mabuchi K, Bross DS, Kessler II: Risk factors for male breast cancer. *JNCI* 1985; 74:371–375.
2. Erlichman C, Murphy KC, Elhakim T: Male breast cancer: A 13-year review of 89 patients. *J Clin Oncol* 1984; 2:903–909.
3. Bafava S, Reyes CV, Choudhury AM: Male breast carcinoma: An updated experience at a Veterans Administration Hospital and review of the literature. *J Surg Oncol* 1983; 24:41–45.
4. Crichlow RW: Breast cancer in men. *Semin Oncol* 1974; 1:145–152.
5. Miller AB, Bulbrook RD: The epidemiology and etiology of breast cancer. *N Engl J Med* 1980; 303:1246–1248.
6. Haagensen CD: Carcinoma of the male breast, in Haagensen CD (ed): *Diseases of the Breast*. Philadelphia, WB Saunders Co, 1971, pp 779–792.
7. Cohen R, Schauer PK: Male breast cancer following repeated fluoroscopy. *Am J Med* 1984; 76:929–930.
8. Spence RA, MacKenzie G, Anderson JR, et al: Long-term survival following cancer of the male breast in Northern Ireland. A report of 81 cases. *Cancer* 1985; 55:648–652.
9. Treves N, Holleb AI: Cancer of the male breast. Report of 146 cases. *Cancer* 1955; 8:1239–1250.
10. Schottenfeld D, Lilienfeld AM, Diamond H: Some observations on the epidemiology of breast cancer among males. *Amer J Public Health* 1963; 53:890–897.

FROM THE LIBRARY

CANCER OF THE BREAST

We have studied the case histories on the 180 malignant tumors of the breast admitted to the Strong Memorial and the Rochester Municipal Hospitals from 1926 to 1934. Radical operation was performed on eighty of these patients; palliative operation on fourteen. . . .

The age incidence for malignant tumors of the breast follows in general that for malignancy elsewhere. The majority of patients fall in the 40 to 70 age period. . . . In our cases, there were eight patients with cancer of the breast who were under 40 years of age. . . There were also three above 70. . . .

The male sex is by no means immune to tumors of the breast. In our group, there were two patients with cancer, both of whom also had syphilis; and nine benign tumors, the commonest of which was fibroadenoma, five times. Cancer, in the male in our experience, here and elsewhere, is always a serious condition. It should be relatively easy to find these tumors early but the spread seems to be rapid even when the tumor is small.

In regard to etiology we will not enter into a discussion of the factors supposed to have a bearing such as lactation, stagnation, and so forth. We have been much interested, however, in the frequency with which trauma has been mentioned by our patients with breast tumors. Trauma to the breast cannot be very uncommon in women. The breast, struck against some object with sufficient violence to be recalled, was a possible cause for the swelling in fifteen cases of malignant tumors; and in eight cases of benign growths. The relation of trauma to breast cancer only assumes etiologic importance from the fact that it may have a medicolegal bearing. In two of our patients with malignant breast tumors, the claim was made that the breast was injured when the patient was thrown against the seat of a trolley car.

No student of the cancer problem today assumes a single etiologic factor to be operating in any neoplastic disease. There is always the hereditary predisposition, about the mechanism of which even the geneticists are not in agreement. And there are time factors and repeated irritation factors, misplaced cell rests, endocrine imbalances, and so forth, all of which may have a bearing.

In order to establish that a single trauma has even a contributory relationship it is necessary to prove, *first*, that a sufficiently severe trauma actually occurred (one patient said that she had bleeding from the nipple following trauma); *second*, that the breast was normal before or immediately at the time of the accident; *third*, that the localization of the neoplasm be reasonably close to the injured area; *fourth*, that a proper time interval elapse between the time of the accident and the development of the new growth; *fifth*, that the histology of the tumor conforms to the time relationship, i.e., a slow growing tumor which appears in a very short time after the injury or a rapidly growing tumor which appears after a long time would not seem logically to have any connection with the trauma. Furthermore, a tumor such as a dermoid cyst would at once be recognized as a congenital defect.

It is obviously almost impossible to fulfill these conditions in any given case. Experimentally, also, it has never been possible to produce a cancer by a single trauma. There is even evidence that single traumas may favorably affect a growth by damaging the blood supply but any such supposed advantage is more than offset by the danger of metastasis. It has been experimentally proved that rough handling or massage of malignant tumors is followed by generalization in a high percentage of cases. . . .

JOHN J. MORTON, MD
SAMUEL J. STABINS, MD
(*NY State J Med* 1935; 35:1137–1142)

PRINCIPLES OF FAMILY SYSTEMS IN FAMILY MEDICINE

Edited by Sergio Henao, MD, and Nellie P. Grose, MD. 438 pp. New York, Brunner/Mazel Publishers, 1985. $40.00 (hardcover)

A refreshing book has finally been written about a major principle in family practice. The appropriate role of the family in health care has been written about for a long time and has resulted in misunderstanding and uninformed arguments about who is the patient—the individual or the family. This book tracks the history of family practice in its early sections, clarifies the role of the family in illness and health care, and then gives examples of how specific medical problems are mediated by the family environment (ie, substance abuse, psychoses, chronic illness). The book ends appropriately with a discussion of the medicolegal implications of family involvement in health care.

The editors, Sergio Henao, MD, and Nellie P. Grose, MD, demonstrate the value of earnest collaboration between two capable people whose fields of expertise complement each other. In addition, they have collected a group of authors who represent all ranges of family practice as well as related fields concerned with family care. The development of a family system method of health care with the individual as the unit of care is amply clarified. The development of a change from the traditional physician-patient dyad to a triad of patient-family-physician is the precept that the specialty of family practice has been contributing to medical education.

Early chapters in this book are written with great clarity and little overlap. A clear understanding of the use of family systems in health care is developed and this understanding would be of value to any practitioner or teacher of family practice as well as any other specialty. Chapter 5, entitled "The Strategic Approach," by John H. Weakland and Richard Fisch, pulls together the appropriate use of the family in the health care of the individual in such a way that any practitioner would recognize its value. Chapter 18, by Gabriel Smilkstein, outlines several ways to assess the family and the individual patient's role in the family. Perhaps the discussion of the value of home visits could have been further developed.

Certainly the biomedical model of health care currently taught in many medical schools and practiced by most physicians is limited. The role of the physician as a respected member of the community has declined and patient confidence in physicians has also fallen. A major reason for these changes is the classic biomedical model in which the medical profession ignores psychosocial factors in the patient's background and the effects of these factors on the patient's illness and response to illness. Recognition of the true biopsychosocial systems of patient illness will certainly improve the relationship between the physician and the patient, as well as raise the understanding of the physician as the patient's advocate. Knowledge about family systems is an important segment of knowledge about the biopsychosocial individual who is seeking health care.

This book is written clearly in terms of both content and style. The individual chapters are frequently punctuated by subheadings that give the reader a true picture of the content at a glance as well as make the text easy and enjoyable reading. Other publications dealing with the family unit in health care are not as successful in demonstrating rational definitions, clear objectives, and examples that readily augment the reading. The reader who believes in the important role of the family in an individual's health is refreshed and energized by this book.

Deficiencies of this thoughtfully prepared work are few. Examples occasionally become mildly tedious as the same points are reiterated several times. But, in general, the examples do serve to clarify the text. The need to expand the classical social history obtained when evaluating the patient is well documented. A limited selection of ways to do this is described, but how these methods can really fit into the busy practitioner's efforts and which single method or combination of methods is best is unclear. Even more unclear is how to train medical students early in their education to incorporate family systems into patient evaluations. Probably the best way to manage this would be to convince the medical establishment of the validity of the family system approach and then to develop teaching modalities that clearly demonstrate these precepts to the students. What a remarkable change in the public's perception of doctors would occur with an emphasis placed on family systems in the medical schools!

RICHARD SADOVSKY, MD
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

THE HOME HEALTH CARE SOLUTION

By Janet Zhun Nasif. 433 pp. New York, Harper & Row, 1985. $9.95 (paperback)

Since the imposition of a prospective payment system using DRGs came into effect on October 1, 1983, hospital patients are being discharged "quicker and sicker," according to a US Government Accounting Office study. That circumstance led to a tremendous growth in the home health care industry. Creation of hospital-based home health care units has been one of the responses to the need for more services to patients requiring a higher level of care at home.

Offering a complete consumer guide, Nasif's book directly entices the potential reader on the cover. Under the title appear the following phrases: home health care resolves to help you get well faster; to cut hospital costs; to shorten hospital stays; to avoid nursing homes; to choose the best caregivers; to get high-tech care at home; and, as an added bonus, to discover tax savers. Despite these words—probably thought up by a promotions expert—Nasif does deliver in this compact and informative book.

Using case examples, the book begins with the basics about home health care, or preferably home care, and points out why there is no place like home and the rationale for the home care revolution. Next, the reader is given the nuts and bolts about home care including the types of services provided, the agencies in the field, the personnel available, and even how to hire your own home care team. Since paying for home care is a huge tangle of insurance and private payment, Nasif details the costs for each type of service and the alternatives. She describes the use of HMOs, PPOs, S/HMOs, and home equity conversions along with tax tips and 24 dollar saving suggestions. A section follows on the special services that may be secured at home, including total enteral or parenteral nutrition, chemotherapy, antibiotic therapy, home dialysis, apnea monitoring, and respiratory therapy. Of course, the more common durable medical equipment such as wheelchairs, hospital beds, walkers, and home oxygen is also covered. Emergency response systems that allow patients to telephone for help by merely pressing a remote control device kept on their person are the answer to questions about an immediate emergency situation at home.

The author elaborates on the use of hospice care for the terminally ill, which is another growing movement in the health care field. A quality of care chapter deals with the issues of fraud and abuse, malpractice, the evaluation of providers, and what to do before an individual enters into a home care situation. This chapter also advises consumers how to register and resolve their complaints. Alternatives to home care are also detailed including adult day care, adult foster care, and life care communities. A variety of board and care schemes describe group homes, congregate living, domiciliary arrangements, and shared housing.

Finally, Nasif presents a resource guide with checklists and directories that should be most helpful for consumers. There is a self-assessment checklist for determining the level of aid required relative to activities of daily living, nursing care, skilled nursing care, and social services. Another checklist helps to choose a home care agency. An insurance worksheet guides people through their insurance coverage. If durable medical equipment is required, a checklist provides help in evaluating providers of such equipment. More than 50 possible home care tax deductions are listed on still another guide. In addition, Nasif provides a specific list of organizations, addresses, and even telephone numbers for state home care associations, state aging units, state hospice agencies, self-help clearinghouses, and related home care organizations.

While there is an index, there are no references other than those cited in the text of the book. However, this book is intended for consumers and references may not be necessary. In any event, the book is replete with organization addresses and telephone numbers for those seeking additional information.

Health care providers who wish to supply their patients with a readable, comprehensive guide to home health care will not go wrong in recommending this paperback. Patients and their families who are considering home care will thank you for your suggestion.

ALLEN D. SPIEGEL, PhD
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

PSYCHOBIOLOGICAL ASPECTS OF ALLERGIC DISORDERS

Edited by Stuart H. Young, MD, James M. Rubin, MD, and Harlan R. Daman, MD. 383 pp, illustrated. New York, Praeger Publishers, Inc, 1986. $37.95 (hardcover)

The authors' stated goal in compiling this book is to "provide the reader with an up-to-date overview of the exciting new approaches that are now emerging in the study and treatment of allergic disease." The background and areas of expertise of the contributors certainly qualify them for this task.

The book is organized so that it may be used as a reference text, and the bibliographies are extensive. I suspect that this compendium of sources is unique for this field and potentially useful in a practical way.

Section I, describing allergic diseases for the nonallergist, is well done and clearly presented. It includes a discussion of the semantics involved in the terms describing the respiratory disease spectrum. The descriptions of the effects and side effects of various treatment modalities are excellent.

Chapter 5 of Section II is an especially practical discussion of the use of drugs in pregnancy, as well as of the hormonal effects and psychologic effects in relation to drug actions.

With many scientifically unfounded practices abounding in the fields of "clinical ecology" and in allergy itself, the chapter by Drs Selner and Staudenmayers, "The Relationship of the Environment and Food to Allergic and Psychiatric Illness," should be required reading for all primary health care physicians. It gives a rational basis for judging the scientific competence of the practitioner of ecology and allergy. The discussion of reactions to food and to chemicals helps set a foundation for a sensible evaluation of the problem. Reference to questionable procedures and techniques is made.

Chapter 7, "Central Nervous System Effects of Drugs Used in Treating Atopic Illness and Asthma," will be of particular interest to the primary care physician who does not frequently prescribe such drugs. Chapters 8 and 9 put into good perspective the potential place of behavioral therapy and psychoanalytic treatment in these diseases.

Part IV, "Psychobiologic Interventions in Allergic Disorders," is an interesting discussion of various methods of stress management in relation to the symptomatology of allergic disorders.

The final chapter puts psychoneuroimmunology in proper perspective and discusses briefly its potential for future management. Overall the book is structured soundly, and the discussions and information in each section are pertinent to the topic.

It should be stressed that this is not a textbook. It is a narrative of the complexity of interrelationships of the immune system, the environment, and the nervous system in the production of allergic disorders. It adds a full dimension to the control of these disorders through control of the physical and emotional symptoms with avoidance, drugs, and modes of psychotherapy. The editors have compiled an interesting, catholic perspective on allergic disorders which puts present beliefs, fallacies, and practices in sensible order for the nonallergist.

JOSEPH F. KELLEY, MD
The Cleveland Clinic Foundation
Cleveland, Ohio 44106

AN ANTHOLOGY OF PLASTIC SURGERY

Edited by Harry Hayes, Jr, MD. 338 pp, illustrated. Rockville, Md, Aspen Publishers, Inc, 1986. $55.00 (hardcover)

In the preface of this book, the author suggests that the reader ". . . pull up an easy chair, light a fire, pour . . . a neat Scotch . . . and turn the pages slowly," and that the reward will be ". . . finding a new idea, or a new slant on an old idea." Written in a comfortable narrative style, this volume consists of 197 vignettes, each of 1–2 pages in length, divided among 15 chapters. The book is laced with illustrations. Chapter headings range from "Historical References" to "Illustrious Patients," and end with "From the Cartoonist's Pen," while individual vignette titles include "Hand injury and medieval law," "The first blepharoplasty (1583)," and "The tragedy of Valentino's brother's nose." The vignettes are abstracted from a wide variety of sources, with each reference listed at the end of the vignette. There are also separate subject and author indexes at the end of the book.

This book should probably be read exactly as the author suggests. It can be read cover-to-cover, or at random. Either way, the reader is likely to smile frequently as he or she encounters the legacies of the people who shaped modern plastic surgery, and sees that, clearly, little in plastic surgery is completely new. Concepts such as wound closure strips, skin staples, tissue expansion, reduction and fixation of maxillary fractures, and pedicle flaps were all described before 1900, sometimes centuries before, and their origins are recounted here. Not all of the history of plastic surgery is laudable, however. A chapter entitled "Quackery and Nostrums" chronicles a series of charlatans, and advertisements that promised to eliminate wrinkles and fat and to develop the bust. A vignette on patents details devices for bust-development (based on vacuum suction cups attached to the breasts), and hair-growing (in which male baldness is assumed to be due to a lack of ventilation in men's hats!). There are also chapters on plastic surgery in painting, sculpture, poetry, and literature.

The book is entertaining and fascinating, with humor sprinkled throughout.

ALAN M. ENGLER, MD
800B Fifth Avenue
New York, NY 10021

CARDIOLOGY 1986

Edited by William C. Roberts, MD. 468 pp, illustrated. New York, Yorke Medical Books, 1986. $55.00 (hardcover)

The sixth annual review of cardiology literature follows the same format as the previous reviews. Five leading cardiologists and two cardiovascular surgeons have reviewed nearly 800 articles published in 1985 from among 28 journals. The actual number of articles summarized by each reviewer is presented in the preface. Once again, Dr Roberts does yeoman service, reviewing 56% of the articles.

The major subjects into which the reviews are divided include all of the standard forms of heart disease and are similar to those topics covered in previous years: coronary artery disease; acute myocardial infarction and its consequences; arrhythmias, conduction disturbances, and cardiac arrest; systemic hypertension; valvular heart disease; myocardial heart disease; congenital heart disease; congestive heart failure; and miscellaneous topics. Yet, a comparison of subtitles shows the changing emphasis on various aspects of cardiac problems. For instance, such topics as fibrinogen and vasectomy are replaced by obesity, coffee drinking, alcohol drinking, estrogen, hemocystinuria, and prostaglandins and platelet inhibitors. New drugs appear under the topic of angina pectoris, such as molsidomine, nicardipine, nisolipine, captopril, coenzyme Q10, and amiodarone. Variant angina pectoris is now variant angina and/or coronary spasm. Prognostic indexes for acute myocardial infarction include initial ECG, ECG ST segment depression, LV ejection fraction, and infarct extension, instead of such topics as arrhythmia, LV function, exercise-induced ST segment elevation, and diabetes mellitus. Incidentally, these changing emphases are part of the fascinating area of cardiology and all of its ramifications. There is a larger section on percutaneous transluminal coronary angioplasty. The list of drugs used for ventricular arrhythmias continues to be long, suggesting that the ideal drug still has not appeared. The section on idiopathic dilated cardiomyopathy includes a review of its relation to acute myocarditis and a review of the correlation of myocardial ultrastructural findings to myocardial function.

The articles reviewed and the reviews themselves are of the highest caliber. The descriptions are clear and succinct and are frequently illustrated with graphs and tables. Once again, the cardiology annual serves as an excellent review of what has been going on in the field during the past year. The bibliography at the end of each chapter serves as an excellent starting point for the review of almost any subject relating to cardiac diseases.

In comparison with other cardiology annual reviews, *Cardiology 1986* stands out as a superior volume for anyone interested in the subject.

JOHN N. EDSON, MD
Long Island College Hospital
Brooklyn, NY 11201

PRINCIPLES OF AMBULATORY MEDICINE

Second Edition. Edited by Randol Barker, MD, John R. Burton, MD, and Philip D. Zieve, MD. 1,494 pp, illustrated. Baltimore, Md, Williams and Wilkins, 1986. $82.50 (hardcover)

Since the first edition in 1982, 300 pages have been added to this already large standard work on ambulatory medicine. The text is devoted to the adult aspects of ambulatory medicine and is, therefore, limited in the areas of obstetrics-gynecology, pediatrics, and ambulatory surgery. It is organized into 14 broad sections categorized by specialty and 102 chapters. Each chapter begins with a short outline of its thematic contents with appropriate page assignments. It is easy for the reader to grasp the structure of the chapter and access the particular area of interest.

The symptoms, signs, or problems of management of a certain clinical entity constitute the goal of each chapter. Established guidelines and protocols are presented, as are more controversial areas. Clinical methodologies have been consistently updated since the first edition. A clear succinct understanding of a section may be obtained by reading its chapter independent of the rest of the text.

Regrettably, the "Patient Experiences" statements present in the first edition are no longer utilized. Although they have in part been replaced by patient education and prevention sections, this humane characteristic of the book has been lost. On the other hand, however, useful graphs and charts are plentiful throughout the book. The pathophysiology of the various clinical diseases is, for the most part, concisely presented, and unlike the first edition, the pharmacology sections are adequately covered. Finally, the references are current and of two types: general and specific. The former are accompanied by a short, useful abstract.

Like the first edition, the second suffers from one major oversight. None of the contributors, many of whom are outstanding clinicians, are full-time family physicians. The first chapter clearly notes that 33% of all office visits in 1981 were made to general and family practice physicians while 13% and 11% were made to internists and pediatricians, respectively. Although the oversight does not lessen the text's scientific merit, it does reduce its overall credibility.

The editors have done a skilled job in synthesizing the board area of ambulatory medicine into meaningful, readable units. Although its size prevents easy bedside access and despite its significant cost, it is a worthwhile addition to the library of any person regularly involved in this field of medical care. Institutions dealing with primary care should also have a copy of this resource. Generalists and family physicians will have to supplement the text with additional works in surgery, pediatrics, and obstetrics and gynecology.

RICHARD B. BIRRER, MD
Consulting Editor

BLOOD DISORDERS. THE FACTS

By Sheila T. Callender. 164 pp, illustrated. New York, Oxford University Press, 1985. $13.95 (hardcover)

This little book is part of a series of approximately 25 similar volumes addressed to the lay public on various medical problems and published by Oxford University Press. This particular volume differs from the other books in the series in that it covers a broad range of blood diseases. The author is a distinguished hematologist and former clinical reader in internal medicine at the John Radcliff Hospital of the University of Oxford.

The purpose of this book as stated in the preface is to provide patients with "some understanding of the functions of the various components of the blood and to outline some of the disorders arising from disturbances in normal function." Essentially this is a short textbook of hematology and includes figures, plates, diagrams, and tables which would not be out of place in standard textbooks of hematology. In 18 chapters and an epilogue the author ranges over almost the entire span of topics in the discipline of clinical hematology, including tests for blood disorders, symptoms of anemia, iron, folate and B12 deficiency, hemoglobinopathies, leukemia, bone marrow transplantation, myeloma, lymphomas, and bleeding disorders. A glossary of medical terms and an index are included.

For the intelligent, uninitiated reader this book provides a readable, authoritative introduction to almost all the problems encountered in the practice of clinical hematology. In this it is a unique work in my experience, but I must emphasize that this book is aimed at a well-educated audience. It is written at a level of sophistication not far below that of the average article in the *Scientific American*. Thus it will appeal to and be appropriate for a minority of patients. Most others, however, will find it rough going. On the other hand, it might prove suitable for courses for high school science students, college undergraduates, or ancillary medical personnel.

RONALD F. RIEDER, MD
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

Battling the effects of Parkinson disease

The majority of patients with Parkinson disease will experience mental changes, including hallucinations, nightmares, and periods of confusion, as well as physical disability that can lead to falling. Dr George Paulson and colleagues have examined the possible causes of these mental and physical impairments (*Geriatrics* 1986; 41(8): 59-62, 67).

From the survey of 211 patients with Parkinson disease, the authors concluded that patients attribute the mental changes to antiparkinsonian medications (57% of patients surveyed), whereas the tendency to fall is believed to result from the effects of the disease itself (90%). The most common mental disturbance reported was confusion (34%). Sleep and dream disturbances, including insomnia, nightmares, and hallucinations, are possible side effects of dopamine and levodopa, two common antiparkinsonian drugs.

Falling was a common occurrence among 53% of patients. The authors reviewed possible causes of falling, including dyskinesia caused by medication and parkinsonian effects such as rigidity, loss of some visual and postural ability, decreased sensory input, and disturbances in balance. Forty-eight percent of patients who were prone to falling found regular exercise beneficial in reducing the tendency to fall and in avoiding injuries.

The authors emphasize the importance of dosage adjustment and regular follow-up for patients with Parkinson disease. They conclude that treatments such as physical therapy and family counseling may be more effective than neurologic or psychiatric approaches to overcoming the mental and physical impairments that often accompany Parkinson disease.

Elimination of tobacco use is focus of New York Academy of Medicine report

In a recent report entitled *Tobacco and Health*, the Committee on Public Health of the New York Academy of Medicine calls for a nationwide campaign to eliminate tobacco use. Drawing on information compiled by the surgeon general, the World Health Organization, the US Office of Technology Assessment, and other health authorities, the Academy concludes that the damaging effects of tobacco use on Americans' health and the related financial toll are cause for immediate action. The Academy is recommending a multifaceted approach to the problem, aimed at reducing both the demand for and the availability of tobacco products—including cigarettes and smokeless tobacco.

To lessen the demand for tobacco products, the Academy proposes a consumer education program that would alert the public to the adverse health effects associated with tobacco use and to the addictive quality of tobacco. This initiative would include educational programs in the schools, a multimedia advertising campaign against tobacco products using established role models, improved warning labels on tobacco packaging making them more visible and specifying the potential health risks, and government-sponsored programs to help people stop smoking.

To diminish the supply of tobacco products, the Academy suggests banning tobacco advertising, the distribution of free samples, and the sale of tobacco products to minors; increasing excise taxes on tobacco; restricting smoking in public and in the workplace; and diverting government funds used to subsidize the tobacco industry to assist farmers in giving up tobacco farming.

Recognizing the health hazards associated with smoking, passive smoking, and smokeless tobacco—including cancer, respiratory diseases, and heart disease—the New York Academy of Medicine is seeking legislative action to eliminate tobacco use and availability.

Gentler treatment reduces risk of chronic lung disease in premature infants

A two-year, multicenter study has shown that less aggressive therapy for breathing disorders in low birth weight newborns may prevent the development of chronic lung disease. Premature babies frequently experience respiratory distress due to underdeveloped lungs. This condition can lead to chronic lung disease, in which the infant requires oxygen therapy for several weeks after birth and may be more susceptible to respiratory infections in the future.

Typical treatment for newborns with breathing problems involves insertion of an endotracheal tube to facilitate breathing, artificial ventilation with a respirator, and the use of muscle relaxants to prevent the baby from resisting the therapy. Doctors in the perinatology division of the Presbyterian Hospital/Columbia-Presbyterian Medical Center in New York have modified this approach, with promising results. The study, published in *Pediatrics* (1987; 79:26-30), found that premature infants treated at Presbyterian were less likely to require oxygen therapy at one month of age.

The treatment approach adopted at Presbyterian includes early intervention with respiratory assistance in the form of warm, moist air introduced at a continuous rate through nasal prongs, avoidance of intubation and hyperventilation associated with artificial ventilatory support, and elimination of muscle relaxant therapy. In addition, the authors of the study noted that a single anesthesiologist oversees the care of premature infants at Presbyterian, ensuring a singular approach to care.

The authors could not specify which aspects of treatment at Presbyterian were responsible for the improved patient outcome. They proposed further research into the beneficial effects—including less trauma and less risk of infection—and possible adverse effects of a less aggressive approach to treating breathing difficulties in premature infants.

Researchers identify animal model for hypercholesterolemia and atherosclerosis

Researchers at the University of Wisconsin-Madison have found an association between hereditary mutations

---

NEWS BRIEFS are compiled and written by Vicki Glaser.

in plasma lipoproteins and the development of atherosclerosis in a strain of pigs. This finding supports the role of genetics as a predisposing factor in atherosclerosis and premature death due to coronary heart disease.

Rapacz et al (*Science* 1986; 234:1573-1577) identified three markers in genetic material that either codes for the protein portion of low density lipoprotein (LDL)—the main carrier of cholesterol in the blood—or is situated near genes that control plasma cholesterol levels. Pigs with these three markers had two to three times higher plasma cholesterol levels than other pig strains, despite being fed a low-fat, cholesterol-free diet. The increased levels were mainly due to the presence of more LDL.

The mutations in these pigs differ from those associated with familial hypercholesterolemia (FH)—a genetic disorder associated with premature atherosclerosis and elevated cholesterol levels. The mutation in FH occurs in a gene that codes for an LDL receptor, thus interfering with the catabolism of LDL. Rapacz et al conclude that mutations in the apolipoproteins themselves caused the hypercholesterolemia and accelerated atherosclerosis seen in these pigs.

Histologic analysis of hearts from several of the mutant pigs revealed that the coronary atherosclerosis was similar to that seen in humans. The authors noted that "although pigs often develop atherosclerosis between 6 and 8 years of age, the mutants deviated markedly from the normal animals in developing severe disease within 2 years." The mutant pigs typically survived less than one third of their normal life expectancy.

Public Health Research Institute commemorates 45th anniversary

The Public Health Research Institute (PHRI; New York, NY), a nonprofit organization that pursues basic research in infectious diseases, celebrated its 45th anniversary in December. In the past, research by PHRI scientists has contributed to diagnostic and therapeutic breakthroughs in the fight against diseases such as poliomyelitis, cancer, influenza, and diptheria. For example, in 1983, a PHRI researcher discovered the gene responsible for toxic shock syndrome. The institute's research program in AIDS focuses on the structure and function of proteins on the surface of the AIDS virus, as part of a larger project aimed at understanding the mechanism of infection of retroviruses.

PHRI—established by Mayor Fiorello H. LaGuardia as part of the New York City Department of Health, and originally called Health Research, Inc—maintains a staff of 22 principal investigators. In addition to funding from New York City, the National Institutes of Health, and other agencies, PHRI will begin to seek financial support from the private sector.

Nobel laureates attend medical update in Trinidad

The Department of Medicine of the University of the West Indies held its tenth annual update of important topics in medicine in Port of Spain on January 10-11, 1987. Organized by Professor Courtenay Bartholomew, the presentations at the update covered both recent scientific research findings and practical applications of newer diagnostic and therapeutic modalities. The update was opened by Dr Emanuel Hosein, Minister of Health of Trinidad and Tobago, and attended by the country's president, Ellis Clarke. The meeting attracted over 300 physicians from the Caribbean, Canada, and the United States.

Sir Christopher Booth, president of the British Medical Association, spoke on the role of general practitioners in research, pointing out their major contributions over many years. Professor Courtenay Bartholomew of the University of the West Indies, who is well known for his pioneering studies of the HTLV-I leukemia virus, presented recent findings on the mode of transmission of this virus. Dr Robert Gallo of the National Cancer Institute, National Institutes of Health, well known for his isolation of the causative agent of the acquired immunodeficiency syndrome (AIDS), presented the results of recent research in his laboratory.

Dr Carleton Gadjusek, Nobel laureate and currently with the Laboratory of Central Nervous System Studies, National Institutes of Health, described several high incidence foci of neurologic disease in the Pacific, including Kuru and amyotrophic lateral sclerosis, and discussed their pathogenesis. His presentation was the Dr Lennox Pawan Lecture, attended by the president of Trinidad and Tobago.

Dr Baruch Blumberg, a Nobel laureate well known for his pioneering work on hepatitis B, discussed the relationship between hepatitis B and hepatoma and the need for mass immunization programs among very young children in high prevalence areas to prevent the disease and its complications.

Dr Graham Hughes of St Thomas Hospital in London discussed recent research advances in rheumatology and Dr Richard Hunt gave an update on peptic ulcer disease management. Dr Ram Mahabir of the San Fernando Hospital, Trinidad, spoke on proteinuria and Dr Albert Persaud of the Caura Chest Hospital, Trinidad, spoke on the management of asthma. Professor Soad Tobaqchali of St Bartholomew's Hospital, London, spoke on antibiotics and the gut.

Continuing medical education, which is not institutionalized in Caribbean countries, was the subject of an extensive panel discussion involving physicians from Trinidad, Barbados, Great Britain, and the United States. Information for this panel discussion was provided by the Medical Society of the State of New York.

The tenth annual update of important topics in medicine was a unique meeting in that it simultaneously served as a forum for the presentation of both recent and in some cases unpublished medical research findings and of practical information for general practitioners. The meeting was closed with comments from Dr Theo Poon-King of the University of the West Indies.

Guidelines for authors

Originality

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1983; 83:1089–1094). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

Preparation

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

Categories

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

Figures

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

References

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith[2] reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al*. Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83: 925–927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337–338.

Review

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, P.O. Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

Added Control For America's Coughs

The expectorant action of guaifenesin plus the Advantage of Codeine!

Robitussin A-C®Ⓒ

Robitussin and codeine. Each 5 ml (*1 teaspoonful*) contains: Guaifenesin, USP, 100 mg; Codeine Phosphate, USP, 10 mg (*warning: may be habit forming*); alcohol, 3.5 percent; in a palatable, aromatic syrup

A·H·ROBINS

Pharmaceutical Division, Richmond, Virginia 23261-6609

©A.H. Robins Company 1985

PHYSICIANS RECIPROCAL INSURERS

Offering New York State Physicians the Broadest Claims-Made Professional Liability Coverage

OUR UNIQUE PROGRAM INCLUDES...

■ SELECTIVE UNDERWRITING	A preferred risk group
■ STATEWIDE DISTRICT REPRESENTATIVES	Knowledgeable and sensitive to physicians' concerns
■ GROUP PRACTICE COVERAGE	Additional layer of protection at nominal cost
■ NEW DOCTOR & PART-TIME RATES	Aware of the special needs and unique situations of our fellow professionals
■ AND MORE....	

FOR ADDITIONAL INFORMATION CONTACT

PHYSICIANS RECIPROCAL INSURERS

111 East Shore Road, Manhasset, New York 11030
212/936-3050 • 516/365-6690 • TOLL FREE 800/632-6040

(*continued from p 7A*)

ROCHESTER

Apr 4. **Tenth Annual Spring Seminar in Anesthesiology.** 6 Cat 1 Credits. Douglas Auditorium—Genesee Hospital. Contact: Dawn Rivera, Department of Anesthesiology, The Genesee Hospital, 224 Alexander St, Rochester, NY 14607. Tel: (716) 263-5265.

CALIFORNIA

Apr 2–4. **Advances and Controversies in Clinical Pediatrics.** 16 Cat 1 Credits. San Francisco. *Apr 9–11.* **Thoracic Imaging Update.** 13 Cat 1 Credits, Monterey. *Apr 10–12.* **OB/GYN and Abdominal Sonography: Update '87.** 14½ Cat 1 Credits. Hyatt Union Square Hotel, San Francisco. *Apr 30–May 2.* **Postgraduate Course in General Surgery.** 15 Cat 1 Credits, San Francisco. Contact: University of California, Extended Programs in Medical Education, Room U-569, San Francisco, CA 94143-00742. Tel: (415) 476-5808.

Apr 25–26. **Transurethral Ureteroscopy.** 13 Cat 1 Credits. LaJolla Marriott, San Diego. Info: Office of Continuing Medical Education, M-017, UC San Diego School of Medicine, LaJolla, CA 92093. Tel: (619) 534-3940.

HAWAII

Apr 25–May 1. **Critical Care Medicine.** 20 Cat 1 Credits. Intercontinental Hotel, Maui. Info: University of Colorado School of Medicine, Office of Continuing Medical Education, 4200 East Ninth Ave, Box C-295, Denver, CO 80262. Tel: (303) 394-5195.

KENTUCKY

Apr 10–11. **Hypertension: New Directions.** Hyatt Regency Hotel, Lexington. *Apr 10–12.* **Contact Lens Workshop.** Hyatt Regency Hotel, Lexington. *Apr 24–25.* **Contemporary Pediatrics for the Practicing Physician.** Hyatt Regency Hotel, Lexington. Contact: Joy Greene, Continuing Medical Education, 132 College of Medicine Office Building, University of Kentucky, Lexington, KY 50536-0086. Tel: (606) 233-5161.

LOUISIANA

Apr 2–5. **American College of Physicians 68th Annual Session.** New Orleans Convention Center. Contact: American College of Physicians, 4200 Pine St, Philadelphia, PA 19104. Tel: (800) 523-1546.

Apr 26–May 2. **36th International Seminar on Leprosy.** National Hansen's Disease Center, Carville. Info: Dr W. Felton Ross, American Leprosy Missions, One Broadway, Elmwood Park, NJ 07407. Tel: (201) 794-8650.

MASSACHUSETTS

Apr 13–17. **Postgraduate Course in Gynecologic and Obstetric Pathology with Clinical Correlation.** 35 Cat 1 Credits, Boston. Contact: Department of Continuing Education, Harvard Medical School, 25 Shattuck St, Boston, MA 02115. Tel: (617) 732-1526.

Apr 24–25. **The Terminally Ill Patient: Psychological, Social, Legal, and Ethical Issues.** Copley Plaza Hotel, Boston. Contact: Harvard Medical School, Department of Continuing Education, Boston, MA 02115. Tel: (617) 732-1525.

(*continued on p 14A*)

Photography by Ken Nahoum

AIDS is a complex disease, assaulting the psyche as well as the body. The Spellman Center for the Treatment of Persons With AIDS treats both. A comprehensive medical facility, Spellman is devoted to the care of those suffering from Acquired Immune Deficiency Syndrome.

Respect for individual dignity guides the Spellman program. Staffed by trained personnel dedicated to working with AIDS patients, the Center includes family and friends in the management of the patient's health crisis. Spellman also operates a 24-hour information hotline at 1-800-433-AIDS.

For criteria on admission and referrals, please contact the Medical Director, at 1-212-307-0735. Spellman is a division of St. Clare's Hospital and Health Center, designated the first AIDS treatment center by New York State.

The Spellman Center
for the Treatment of Persons With AIDS
St. Clare's Hospital and Health Center

SC

415 W 51st St
New York, NY 10019

An Affiliate of New York Medical College

An AZT Clinical Site

Medical experts,
providers of care

June 13, 1987

7TH ANNUAL ADVANCES IN GASTROENTEROLOGY

Bally's Park Place Hotel
Atlantic City, New Jersey

Sponsored by the University of Pennsylvania Gastrointestinal Department and the Continuing Medical Education Department of the Underwood Memorial Hospital,
Woodbury, New Jersey

Accreditation: Category 1 credit offered
Information: Registration Supervisor
SLACK Incorporated
6900 Grove Road
Thorofare, New Jersey
08086-9447
(609) 848-1000

AIDS SYMPOSIUM

Mt. Sinai Medical Center, New York City
Saturday, April 4, 1987 9 am—6 pm

Peter Herman, M.D.
Course Director

David M. Simpson, M.D.
Associate Course Director

Topics Included:

1. **Infectious Diseases**
2. **Epidemiology**
3. **Virology**
4. **Neurology**
5. **Mental Health**
6. **Oncology**
7. **Pediatrics**
8. **Therapeutics**

Guest Speakers from NIH, Mass. General Hospital, University of California, CDC Georgia, Faculty of Mt. Sinai Medical Center and Salpetriere Hospital, Paris, France.

Fees: $75 ($25 for Residents, Interns and Medical Students)

Credit Hours: 8

Send Check to: Peter Herman, M.D.
AIDS SYMPOSIUM
Mt. Sinai Medical Center
P.O. Box 1282
New York, New York 10029

(*continued from p 12A*)

NEBRASKA

Apr 27–May 8. **Family Practice Review—A Comprehensive Update.** Omaha. Contact: Marge Adey, University of Nebraska Medical Center, Center for Continuing Education, 42nd and Dewey Ave, Omaha, NE 68105. Tel: (402) 559-4152.

NEVADA

Apr 27–30. **35th Annual Clinical Meeting.** 17 Cat 1 Credits. Las Vegas. Info: American College of Gynecologists Registrar, 600 Maryland Ave, SW, Suite 300 East, Washington, DC 20024-2588. Tel: 1-800-533-8811.

PENNSYLVANIA

Apr 9–11. **Current Concepts in Vascular Surgery.** Adam's Mark Hotel, Philadelphia. Contact: Fay Zelle, Hahnemann University, Broad and Vine Streets, M.S. 623, Philadelphia, PA 19102. Tel: (215) 448-8263.

Apr 22–25. **Seventh Annual Conference on Evoked Potentials.** Philadelphia. Info: Ms F. DiBenedetto, Executive Secretary, American Society for Clinical Evoked Potentials, 445 West 50th St, New York, NY 10019. Tel: (212) 765-5809.

Apr 25–May 1. **American Occupational Health Conference.** Wyndham Franklin Plaza Hotel, Philadelphia Civic Center. Contact: American Occupational Medical Association, 2340 S. Arlington Heights Road, Arlington Heights, IL 60005. Tel: (312) 228-6850.

TEXAS

Apr 23–25. **Occupational Lung Disease.** The Houston Marriott Medical Center, Houston. Info: Vicki L. Forgac or Lila K. Lerner, Office of Continuing Education, Baylor College of Medicine, One Baylor Plaza, Houston, TX 77030. (713) 799-6020.

VIRGINIA

Apr 3–5. **Ophthalmologic Plastic Surgery, Orbital Disease, and Neuro-Oph-**

(*continued on p 16A*)

you're invited...

CLINICAL DECISION-MAKING IN GERIATRICS

A three-day course to be held at Mount Sinai Medical Center, New York City, April 6-8, 1987

Sponsored by the Department of Geriatrics and Adult Development, The Page and William Black Postgraduate School of Medicine of the Mount Sinai Medical Center. Course Directors: David T. Lowenthal, MD, PhD, and Barbara Katzeff, MD.

Monday, April 6: Lectures will be presented on general aspects of clinical geriatrics, patient evaluation, and special drug therapy considerations. Attention will be directed to the evaluation and treatment of peripheral vascular disease and problems such as ischemic heart disease, hypertension, dizziness and syncope, and stroke. The closing session will feature a special lecture on the Goteburg Longitudinal Study.

Tuesday, April 7: This segment will concentrate on musculoskeletal problems—pain, hip fracture, and osteoporosis; nutrition in the elderly; various infectious diseases; and use of antibiotics.

Wednesday, April 8: The final day will begin with lectures on the central nervous system—brain function, dementia, depression, and pharmacologic management of Alzheimer's disease.

Special attention will be given to the significant role played by allied health professionals. Experts will also delve into matters such as nursing home placement, legal issues, and financing.

The course will conclude with the annual Mount Sinai Medical Center Alzheimer's Disease Update.

Supported through a grant-in-aid by the Sandoz Pharmaceuticals Corporation.

For registration and further information, contact Kenneth Smith, PhD, at The Page and William Black Postgraduate School of Medicine. Phone: (212) 650-7991.

INST-387-1

Portfolio Management As Individual As You Are.

Whether your financial objective is maximum income or maximum growth, the Private Clients Group individually manages your portfolio for maximum results–profitable performance for you. Our dedicated investment research team and full business integration in support of the private investor make us unique. No one serves the individual investor better.

To discuss your portfolio of $500,000 or more, write or call Caroline M. Calder, Marketing Officer, Private Clients Group, 280 Park Avenue, New York, N.Y. 10017. Tel. 212/850-1473.

Bankers Trust Company
Private Clients Group
We Make Money For People Who Make Money

© 1986 Bankers Trust Company
Member FDIC

(*continued from p 14A*)

thalmology. The Williamsburg Hospitality House, Williamsburg. *Apr 10–12.* **5th MCV Symposium: New Trends in Anesthesia.** The Williamsburg Hilton, Williamsburg. *Apr 10–12.* **22nd Annual Pediatric Springfest.** The Williamsburg Hospitality House, Williamsburg. *Apr 23–25.* **23rd Annual Postgraduate Course in Radiology: The Chest.** The Richmond Marriott, Richmond. *Apr 24–26.* **9th Annual Conference on Emergency Medicine for the Primary Care Physician.** Fort Magruder Inn, Williamsburg. *Apr 24–26.* **7th Annual Clinical Concerns in Primary Care: Office Cardiology.** The Williamsburg Hospitality House, Williamsburg. Contact: Kathy Martin, Office of Continuing Medical Education, Medical College of Virginia, Box 48, MCV Station, Richmond, VA 23298-0001. Tel: (804) 786-0494.

AROUND THE WORLD

CANADA

Apr 21–24. **Prediction of Tumor Treatment Response.** Banff Springs Hotel, Banff, Alberta. Contact: Meg Keiser, American College of Radiology, 925 Chestnut St, Philadelphia, PA 19107. Tel: (215) 574-3187.

ISRAEL

Apr 10–24. **Medical Seminar.** 25 Cat 1 Credits. Tel Aviv, Jerusalem, Beer Sheva. Contact: George E. Membrino, PHD, Associate Dean, Office of Continuing Education, University of Massachusetts Medical School, 55 Lake Ave North, Worcester, MA 01605. Tel: (617) 856-3041.

Index to Advertisers

Ayerst Laboratories 4A, 5A, 6A, 19A, 20A, 21A, 22A
Bankers Trust Company 16A
St. Clare's Hospital 13A
Classified Advertising 8A, 14A, 17A
Eli Lilly and Company 10A
Marion Laboratories 1A, 2A
McGraw Hill Publications 7A
Physician's Reciprocial Insurers 12A
A. H. Robins Company 9A, 11A
Roche Laboratories .... 3rd & 4th Cover
Sandoz Pharmaceuticals 15A
Upjohn Company .... 2nd Cover
U.S. Air Force 18A

REAL ESTATE FOR SALE OR RENT—CONT'D

DOCTOR'S OFFICE NEAR MT. SINAI, high east 90's. 1200-square-foot office has a large waiting room, 2 consulting rooms, 3 examining rooms, 2 half baths, kitchenette and wall-to-wall carpeting. Office faces South and is very sunny and bright. Well priced with low maintenance and 75% financing allowed. Available exclusively with: Cyrilla Layland, (212) 439-4524, Res. (212) 427-7577. Albert B. Ashford, Inc.

82 STREET BETWEEN COLUMBUS & AMSTERDAM architect designed new Medical Office to share. Furnished and equipped. Exclusive use of consult office. Available April 1. For information call (212) 675-8312.

5th AVENUE/11th ST. Newly renovated, fully equipped medical office to share in elegant townhouse on historic 11th St. Ideal for primary care internists, FP, or OB/GYN. Call (212) 529-4330 10–5.

MISCELLANEOUS

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuarial certification and employee statements of participation—partnership agreements and professionals incorporation—No insurance required—references upon request—Wachstock and Dienstag Attorneys at Law, 122 Cutter Mill Road, Great Neck, NY 11021 (516) 773-3322.

ARE YOU HAVING A PROBLEM RELATING TO CHEMICAL DEPENDENCE? If your answer is affirmative, contact Susan Kaplan, Attorney-at-Law, (212) 877-5998. Practice limited to legal problems directly involving your license to practice medicine—with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professionals), and as an Assistant District Attorney in Nassau County. Susan Kaplan, Esq., 165 West End Avenue, Suite 27P, New York, N.Y. 10023.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta, GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

FIRM OF GERALD GOLDBERG, CPA-ATTORNEY, formerly with IRS, announces his availability to prepare taxes and plan under changing tax environment, accounting, tax appeals, legal representation on practice transfers. (212) 406-1156 and (516) 565-6260.

Doctor . .

Do you, or a physician you know, have a problem with alcohol, drugs, or emotional illness?

If so, please contact the Medical Society of the State of New York's Committee for Physicians' Health, whose function is to assist, *confidentially.*

Telephone: 516-488-7777.

MISCELLANEOUS—CONT'D

HAVE YOU BEEN CONTACTED BY A REPRESENTATIVE FROM THE OFFICE OF PROFESSIONAL MEDICAL CONDUCT? If the answer is yes, contact Susan Kaplan, Attorney-at-Law, (212) 877-5998. Practice limited to the crucial legal problems directly affecting your license to practice medicine—with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professions), and as an Assistant District Attorney in Nassau County. Susan Kaplan, Esq., 165 West End Avenue, Suite 27P, New York, N.Y. 10023.

PROFESSIONAL MISCONDUCT ATTORNEYS. William L. Wood Jr., formerly Executive Director of the New York State Office of Professional Discipline and Anthony Z. Scher, formerly Director of Prosecutions, announce the opening of their law office, Wood & Scher. Our recent tenure as chief enforcement officers has given us experience which will allow us to represent physicians in professional misconduct proceedings, malpractice licensure, license restoration, controlled drug proceedings, reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practitioners. Wood and Scher, Attorneys at Law, One Chase Road, Scarsdale, New York 10583. Telephone 914-723-3500.

MISCELLANEOUS—CONT'D

COMPUTERIZE YOUR BILLING—Remarkably easy-to-use software automatically prints bills, statements, insurance forms. Menu-driven. Reports ageing balances, referral sources, income by time period, procedure & more. Installs automatically. IBM and compatibles. Solo practice only $399. Group practice, only $499. Free information. Demonstration disk with complete manual, $10. Call or write REM Systems, Inc., Dept. NY3, 281 Dug Hill Road, Brownsboro, AL 35741 (205) 534-3457. VISA, MC Accepted.

WEIGHT LOSS TUTORIAL, NO FEE for physicians and their staff. Individual help in setting up a medically documented, effective, economical, uncomplicated weight loss program in your office practice. Contact Allan Lazar, M.D., 420 Grand Avenue, Englewood, N.J. 07631. (201) 568-3742.

GREAT LAKES ASS'N OF CLINICAL MEDICINE PRESENTS: True Preventive Medicine, "Nutrition is Therapy": Jonathan Wright, M.D. of Wright/Gaby Foundation, March 26–27 (1½ days). "Elemental Medicine": International Congress with noted speakers, March 27–28 (1½ days). Contact Warren Delano, 24700 Center Ridge Road, Westlake, Ohio 44145. (216) 835-1212.

AIM HIGH

A PRESCRIPTION FOR PHYSICIANS

BOTHERED BY:

- ★ Too much paperwork?
- ★ The burden of office overhead?
- ★ Malpractice insurance costs?
- ★ Not enough time for the family?
- ★ No time to keep current with technology and new methods?
- ★ No time or money for professional development?

JOIN THE AIR FORCE MEDICAL TEAM;
WE'LL PROVIDE THE FOLLOWING:

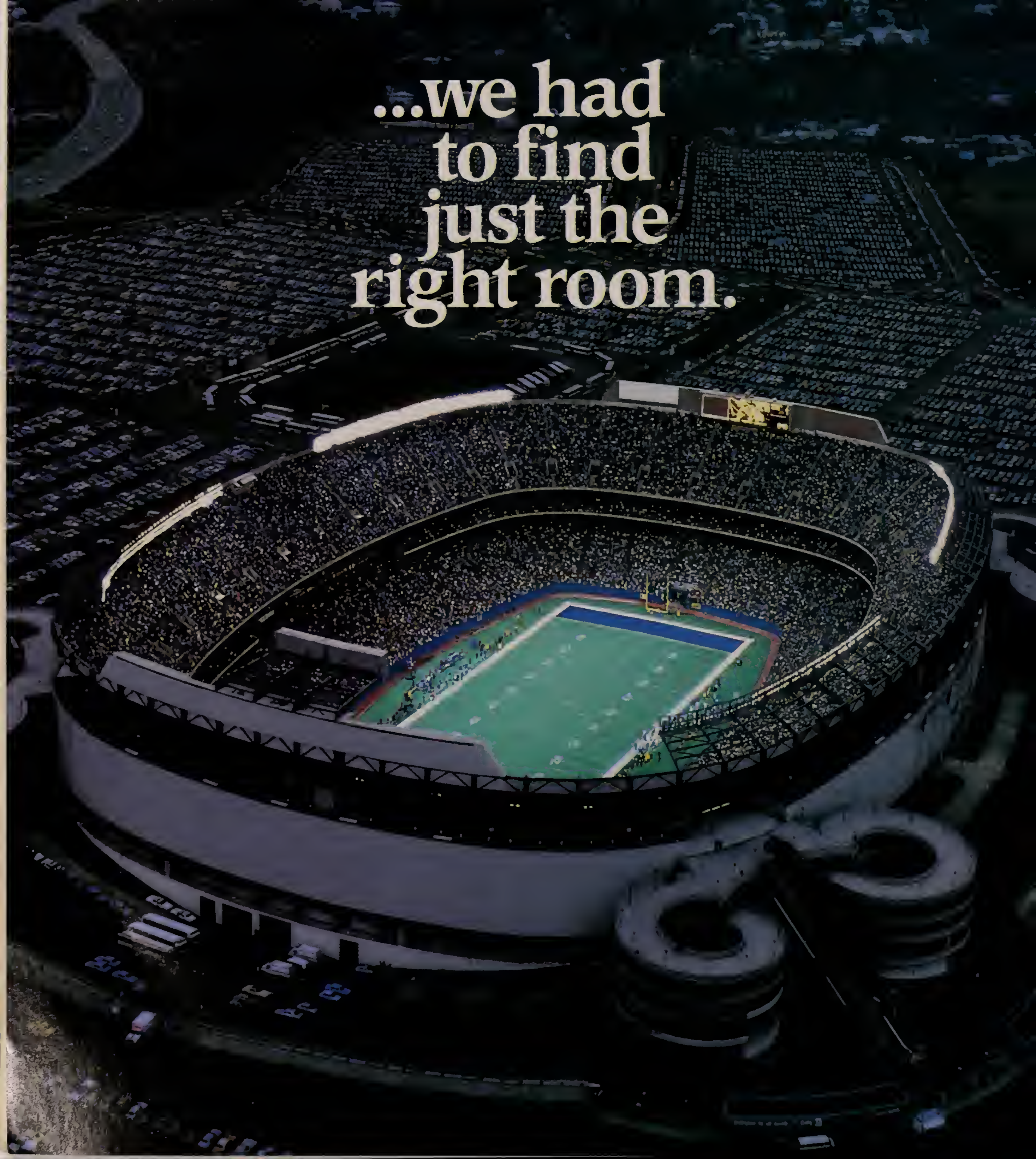
- ★ Competent and dedicated professional staff.
- ★ Time for patients and for keeping professionally current.
- ★ Financial security, a generous retirement for those who qualify.
- ★ If qualified, unlimited professional development.
- ★ Medical facilities all around the world.
- ★ 30 days of vacation with pay each year.
- ★ Complete medical and dental care.
- ★ Low cost life insurance.

Want to find out more? Contact your nearest Air Force recruiter for information at no obligation. Call

Capt Al Gugenberger
at 1-800-USA-USAF

AIR FORCE

To show you how many hypertensives stayed on INDERAL® LA (PROPRANOLOL HCl) after a major nationwide trial...


...we had
to find
just the
right room.

60,073 patients (90%) who started on INDERAL® LA stayed on INDERAL LA.[1]*

Surprising? Not really.

Because most patients on INDERAL LA (propranolol HCl) don't even know it's working.

A recent double-blind, placebo-controlled, crossover study in 138 hypertensive patients[2] revealed that INDERAL LA has a side effects profile unsurpassed by atenolol or metoprolol — which shows how well-tolerated once-daily INDERAL LA can be.

Sole therapy or concomitant therapy? Fifty-nine percent of the time, INDERAL LA stood on its own.

The patients in the nationwide compliance trial were no different from yours. Generally when the antihypertensive regimen is complicated, compliance may become a problem. So, the effectiveness of INDERAL LA as once-daily monotherapy is a big plus. Of the remaining hypertensives in the program, 36% were controlled merely with the addition of a diuretic to INDERAL LA.

For the noncompliant patients in your practice, INDERAL LA may well be the answer.

Almost 20,000 of the patients in the nationwide compliance trial were identified as having been noncompliant with their previous antihypertensive therapy. Their physicians reported that 88% showed improved compliance when placed on once-daily INDERAL LA.

Control, comfort, and compliance

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl) LONG ACTING CAPSULES

Like conventional INDERAL Tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree, and bronchial asthma.

*After a 30-day trial with INDERAL LA, physicians reported that 90% of the patients would remain on INDERAL LA.

The one you know best keeps looking better

Please see next page for brief summary of prescribing information

The one you know best keeps looking better

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules

CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol resulting from the slower rate of absorption of propranolol Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product INDERAL LA can provide effective beta blockade for a 24-hour period

The mechanism of the antihypertensive effect of INDERAL has not been established Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use Effects on plasma volume appear to be minor and somewhat variable INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential The significance of the membrane action in the treatment of arrhythmias is uncertain

The mechanism of the antimigraine effect of propranolol has not been established Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient But there are also situations in which sympathetic stimulation is vital For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm

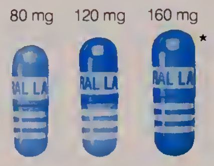
Propranolol is not significantly dialyzable

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl)

LONG ACTING CAPSULES



INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope INDERAL LA also improves exercise performance The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible)

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema) — PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e g , dobutamine or isoproterenol However, such patients may be subject to protracted severe hypotension Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers

DIABETES AND HYPOGLYCEMIA Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes In these patients, it may be more difficult to adjust the dosage of insulin

THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm Propranolol does not distort thyroid function tests

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker In one case, this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. *General* Propranolol should be used with caution in patients with impaired hepatic or renal function INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that INDERAL may interfere with the glaucoma screening test Withdrawal may lead to a return of increased intraocular pressure

Clinical Laboratory Tests Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension

Carcinogenesis, Mutagenesis, Impairment of Fertility Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

Pregnancy Pregnancy Category C INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose

There are no adequate and well-controlled studies in pregnant women INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers INDERAL is excreted in human milk Caution should be exercised when INDERAL is administered to a nursing woman.

Pediatric Use Safety and effectiveness in children have not been established

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy

Cardiovascular bradycardia, congestive heart failure, intensification of AV block, hypotension, paresthesia of hands, thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type

Central Nervous System lightheadedness, mental depression manifested by insomnia, lassitude, weakness, fatigue, reversible mental depression progressing to catatonia, visual disturbances, hallucinations; an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics.

Gastrointestinal nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis

Allergic pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress

Respiratory bronchospasm

Hematologic agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported

Miscellaneous alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained INDERAL LA should not be considered a simple mg for mg substitute for INDERAL INDERAL LA has different kinetics and produces lower blood levels Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval

HYPERTENSION — *Dosage must be individualized* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic The dosage may be increased to 120 mg once daily or higher until adequate blood-pressure control is achieved The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage of 640 mg may be required The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks

ANGINA PECTORIS — *Dosage must be individualized* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS)

MIGRAINE — *Dosage must be individualized* The initial oral dose is 80 mg INDERAL LA once daily The usual effective dose range is 160-240 mg once daily The dosage may be increased gradually to achieve optimum migraine prophylaxis If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued It may be advisable to withdraw the drug gradually over a period of several weeks

HYPERTROPHIC SUBAORTIC STENOSIS — 80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE — At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use

*The appearance of these capsules is a registered trademark of Ayerst Laboratories

---

REFERENCES:

1. INDERAL LA National Compliance Evaluation Program Data on file, Ayerst Laboratories

2. Ravid M Lang R, Jutrin I The relative antihypertensive potency of propranolol, oxprenolol, atenolol, and metoprolol given once daily *Arch Intern Med* 1985, 145 1321-1323

6412/187

Ayerst®

AYERST LABORATORIES
New York, NY 10017

© 1986 Ayerst Laboratories

YOUR ROCHE REPRESENTATIVE
WOULD LIKE YOU TO HAVE
SOMETHING THAT WILL...

ROCHE ®

... improve patient satisfaction with office visits

... improve patient compliance with your instructions

... reduce follow-up calls to clarify instructions

The new Roche product books

- Offer a supplement to, not a substitute for, patient contact
- Support your specific instructions to the patient
- Provide a long-term reinforcement of your oral counseling

Because you are the primary source of medical information for your patients, we invite you to look over the Roche Product Booklets shown below and ask your Roche representative for a complimentary supply of those applicable to your practice.

You, your medical problem and your treatment with
DALMANE®
BACTRIM™
brand of trimethoprim and sulfamethoxazole/Roche
LIBRIUM®
VALIUM®
LIMBITROL®
ROCHE MEDICATION ME EDUCATION

ROCHE
MEDICATION
ME
EDUCATION

Medicines that matter from people who care

COMMITTED TO EXCELLENCE

ROCHE

PRESIDENT'S ACHIEVEMENT AWARD

Presenting the winners of the 1987 Roche President's Achievement Awards

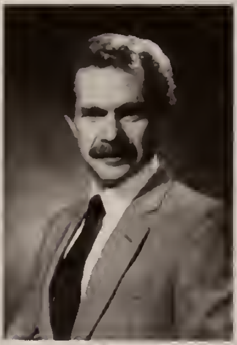
Hoffmann-La Roche is pleased to honor these outstanding sales representatives, chosen for their unparalleled dedication to the health-care field, professionalism and consistent high level of performance. Please join us in congratulating these exceptional individuals.

Gene E. Barone

Alan Berk

Joseph Kessler

Ralph Kressel


Sheldon Maginsky

Thomas J. O'Donnell

Alan Roiphe

Frederick G. Ruder, III

Firoz S. Singaporewala

Turn to the preceding page and find out how your award-winning Roche representative can help both you *and* your patients.

STACKS

NEW YORK STATE JOURNAL OF MEDICINE

APRIL 1987 • Volume 87, Number 4

MEDICAL SOCIETY OF THE STATE OF NEW YORK ORGANIZED 1807

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA
APR 23 1987

M.D.S.

Contents

COMMENTARIES

New prospects for preventing *Haemophilus influenzae* type b meningitis through immunization 195
CARL POCHEDLY, MD

Indoor air pollution 196
LAWRENCE E. HINKLE, JR, MD

Epithelioid sarcoma 197
CONSTANTINE P. KARAKOUSIS, MD, PhD

RESEARCH PAPER

Nosocomial pneumonias in a community teaching hospital 199
DAVID S. PRATT, MD; LORANN STALLONES, PhD; NANCY JOHNSON, RN; LINDA CHIARELLO, RN; JOHN J. MAY, MD

REVIEW ARTICLE

The role of building construction and ventilation in indoor air pollution: Review of a recurring problem 203
ROBERT J. McCUNNEY, MD

HISTORY

Arthur Donaldson Smith, MD: Physician, explorer, naturalist, and diplomat—Part III 210
PASCAL JAMES IMPERATO, MD

ESSAYS IN PEDIATRICS

Chemical perspectives in the design of a pediatric meningitis vaccine 226
CHRISTOPHER L. PENNEY, PhD

CASE REPORTS

Lithium poisoning 230
PAOLO DECINA, MD; ANNE M. SCHLEGEL, RN; RONALD R. FIEVE, MD

Isolated cardiac metastatic recurrence of epithelioid sarcoma after two and a half disease-free years 231
LAUREL J. STEINHERZ, MD; GERALD ROSEN, MD; PETER G. STEINHERZ, MD; JUNE ROBINS, RDMS; ANDREW HUVOS, MD; PHILIP R. EXELBY, MD

The use of computed tomographic scanning to diagnose sterile gluteal abscesses 233
RIFFAT R. CHAUDARY, MD, KUMUDHA RAMANATHAN, MD

LETTERS TO THE EDITOR

Gastric antacid bezoar in a patient with chronic renal failure 235
PAUL MOH, MD, BURTON GOLD, MD

Central phlebothrombosis with two venous irritants 236
HENRY SCHNEIDERMAN, MD; RICHARD A. GARIBALDI, MD; PAUL F. DAVERN, MBA, RPh

Extensive calcified pleural plaques 236
SUBRAHMANYA BALLAMBATTU, MD; STEVEN POWELL, MD; DAWN PETERS, BA

Acyclovir in the treatment of primary varicella pneumonia in non-immunocompromised adults 237
RAJINDER CHITKARA, MD; RICHARD E. GORDON, MD; FAROQUE A. KHAN, MB

ESSAY REVIEW

Taking stock: Physicians' memoirs 239
NAOMI R. BLUESTONE, MD, MPH

BOOK REVIEWS 242
NEWS BRIEFS 246
OBITUARIES 248
MEDICAL MEETINGS AND LECTURES 4A

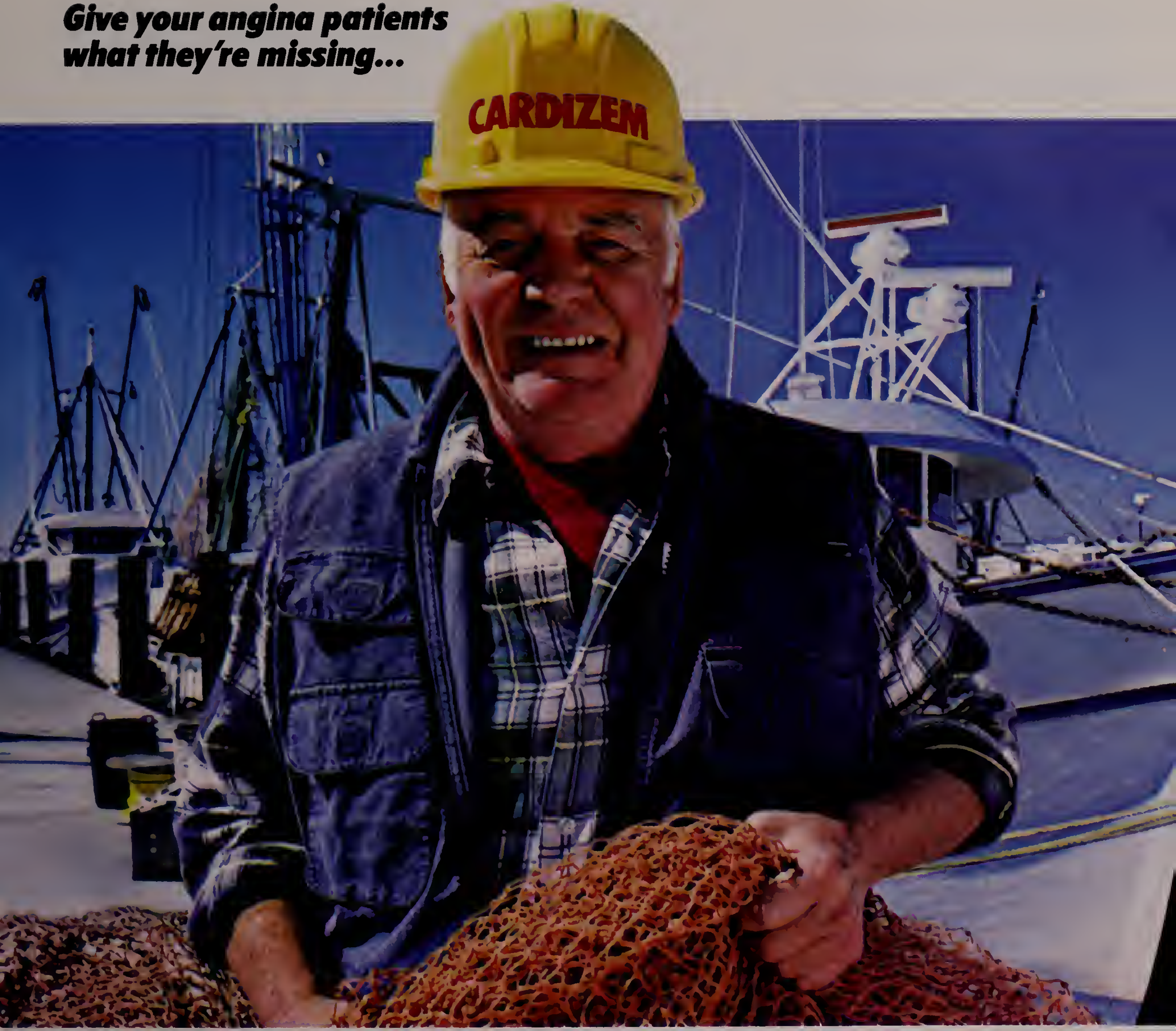
Motrin® 800 TABLETS mg
ibuprofen

Extra strength
Convenience
Economy

Upjohn
100 A Century
of Caring
1886·1986

©1986 The Upjohn Company

J-6138 January 1986


Give your angina patients what they're missing...
CARDIZEM

CARDIZEM®: FEW SIDE EFFECTS

diltiazem HCl/Marion

- **Antianginal action includes dilatation of coronary arteries, a decrease in vascular resistance/afterload, and a reduction in heart rate**
- **Proven efficacy when used alone in angina[1]**
- **Compatible with other antianginals[2,3]***
- **A safe choice for angina patients with coexisting hypertension, asthma, COPD, or PVD[4,5]**

***See Warnings and Precautions.**

Please see brief summary of prescribing information on the next page.

CARDIZEM® diltiazem HCl/Marion FEW SIDE EFFECTS IN ANTIANGINAL THERAPY

60 mg tid or qid

Brief Summary
Professional Use Information

CARDIZEM®
(diltiazem HCl) 30 mg and 60 mg Tablets

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1,243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt).
   Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, significant elevations in enzymes such as alkaline phosphatase, CPK, LDH, SGOT, SGPT, and other symptoms consistent with acute hepatic injury have been noted. These reactions have been reversible upon discontinuation of drug therapy. The relationship to CARDIZEM is uncertain in most cases, but probable in some. (See PRECAUTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. Diltiazem is excreted in human milk. One report suggests that concentrations in breast milk may approximate serum levels. If use of CARDIZEM is deemed essential, an alternative method of infant feeding should be instituted.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences as well as their frequency of presentation are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%). In addition, the following events were reported infrequently (less than 1%):

Cardiovascular:	Angina, arrhythmia, AV block (first degree), AV block (second or third degree — see conduction warning), bradycardia, congestive heart failure, flushing, hypotension, palpitations, syncope.
Nervous System:	Amnesia, gait abnormality, hallucinations, insomnia, nervousness, paresthesia, personality change, somnolence, tinnitus, tremor.
Gastrointestinal:	Anorexia, constipation, diarrhea, dysgeusia, dyspepsia, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH (see hepatic warnings), vomiting, weight increase.
Dermatologic:	Petechiae, pruritus, photosensitivity, urticaria.
Other:	Amblyopia, dyspnea, epistaxis, eye irritation, hyperglycemia, nasal congestion, nocturia, osteoarticular pain, polyuria, sexual difficulties.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: alopecia, gingival hyperplasia, erythema multiforme, and leukopenia. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

Issued 7/86

See complete Professional Use Information before prescribing.

References: 1. Pepine CJ, Feldman RL, Hill JA, et al: Clinical outcome after treatment of rest angina with calcium blockers: Comparative experience during the initial year of therapy with diltiazem, nifedipine, and verapamil. Am Heart J 1983; 106(6):1341-1347. **2.** Shapiro W: Calcium channel blockers: Actions on the heart and uses in ischemic heart disease. Consultant 1984;24(Dec):150-159. **3.** Johnston DL, Lesoway R, Humen DP, et al: Clinical and hemodynamic evaluation of propranolol in combination with verapamil, nifedipine and diltiazem in exertional angina pectoris: A placebo-controlled, double-blind, randomized, crossover study. Am J Cardiol 1985;55:680-687. **4.** Cohn PF, Braunwald E: Chronic ischemic heart disease, in Braunwald E (ed): Heart Disease: A Textbook of Cardiovascular Medicine, ed 2. Philadelphia, WB Saunders Co, 1984, chap 39. **5.** Schroeder JS: Calcium and beta blockers in ischemic heart disease: When to use which. Mod Med 1982;50(Sept):94-116.

Another patient benefit product from

M PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC.
KANSAS CITY, MO 64137

0246M6

NEW YORK STATE
JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
ELIZA H. CALDWELL, MD
JOSEPH F. MURATORE, MD
JOHN T. PRIOR, MD
GITA S. SINGH*
STANFORD WESSLER, MD

**Medical student*

Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Consulting Editor and Book Review Editor	RICHARD B. BIRRER, MD
Consulting Editor	NAOMI R. BLUESTONE, MD, MPH
Consulting Editor	CARL POCHEDLY, MD
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Managing Editor	CAROL L. MOORE
Librarian	ELLA ABNEY
Assistant Librarian	ELEANOR BURNS
Consulting Medical Writer	VICKI GLASER
Editorial Assistant	MILDRED J. ARFMANN
Secretary	ELIZABETH J. SOMERS
Advertising Director	ROBERT MILLER

ASSOCIATE EDITORIAL BOARD 1987

MICHAEL E. BERLOW, MD
RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MYLES S. GOMBERT, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
JAMES M. MORRISSEY, ESQ
STEPHEN NORDLICHT, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. United Media Associates, Inc, 16 Bruce Park Ave, Greenwich, CT 06830. Telephone: (203) 661-9702.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

SAMUEL M. GELFAND, MD,	*President*
JOHN A. FINKBEINER, MD,	*Past-President*
CHARLES D. SHERMAN, JR, MD,	*President-Elect*
JOSEPH F. SHANAPHY, MD,	*Vice-President*
JOHN H. CARTER, MD,	*Secretary*
GEORGE LIM, MD,	*Assistant Secretary*
MORTON KURTZ, MD,	*Treasurer*
ROBERT A. MAYERS, MD,	*Assistant Treasurer*
CHARLES N. ASWAD, MD,	*Speaker*
SEYMOUR R. STALL, MD,	*Vice-Speaker*

Councilors

Term Expires 1988

JAMES H. COSGRIFF, JR, MD,	Erie
RICHARD A. HUGHES, MD,	Warren
ANTONIO F. LASORTE, MD,	Broome
SIDNEY MISHKIN, MD,	Nassau

Term Expires 1989

ROBERT E. FEAR, MD,	Suffolk
STANLEY L. GROSSMAN, MD,	Orange
THOMAS D. PEMRICK, MD,	Rensselaer
RALPH E. SCHLOSSMAN, MD,	Queens

Term Expires 1990

DAVID M. BENFORD, MD,	New York
DUANE M. CADY, MD,	Onondaga
WILLIAM A. DOLAN, MD,	Monroe
ROBERT E. GORDON, MD,	Kings

Resident Councilor (representing the resident physician membership)

KATHLEEN E. SQUIRES, MD	New York

Student Councilor (representing the medical student membership)

MICHAEL PACIOREK,	Onondaga

Trustees

RICHARD D. EBERLE, MD, (*Chairman*)	Onondaga
EDGAR P. BERRY, MD,	New York
JAMES M. FLANAGAN, MD,	Wayne
ALLISON B. LANDOLT, MD,	Westchester
DANIEL F. O'KEEFFE, MD,	Warren
BERNARD J. PISANI, MD,	New York
VICTOR J. TOFANY, MD,	Monroe

Executive Vice-President	DONALD F. FOY
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Vice-President Emeritus	GEORGE J. LAWRENCE, JR, MD
Director, Division of Policy Coordination	IRMA A. ERICKSON
Director, Division of Scientific Publications	PASCAL JAMES IMPERATO, MD
Director, Division of Communications	EDWARD A. HYNES
Director, Division of Computer Services	IVAN H. NAJMAN
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Governmental Affairs	GERARD L. CONWAY, ESQ
General Counsel	DONALD R. MOY, JD
Director, Division of Management and Meeting Services	ROBERT MILLER
Director, Division of Membership Support Services	EUNICE M. SKELLY
Director, Division of Scientific and Educational Activities	PETER B. FARNSWORTH, MD
Director, Division of Socio-Medical Economics	MARIO V. MENGHINI
Medical Director, Committee for Physicians' Health	BARTON A. HARRIS, MD
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York.

Copyright © 1987, Medical Society of the State of New York. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.

FAMOUS PHYSICIANS ARE SHOWING UP FOR OUR 40th YEAR CELEBRATION.

40th YEAR
Postgraduate Medicine
Faith T. Fitzgerald, M.D.
Richard L. Byyny, M.D.
Frederick C. Goetz, M.D.
Frank A. Finnerty Jr., M.D.
J. Willis Hurst, M.D.
Paul P. Griffin, M.D.

Read Every Issue From Cover to Cover:

April
Symposium: Focus on General Internal Medicine . . . Faith T. Fitzgerald, M.D. coordinator
Special Article: General Internal Medicine 1947-1987 . . . . . Richard L. Byyny, M.D.

May 1
Symposium: Diabetes Mellitus
Special Article: Diabetes Mellitus 1947-1987 . . . . Frederick C. Goetz, M.D.

May 15
Symposium: Hypertension . . . . Frank A. Finnerty, Jr. coordinator
Special Article: Cardiology 1947-1987 . . . . J. Willis Hurst, M.D.

June
Symposium: Orthopedics in Primary Care
Special Article: Orthopedics 1947-1987 . . . . Paul P. Griffin, M.D.

40th YEAR
Postgraduate Medicine
Every Issue is a Celebration of Clinical Information

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices unless the information is submitted at least three months prior to the event.*

MAY 1987

AROUND THE STATE

ALBANY

May 7. **Endocrinology 1987.** Albany Medical College. *May 28.* **Dermatology 1987.** Albany Medical College. Contact: Lynne Wechsler, Conference Coordinator, Albany Medical College, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

May 14. **Effectiveness of Multidimensional Geriatric Assessment Units in Clinical Decision Making.** The Desmond Americana, Albany. Contact: Barbara Post, Sunnyview Hospital and Rehabilitation Center, 1270 Belmont Ave, Schenectady, NY 12308. Tel: (518) 382-4595.

BRONX

May 27. **Gunshot Wounds.** Contact: Mark L. Taff, MD, Nassau County Medical Examiner's Office, PO Box 160, East Meadow, NY 11554. Tel: (516) 542-2400.

MANHATTAN

May 4–8. **Surgical Neuroangiography.** 28 Cat 1 Credits. NYU Medical Center. Contact: NYU Medical Center Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

May 6. **Lower Extremity Orthotics and Prosthetics Update.** *May 13.* **Muscle Diseases—Update 1987.** Beth Israel Medical Center. Info: Beverley R. Baptiste, Medical Education Office, Beth Israel Medical Center, First Ave at 16th St, New York, NY 10003. Tel: (212) 420-2849.

May 9. **Vitrectomy in Anterior and Posterior Segment Surgery.** Manhattan Eye, Ear & Throat Hospital. Contact:

(continued on p 8A)

Photography by Ken Nahoum

AIDS is a complex disease, assaulting the psyche as well as the body. The Spellman Center for the Treatment of Persons With AIDS treats both. A comprehensive medical facility, Spellman is devoted to the care of those suffering from Acquired Immune Deficiency Syndrome.

Respect for individual dignity guides the Spellman program. Staffed by trained personnel dedicated to working with AIDS patients, the Center includes family and friends in the management of the patient's health crisis. Spellman also operates a 24-hour information hotline at 1-800-433-AIDS.

For criteria on admission and referrals, please contact the Medical Director, at **1-212-307-0735**. Spellman is a division of St. Clare's Hospital and Health Center, designated the first AIDS treatment center by New York State.

The Spellman Center
for the Treatment of Persons With AIDS
St. Clare's Hospital and Health Center

415 W 51st St
New York, NY 10019

An Affiliate of New York Medical College

An AZT Clinical Site

Medical experts,
providers of care

Classified Advertising

PHYSICIANS WANTED

PHYSICIANS—The New York City Police Department is accepting contract proposals from Board Certified Physicians in the fields of Neurology, Orthopedics, Psychiatry, and Radiology to serve as consultants. Duties will include examination of Police Officers and Police Officer Candidates. Radiologists will read and evaluate X-Rays. Board certified physicians in the above fields of medicine may request applications from the New York City Police Department, Health Services Division, One Lefrak Plaza, Corona, New York 11368, or call (718) 803-4554. All contract proposals must be received by 3PM 4/30/87 at above address. N.Y.P.D. is an Equal Opportunity Employer.

PHYSICIANS—The New York City Police Department is accepting contract proposals from Licensed Medical Doctors to serve on the New York City Police Department Retirement Board. Duties will include examinations of Police Officers to determine fitness-for-duty and eligibility for Disability Retirement. Special consideration will be given to physicians possessing expertise in orthopedics, cardiology, psychiatry and disability evaluation. Physicians may apply to the N.Y.P.D., Health Services Division, One Lefrak City Plaza, Corona, New York 11368, or call (718) 803-4554. All applications must be received by 3PM 4/30/87 at the above address. N.Y.P.D. is an Equal Opportunity Employer. (Hiring contingent on Mayor's Contract Review Committee and Board of Estimate approval. Any hiring will be rescinded if such approval is not obtained.)

PSYCHOLOGIST/PSYCHIATRIST CONSULTANTS—The New York City Police Department is seeking psychologist/psychiatrist consultants to review police officer candidate evaluations and/or conduct fitness-for-duty evaluation of police personnel. Hours and salary vary depending on type of evaluation and experience. Applicants must possess the following minimum requirements: (1) Doctorate in clinical, counseling, or school psychology, five (5) years post-degree experience, and New York State license or; (2) Board certification in psychiatry plus five (5) years post residency experience. In addition, it is desirable for applicants to have experience dealing with law enforcement issues and supervising others. Applications may be obtained from the N.Y.P.D., Health Services Division, One Lefrak City Plaza, Corona, New York 11368, or call (718) 803-4554. All applications must be received by 3PM 4/30/87 at the above address. N.Y.P.D. is an Equal Opportunity Employer.

LABORATORY—The New York City Police Department is accepting contract proposals from Clinical Laboratories to perform basic blood chemistry, Cardiac Profile and CVC with differential tests. Must be licensed by N.Y.C. Department of Health. Applications may be obtained from the N.Y.P.D., Health Services Division, One Lefrak City Plaza, Corona, New York 11368, or call (718) 803-4554. All contract proposals must be received by 3PM 4/30/87 at the above address. N.Y.P.D. is an Equal Opportunity Employer.

PHYSICIANS WANTED—CONT'D

LOCUM TENENS Board eligible Internist or family practitioner needed for Montefiore Internist's private office from April 22nd for 4–6 weeks. Grand Concourse near Montifiore Hospital. (212) 364-3322.

ARIZONA BASED PHYSICIAN RECRUITMENT firm has opportunities Coast to Coast. "Professionals working with Professionals". Over 13 years experience. Call (602) 990-8080, or send CV to: Mitchell & Associates, Inc., P.O. Box 1804, Scottsdale, AZ 85252.

MULTI-SPECIALTY NINE PHYSICIAN GROUP located in a rural setting adjacent to Western New York metropolitan area is seeking the following medical specialties: Internal Medicine/Gastroenterology/Cardiology—board certified/eligible Internist is needed. Sub-specialty interest welcome particularly in Gastroenterology or Cardiology. Approximately one-half of practice could be in either of these fields. Family Practice/Generalist—board certified/eligible Family Practitioner experienced and/or desirous of working in a group practice setting. Obstetrical practice available but not required.
We are currently involved in a wide variety of third party payor programs to include a prepaid (H.M.O.) component. Group is quite progressive and can offer the medical professional a broad array of in-house medical support services. Please direct responses to the address listed below and please include a summary of your education and experience along with salary expectations. Reply Dept. 437 c/o NYSJM.

DO YOU WANT TO SHARE A THRIVING DERMATOLOGY PRACTICE: Do you enjoy year around outdoor recreation? You should contact us—a progressive multi-specialty group of 68 physicians in a pleasant, growing community. We offer a liberal financial package and outstanding benefits. Send curriculum vitae and references to: Michael T. Anderson, M.D., Willmar Medical Center, 101 Willmar Ave. SW, Willmar, MN 56201.

PHYSICIANS needed for 75 children's private residential camps July and/or August. Contact: Association of Independent Camps (JM), 43 West 23rd Street—Sixth Floor, New York, N.Y. 10010 (212) 645-6620.

INTERNIST IS WANTED AS PARTNER to family physician practice in Queens. Yugoslavian origin preferred, but not necessary. Eventually take over this well established practice. Reply Dept. 435 c/o NYSJM.

NEW YORK, WESTERN: Seeking primary care trained physicians for full time Emergency Department positions. Moderate volume. Attractive hourly compensation, plus malpractice insurance, Director position available. Contact: Emergency Consultants, Inc., 2240 S. Airport Road, Room 42, Traverse City, MI 49684: 1-800-253-1795 or in Michigan 1-800-632-3496.

EMERGENCY MEDICINE POSITIONS—Full/part time emergency medicine physicians sought by multi state professional association for openings in metropolitan NY, PA, MD, DC, FL, New England and throughout U.S. Contact or send CV to Liberty Healthcare Corporation, 399 Market Street, Suite 400, Philadelphia, PA 19106, (215) 592-7400 or outside Pa (800) 331-7122.

POSITIONS WANTED

RADIOLOGIST, A.B.R. & A.B.N.M. is seeking a salaried position with a group or a hospital for 4–5 years. Group partnership not a consideration. Expertise in Angiography including neuro (20 years). Special competence in nuclear cardiology. Mammography & xeromammography (10 years), cat-head & body (6 years). Artrography, special procedures, ultrasonography & diagnostic radiology. Location limited to 100 mile radius of New York City. Reply to Dept. 432 c/o NYSJM.

CARDIOLOGIST/INTENSIVIST male 55, 19 years CCU experience, 12 years full time ICU-CCU experience; board certified and recertified in internal medicine, certified in cardiology, desires relocation in Northeast. Reply Dept. 439 c/o NYSJM.

PRACTICES AVAILABLE

"SELLING PRACTICES IS OUR ONLY BUSINESS"

Want to maximize the return on your investment? Before you buy or sell, call for an appraisal to assure the best financial and transfer terms. We guide you through the entire sales process from initial meeting to closing. Serving NY, CT, NJ and MA.

Countrywide Business Brokerage, Inc.
319 East 24th St., Suite 23-G
New York, NY 10010
(212) 686-7902 (203) 869-3666

FOR SALE—Long established Primary Care Internal Medicine practice and office building. Medium sized Finger Lakes City. Ten minutes to 200 bed hospital. Three rental apartments in office building. Will stay on and introduce to patients. Terms generous. Reply Dept. 433 c/o NYSJM.

INTERNAL MEDICINE—White Plains, NY. Unique opportunity to establish practice. Rent modern office/private house, excellent location, walking distance of White Plains Hospital; rent adjacent apartment or entire house (2 additional floors). Acquire active 23 year practice from internist, nearby professional building. Take 2-4 years to transfer practice, terms negotiable. Hospital privileges not a problem. Write Box 105-H, Scarsdale, NY 10583.

EQUIPMENT

DISCOUNT HOLTER SCANNING SERVICES starting at $35.00. Spacelabs holter recorder (cassette) available from $1,275.00. Cardionostic holter recorder (cassette) available from $1,125.00. Smallest and lightest holters update. Fast service (24–48 hours) turn over. Hookup kits starting at $4.95. Special introductory offer of three free tests with any purchase or lease of the recorder. Cardiologist overread available for $15.00. If interested call (301) 870-3626.

50% OFF PREVIOUSLY OWNED MEDICAL—Laboratory, x-ray, ultrasound equipment. We buy, sell, broker, repair. Medical Equipment Resale & Repair, 24026 Haggerty, Farmington Hills, MI 48018, 1-800-247-5826, (313) 477-6880.

Un-switch-ables

Patients and physicians alike prefer

Transderm-Nitro®

nitroglycerin

Over half a billion* patches prescribed in the U.S. since 1982

The only patch with a rate-limiting membrane

Familiar, distinctive tan color and unique shape recognized by patients everywhere

The higher the turnover the greater the profits

There's no substitute for experience

CIBA

(See Brief Summary of Prescribing Information on the next page.)

*Data on file, CIBA Pharmaceutical Co.

629-7750-A

Transderm-Nitro®
nitroglycerin
Transdermal Therapeutic System

BRIEF SUMMARY (FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE INSERT)

INDICATIONS AND USAGE
This drug product has been conditionally approved by the FDA for the prevention and treatment of angina pectoris due to coronary artery disease. The conditional approval reflects a determination that the drug may be marketed while further investigation of its effectiveness is undertaken. A final evaluation of the effectiveness of the product will be announced by the FDA

CONTRAINDICATIONS
Intolerance of organic nitrate drugs, marked anemia, increased intraocular pressure or increased intracranial pressure.

WARNINGS
In patients with acute myocardial infarction or congestive heart failure, Transderm-Nitro system should be used under careful clinical and/or hemodynamic monitoring
In terminating treatment of anginal patients, both the dosage and frequency of application must be gradually reduced over a period of 4 to 6 weeks to prevent sudden withdrawal reactions, which are characteristic of all vasodilators in the nitroglycerin class.
Transdermal nitroglycerin systems should be removed before attempting defibrillation or cardioversion because of the potential for altered electrical conductivity which may enhance the possibility of arcing, a phenomenon associated with the use of defibrillators.

PRECAUTIONS
Symptoms of hypotension, such as faintness, weakness or dizziness, particularly orthostatic hypotension may be due to overdosage. When these symptoms occur, the dosage should be reduced or use of the product discontinued.
Transderm-Nitro system is not intended for immediate relief of anginal attacks. For this purpose occasional use of the sublingual preparations may be necessary

ADVERSE REACTIONS
Transient headaches are the most common side effect, especially when higher doses of the drug are used These headaches should be treated with mild analgesics while Transderm-Nitro therapy is continued When such headaches are unresponsive to treatment, the nitroglycerin dosage should be reduced or use of the product discontinued
Adverse reactions reported less frequently include hypotension, increased heart rate, faintness, flushing, dizziness, nausea and vomiting. These symptoms are attributable to the known pharmacologic effects of nitroglycerin, but may be symptoms of overdosage. When they persist the dose should be reduced or use of the product discontinued. In some patients, dermatitis may occur.

DOSAGE AND ADMINISTRATION
Therapy should be initiated with application of one Transderm-Nitro 5 system to the desired area of skin. Many patients prefer the chest; if hair is likely to interfere with system adhesion or removal, it can be clipped prior to placement of the system. Each system is designed to remain in place for 24 hours, and each successive application should be to a different skin area. Transderm-Nitro system should not be applied to the distal parts of the extremities
The usual dosage is one Transderm-Nitro 5 system every 24 hours. Some patients, however, may require the Transderm-Nitro 10 system. If a single Transderm-Nitro 5 system fails to provide adequate clinical response, the patient should be instructed to remove it and apply either two Transderm-Nitro 5 systems or one Transderm-Nitro 10 system. More systems may be added as indicated by continued careful monitoring of clinical response The Transderm-Nitro 2.5 system is useful principally for decreasing the dosage gradually, though it may provide adequate therapy for some patients when used alone
The optimal dosage should be selected based upon the clinical response, side effects, and the effects of therapy upon blood pressure. The greatest attainable decrease in resting blood pressure that is not associated with clinical symptoms of hypotension especially during orthostasis indicates the optimal dosage To decrease adverse reactions, the size and/or number of systems should be tailored to the individual patient's needs
Do not store above 86°F (30°C).

PATIENT INSTRUCTIONS FOR APPLICATIONS
A patient leaflet is supplied with the systems

HOW SUPPLIED

Transderm-Nitro System Rated Release *in vivo*	Total Nitroglycerin in System	System Size	Carton Size
2.5 mg/24 hr	12.5 mg	5 cm²	30 Systems (NDC 0083-2025-26) *100 Systems (NDC 0083-2025-30)
5 mg/24 hr	25 mg	10 cm²	30 Systems (NDC 0083-2105-26) *100 Systems (NDC 0083-2105-30)
10 mg/24 hr	50 mg	20 cm²	30 Systems (NDC 0083-2110-26) *100 Systems (NDC 0083-2110-30)
15 mg/24 hr	75 mg	30 cm²	30 Systems (NDC 0083-2115-26) *100 Systems (NDC 0083-2115-30)

*Hospital Pack 100's

C85-35 (Rev 11/85)

Dist. by:
CIBA Pharmaceutical Company
Division of CIBA-GEIGY Corporation
Summit, New Jersey 07901

All transdermal nitroglycerin products are being marketed pending final evaluation of effectiveness by the FDA.

© 1986, CIBA. 629-7750-A

C I B A

(*continued from p 4A*)

Martha Klapp, Manhattan, Eye, Ear & Throat Hospital, 210 East 64th St, New York, NY 10021. Tel: (212) 605-3762.

May 13–16. **Interspecialty Facial Surgery Congress.** Contact: The International Oculoplastic Society, Inc, 630 Park Ave, New York, NY 10021. Tel: (800) 223-5400.

May 14–16. **Vascular Surgery 1987.** 24 Cat 1 Credits. The New York Hilton. Contact: Ann J. Boehme, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

May 14–16. **Suction Lipectomy of the Face and Body.** 18 Cat 1 Credits. The Waldorf Astoria Hotel. Contact: Francine Leinhardt, Coordinator, Manhattan Eye, Ear & Throat Hospital, 210 East 64th Street, New York, NY 10021.

May 20–22. **Magnetic Resonance Imaging.** 19 Cat 1 Credits. NYU Medical Center. Contact: NYU Medical Center Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

NASSAU

May 27. **Management of Bladder Dysfunction in Children.** 4½ Cat 1 Credits. North Shore University Hospital, Manhasset. Contact: Manju Chandra, MD, Program Coordinator, Children's Kidney Center, North Shore University Hospital, 300 Community Dr, Manhasset, NY 11030. Tel: (516) 562-4383.

AROUND THE NATION

CALIFORNIA

May 2–9. **Doppler and 2-D Echocardiography.** 40 Cat 1 Credits. Newport Beach. Contact Lisa Krehbiel, Institute for Medical Studies, 30131 Town Center Dr, Suite 215, Laguna Niguel, CA 92677. Tel: (714) 495-4499.

May 9–10. **Anatomy Review Course.** 15 Cat 1 Credits. University of California. *May 11–15.* **San Francisco Orthopaedic Review Course.** 40 Cat 1 Credits. Holiday Inn Golden Gateway, San Francisco. *May 21–22.* **Annual Meeting: Current Issues in Anatomic Pathology.** 12 Cat 1 Credits. Holiday Inn Union Square, San Francisco. Contact: University of California, Extended Programs in Medical Education, Room U-569, San Francisco, CA 94143-0742. Tel: (415) 476-5808.

FLORIDA

May 4–6. **Mesocortical and Mesolimbic Dopamine Systems.** Biscayne Bay Marriott, Miami. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd St, New York, NY 10021.

HAWAII

May 24–30. **5th Annual Cardiology Update.** 26 Cat 1 Credits. Honolulu. Contact: Lisa Krehbiel, 30131 Town Center Dr, Suite 215, Laguna Niguel, CA 92677. Tel: (714) 495-4499.

KENTUCKY

May 17–22. **Eighteenth Family Medicine Review—Session II.** Hyatt Regency Hotel, Lexington. Contact: Joy Greene, Continuing Medical Education, 132 College of Medicine Office Building, University of Kentucky, Lexington, KY 50536-0086. Tel: (606) 233-5161.

LOUISIANA

May 10–13. Annual Meeting of the American Lung Association/American Thoracic Society. New Orleans. Contact: American Lung Association, 1740 Broadway, New York, NY 10019-4374.

MARYLAND

May 13–15. **Cytotoxic T Cells: Biology and Relevance to Disease.** Bethesda Marriott, Bethesda. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd St, New York, NY 10021.

NEW JERSEY

May 16–17. **International Contact Lens Conference.** Bally's Park Place Casino Hotel, Atlantic City. Contact: International Contact Lens Conference, PO Box 1771, Harrisburg, PA 17105. Tel: (717) 233-8783.

(*continued on p 13A*)

TIME to subscribe

Consider a few features of
The Western Journal of Medicine:

- **Epitomes of Progress** — Each month this section highlights the major advances in a different specialty with 15 or 20 brief epitomes by experts in the field.
- **Clinical Investigation** — Five major clinical research societies coordinate important new research findings, emphasizing clinical value.
- **Socioeconomics** — WJM is often the first to predict, examine and evaluate socioeconomic changes, trends.
- **Yearly Special Issue** — Each December's issue is devoted to a topic vital to physicians: clinical nutrition (1979), cost of care (1980), geriatrics (1981), occupational/environmental (1982), cross-cultural medicine (1983), personal health maintenance (1984), high-tech medicine (1985), medical informatics (1986).

NYSJM 4/87

Enclosed is $______ to cover ______ subscriptions.
Subscription renewal ☐ Regular subscription ☐
Foreign subscription ☐

TO: ____________________

Street ____________________

City/State/Zip ____________________

Specialty ____________________

Gift Card to read "From____________________"

**The Western
Journal of Medicine**

Please mail this coupon with
enclosed payment to:

Circulation Department
P.O. Box 7602, San Francisco,
California 94120-7602

Subscriptions, 12 issues Per Year
U.S. and Canada $30 1 year,
$50 2 years

All other countries $50 1 year,
$90 2 years

THE MOUNT SINAI CENTER FOR FACIAL NERVE DISORDERS

A multi-disciplinary team of physicians and surgeons at The Mount Sinai Hospital for the complete evaluation and treatment of facial nerve disorders.

PROGRAM DIRECTORS

AVRIM R. EDEN, M.D.
CHIEF OF OTOLOGY
THE MOUNT SINAI MEDICAL CENTER

DAVID SIMPSON, M.D.
ASSISTANT PROFESSOR OF NEUROLOGY
CO-DIRECTOR, EMG LABORATORY

Address for patients and correspondence:

FACIAL NERVE CENTER
The Mount Sinai Hospital
19 East 98th Street
Suite 6A, Box 1191
New York, NY 10029

FOR PATIENT APPOINTMENTS OR INFORMATION:

(212) 650-2309

TORONTO NEUROLOGY UPDATE

University of Toronto
June 12th and 13th

Part of a series of continuing Medical Education covering Neurology. Two one day courses primarily for the family practitioner, which will also be of interest to internists, emergency room physicians, psychiatrists and neurologists.

June 12th: EPILEPSY

Concomitant Workshops on:
MS
NEUROPATHY

June 13th: HEADACHE

Concomitant Workshops on
DEMENTIA
MOVEMENT DISORDERS

Guest faculty includes: Dr. J.K. Penry, Dr. B.J. Wilder, Dr. T.J. Murray, Dr. V.C. Hachinski, Dr. F.D. Sheftell

For Details: Dr. M.J. Gawel
(416) 480-4959, (416) 281-3323

This course has been approved for 14 credit hours in Category 1 of the Physician's Recognition Award of the AMA.

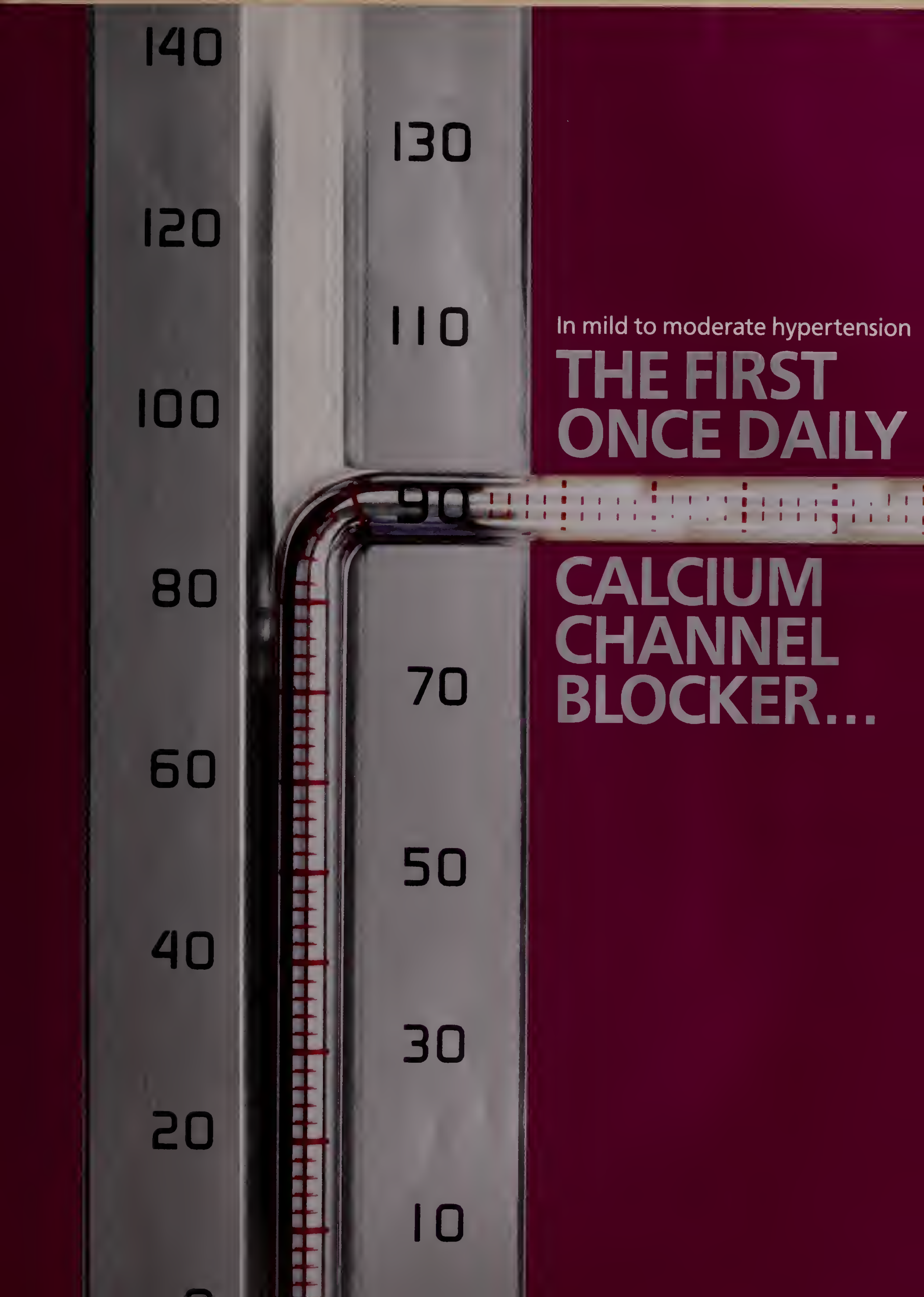
We Protect Money For People Who Make Money.

Our Fiduciary Services Division stands on a record of performance and experience. Our specialists provide highly personalized service and a wealth of resources in trust and estate administration, investments, tax planning, real estate, and oil and gas property management, to prepare for every situation and to help you plan for the future while reaping benefits in the present.

For more information, write or call: Richard S. Ritzel, Vice President, Private Clients Group, 280 Park Avenue, New York, N.Y. 10017. Tel. 212/850-4365.

© 1987 Bankers Trust Company
Member FDIC

Bankers Trust Company
Private Clients Group
We Make Money For People Who Make Money


140
130
120
110
100
90
80
70
60
50
40
30
20
10
In mild to moderate hypertension
THE FIRST
ONCE DAILY
CALCIUM
CHANNEL
BLOCKER…

NEW
ONCE DAILY

ISOPTIN® SR*

(verapamil HCl/Knoll)

240 mg scored, sustained-release tablets

JAMES B.
38, black male, heavy smoker. Prescribed a diuretic by another physician last year for hypertension.

YOUR CONCERNS
Presents with "smoker's cough." Workup reveals a BP of 150/107.

A LOGICAL CHOICE FOR CONTROL OF HIS BP
ISOPTIN® (verapamil HCl/Knoll) because...

- Black hypertensives often have low plasma renin activity and generally do not respond favorably to beta blockers.
- Beta blockers may increase the likelihood of bronchospasm.

ALICE W.
65, diabetic, overweight. Her BP has elevated to 190/98.

YOUR CONCERNS
She's on daily insulin.

A LOGICAL CHOICE FOR CONTROL OF HER BP
ISOPTIN® (verapamil HCl/Knoll) because...

- Unlike most beta blockers and diuretics, ISOPTIN has no adverse effects on serum glucose levels.
- Unlike most beta blockers, ISOPTIN does not mask the symptoms of hypoglycemia.

THOMAS G.
70, asthmatic. In the past, BP adequately controlled with 25 mg hydrochlorothiazide daily.

YOUR CONCERNS
Today patient presents with symptoms of gout. Workup reveals high uric acid level, low serum potassium, and BP elevated to 180/98.

A LOGICAL CHOICE FOR CONTROL OF HIS BP
ISOPTIN® (verapamil HCl/Knoll) because...

- Unlike diuretics, ISOPTIN will not decrease serum potassium levels or elevate uric acid levels.
- Unlike beta blockers, ISOPTIN can be used safely in asthma and COPD patients.

JOHN K.
42, Annual physical uncovered diastolic BP of 102... confirmed on three successive office visits. Unresponsive to nonpharmacologic intervention.

YOUR CONCERNS
Salesman, spends many hours of his working day in car... total cholesterol level 300, HDL 35.

A LOGICAL CHOICE FOR CONTROL OF HIS BP
ISOPTIN® (verapamil HCl/Knoll) because...

- Unlike diuretics, ISOPTIN does not cause urinary urgency.
- Unlike either beta blockers or diuretics, ISOPTIN will not adversely affect his already seriously compromised lipid profile.
- Unlike with propranolol, fatigue and impotence are rarely reported.

Antihypertensive therapy you and your patients can live with

*A product of Knoll research.

© 1986, BASF K & F Corporation

2538/2-87

Knoll Pharmaceuticals
A Unit of BASF K&F Corporation
Whippany, New Jersey 07981

BASF Group

Knoll®

Printed in U.S.A.

Please see adjacent page for brief summary.

In mild to moderate hypertension Brief Summary
THE FIRST ONCE DAILY CALCIUM CHANNEL BLOCKER

ISOPTIN® SR
(verapamil HCl/Knoll)
240 mg scored, sustained-release tablets

CONTRAINOICATIONS: 1) Severe left ventricular dysfunction (see WARNINGS), 2) Hypotension (less than 90 mmHg systolic pressure) or cardiogenic shock, 3) Sick sinus syndrome or 2nd or 3rd degree AV block (except in patients with a functioning artificial ventricular pacemaker).

WARNINGS: Heart Failure: ISOPTIN should be avoided in patients with severe left ventricular dysfunction (see DRUG INTERACTIONS). Patients with milder ventricular dysfunction should, if possible, be controlled before verapamil treatment. Hypotension: ISOPTIN (verapamil HCl) may produce occasional symptomatic hypotension. Elevated Liver Enzymes: Elevations of transaminases with and without concomitant elevations in alkaline phosphatase and bilirubin have been reported. Periodic monitoring of liver function in patients receiving verapamil is therefore prudent. Accessory Bypass Tract (Wolff-Parkinson-White): Patients with paroxysmal and/or chronic atrial flutter or atrial fibrillation and a coexisting accessory AV pathway have developed increased antegrade conduction across the accessory pathway producing a very rapid ventricular response or ventricular fibrillation after receiving intravenous verapamil. While this has not been reported with oral verapamil, it should be considered a potential risk. Treatment is usually O.C -cardioversion. Atrioventricular Block: The effect of verapamil on AV conduction and the SA node may cause asymptomatic 1st degree AV block and transient bradycardia. Higher degrees of AV block, while infrequent (0.8%), may require a reduction in dosage or, in rare instances, discontinuation of verapamil HCl. Patients with Hypertrophic Cardiomyopathy (IHSS): Although verapamil has been used in the therapy of patients with IHSS, severe cardiovascular decompensation and death have been noted in this patient population.

PRECAUTIONS: Impaired Hepatic or Renal Function: Verapamil is highly metabolized by the liver with about 70% of an administered dose excreted in the urine. In patients with impaired hepatic or renal function verapamil should be administered cautiously and the patients monitored for abnormal prolongation of the PR interval or other signs of excessive pharmacological effects (see OVEROOSAGE).

Drug Interactions: Beta Blockers: Concomitant use of ISOPTIN and oral beta-adrenergic blocking agents may be beneficial in certain patients with chronic stable angina or hypertension, but available information is not sufficient to predict with confidence the effects of concurrent treatment in patients with left ventricular dysfunction or cardiac conduction abnormalities. Digitalis: Clinical use of verapamil in digitalized patients has shown the combination to be well tolerated if digoxin doses are properly adjusted. However, chronic verapamil treatment increases serum digoxin levels by 50 to 75% during the first week of therapy and this can result in digitalis toxicity. Upon discontinuation of ISOPTIN (verapamil HCl), the patient should be reassessed to avoid underdigitalization. Antihypertensive Agents: Verapamil administered concomitantly with oral antihypertensive agents (e.g., vasodilators, angiotensin-converting enzyme inhibitors, diuretics, beta blockers, prazosin) will usually have an additive effect on lowering blood pressure. Patients receiving these combinations should be appropriately monitored. Oisopyramide: Oisopyramide should not be administered within 48 hours before or 24 hours after verapamil administration. Quinidine: In patients with hypertrophic cardiomyopathy (IHSS), concomitant use of verapamil and quinidine resulted in significant hypotension. There has been a report of increased quinidine levels during verapamil therapy. Nitrates: The pharmacologic profile of verapamil and nitrates as well as clinical experience suggest beneficial interactions. Cimetidine: Two clinical trials have shown a lack of significant verapamil interaction with cimetidine. A third study showed cimetidine reduced verapamil clearance and increased elimination to 1/2. Anesthetic Agents: Verapamil may potentiate the activity of neuromuscular blocking agents and inhalation anesthetics. Carbamazepine: Verapamil may increase carbamazepine concentrations during combined therapy. Rifampin: Therapy with rifampin may markedly reduce oral verapamil bioavailability. Lithium. Verapamil may lower lithium levels in patient on chronic oral lithium therapy. Carcinogenesis, Mutagenesis, Impairment of Fertility: There was no evidence of a carcinogenic potential of verapamil administered to rats for two years. Verapamil was not mutagenic in the Ames test. Studies in female rats did not show impaired fertility. Effects on male fertility have not been determined. Pregnancy (Category C): There are no adequate and well-controlled studies in pregnant women. ISOPTIN crosses the placental barrier and can be detected in umbilical vein blood at delivery. This drug should be used during pregnancy, labor, and delivery, only if clearly needed. Nursing Mothers: ISOPTIN is excreted in human milk, therefore, nursing should be discontinued while verapamil is administered. Pediatric Use: Safety and efficacy of ISOPTIN in children below the age of 18 years have not been established.

AOVERSE REACTIONS: Constipation 8.4%, dizziness 3.5%, nausea 2.7%, hypotension 2.5%, edema 2.1%, headache 1.9%, CHF/pulmonary edema 1.8%, fatigue 1.7%, bradycardia 1.4%, 3° AV block 0.8%, flushing 0.1%, elevated liver enzymes (see WARNINGS). The following reactions, reported in less than 1.0% of patients, occurred under conditions (open trials, marketing experience) where a causal relationship is uncertain; they are mentioned to alert the physician to a possible relationship: angina pectoris, arthralgia and rash, AV block, blurred vision, cerebrovascular accident, chest pain, claudication, confusion, diarrhea, dry mouth, dyspnea, ecchymosis or bruising, equilibrium disorders, exanthema, gastrointestinal distress, gingival hyperplasia, gynecomastia, hair loss, hyperkeratosis, impotence, increased urination, insomnia, macules, muscle cramps, myocardial infarction, palpitations, paresthesia, psychotic symptoms, purpura (vasculitis), shakiness, somnolence, spotty menstruation, sweating, syncope, urticaria. Treatment of Acute Cardiovascular Adverse Reactions: Whenever severe hypotension or complete AV block occur following oral administration of verapamil, the appropriate emergency measures should be applied immediately, e.g., intravenously administered isoproterenol HCl, levarterenol bitartrate, atropine (all in the usual doses), or calcium gluconate (10% solution). If further support is necessary, inotropic agents (dopamine or dobutamine) may be administered. Actual treatment and dosage should depend on the severity and the clinical situation and the judgment and experience of the treating physician.

OVERDOSAGE: Treatment of overdosage should be supportive. Beta-adrenergic stimulation or parenteral administration of calcium solutions may increase calcium ion flux across the slow channel, and have been used effectively in treatment of deliberate overdosage with verapamil. Clinically significant hypotensive reactions or fixed high degree AV block should be treated with vasopressor agents or cardiac pacing, respectively. Asystole should be handled by the usual measures including cardiopulmonary resuscitation.

Knoll Pharmaceuticals
A Unit of BASF K&F Corporation
Whippany, New Jersey 07981

knoll®

BASF Group
©1986, BASF K&F Corporation 2474/11/86 Printed in U.S.A

(continued from p 8A)

SOUTH CAROLINA

May 15–18. **The Spine—Advanced Concepts of Diagnosis and Treatment.** 19 Cat 1 Credits. Kiawah Island Resort—Charleston. Contact: Pat Curl, Spine Study Group, PO Box 33185, San Diego, CA 92103. Tel: (619) 692-9115.

TENNESSEE

May 28–31. **Flexible Fiberoptic Sigmoidoscopy in the Diagnosis of Colorectal Diseases.** 15 Cat 1 Credits. Opryland Hotel, Nashville. Contact: Seminars & Symposia, Inc, 132 East 70th Street, New York, NY 10021. Tel: (212) 517-7520.

VIRGINIA

May 8–10. **The 6th Annual MCV Cardiology Conference.** The Williamsburg Hospitality House, Williamsburg. *May 18–19.* **14th Annual Hans Berger Day and EEG Symposium.** Baruch Auditorium, Egyptian Building, Medical College of Virginia Campus, Richmond. *May 23–25.* **Gynecologic Urology and Pelvic Surgery.** The Williamsburg Hospitality House, Williamsburg. Contact: Kathy Martin, Office of Continuing Medical Education, Medical College of Virginia, Box 48, MCV Station, Richmond, VA 23298-0001. Tel: (804) 786-0494.

May 28–30. **Basic and Advanced Colposcopy and Laser.** 19 Cat 1 Credits. Contact: The American College of Obstetricians and Gynecologists, 600 Maryland Ave, SW, Suite 300 East, Washington, DC 20024-2588.

AROUND THE WORLD

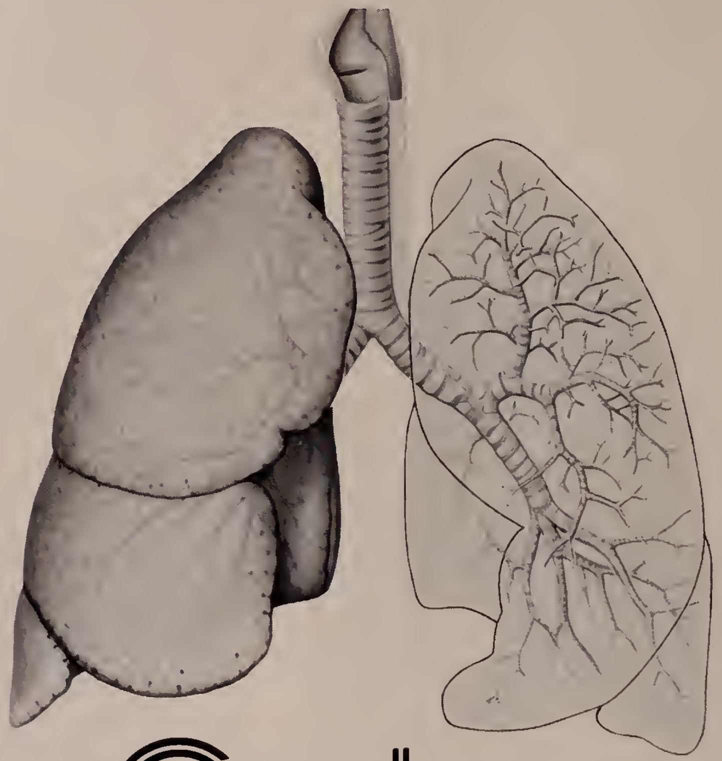
ITALY

May 24–27. **Sudden Infant Death Syndrome: Mechanisms and Interventions.** Villa Olmo, Lake Como. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd St, New York, NY 10021.

UNITED KINGDOM

May 2–10. **Medical Diagnostic Imaging.** 18 Cat 1 Credits. London. Contact: Spring Seminar, 21915 Roscoe Blvd, Suite 222, Canoga Park, CA 91304. Tel: (818) 340-0580, Ext 280.

Consider the causative organisms…



Ceclor®
cefaclor

250-mg Pulvules® t.i.d.

offers effectiveness against the major causes of bacterial bronchitis

Haemophilus influenzae, *H influenzae*, *Streptococcus pneumoniae*, *Streptococcus pyogenes*
(ampicillin-susceptible) (ampicillin-resistant)

Note: Ceclor® is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

Ceclor® (cefaclor)

Summary. Consult the package literature for prescribing information.

Indications: Lower respiratory infections, including pneumonia, caused by susceptible strains of *Streptococcus pneumoniae, Haemophilus influenzae,* and *S. pyogenes* (group A beta-hemolytic streptococci).

Contraindications: Known allergy to cephalosporins.

Warnings: CECLOR SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY. POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS.

Administer cautiously to allergic patients.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea. Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.

Precautions:

- Discontinue Ceclor in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of nonsusceptible organisms.
- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- In renal impairment, safe dosage of Ceclor may be lower than that usually recommended. Ceclor should be administered with caution in such patients.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk. Exercise caution in prescribing for these patients.

Adverse Reactions: (percentage of patients)

Therapy-related adverse reactions are uncommon. Those reported include:

- Gastrointestinal (mostly diarrhea): 2.5%.
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
- Hypersensitivity reactions (including morbilliform eruptions, pruritus, urticaria, erythema multiforme, serum-sickness-like reactions): 1.5%; usually subside within a few days after cessation of therapy. These reactions have been reported more frequently in children than in adults and have usually occurred during or following a second course of therapy with Ceclor. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.
- Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.
- Other: eosinophilia, 2%; genital pruritus or vaginitis, less than 1%.

Abnormalities in laboratory results of **uncertain etiology**

- Slight elevations in hepatic enzymes.
- Transient fluctuations in leukocyte count (especially in infants and children)
- Abnormal urinalysis; elevations in BUN or serum creatinine
- Positive direct Coombs' test
- False-positive tests for urinary glucose with Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (glucose enzymatic test strip, Lilly)

© 1986, ELI LILLY AND COMPANY [060485LR]

Lilly

Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285

Eli Lilly Industries, Inc.
Carolina, Puerto Rico 00630

600332

NEW YORK STATE
JOURNAL OF MEDICINE

April 1987 • Volume 87, Number 4

COMMENTARIES

New prospects for preventing *Haemophilus influenzae* type b meningitis through immunization

During the past 20 years physicians, public health workers, and others responsible for the health care of infants and children have become increasingly aware of the continuing morbidity and mortality resulting from infections due to *Haemophilus influenzae* type b (Hib). Initial concern focused on meningitis, the most striking and frequently encountered result of invasive illness due to this virulent organism.

Despite early diagnosis and proper therapy, 5% to 10% of children with meningitis still die, and neurologic damage (including seizures, deafness, and mental retardation) is reported in up to 45% of survivors. Increased attention to other forms of invasive disease due to Hib has highlighted the importance of epiglottitis, cellulitis, pyogenic arthritis, pneumonia (often with empyema), osteomyelitis, bacteremia, and septicemia.[1]

During the first five years of life, every American child has a 1-in-200 chance of developing Hib disease. An estimated 12,000 to 15,000 cases of Hib meningitis occur annually, and there are probably an equal number of children with other forms of systemic infection due to the same organism. Recent outbreaks of invasive Hib disease in day care centers and the emergence of Hib strains resistant to commonly used antibiotics have underscored the danger of the Hib organism. For these reasons, the possibility of vaccination against Hib infection has become increasingly attractive.[1] Our ultimate goal is to completely prevent Hib diseases by universal immunization.

The potential usefulness of vaccines prepared with the antigenic polysaccharide from the capsule of the Hib organism was first conclusively demonstrated in 1974 in Finland. But in the United States there was continued complacency and lack of interest in vaccination for two reasons. First, the relative effectiveness of antibiotic therapy caused us to ascribe a falsely high level of therapeutic potency to antibiotics and to place undue confidence in the efficacy of these drugs to combat serious Hib infections. Secondly, complacency was fostered by lack of accurate data on the actual incidence of serious Hib infections and lack of detailed assessment of the complications. The emergence of antibiotic-resistant strains, coupled with better epidemiologic data, has shattered this complacency.[1]

The *Haemophilus influenzae* type b polysaccharide vaccine, like its meningococcal and pneumococcal counterparts, is effective in older infants and children above 18 months of age, but unreliable in younger infants. This has also been a deterrent to further development of vaccines in this country, since approximately 50% of Hib patients are infants under 18 months of age, and an additional 25% of cases occur in those under 24 months of age. However, the convincing data from the Finnish study demonstrate the efficacy of this vaccine in older infants. An extrapolation of the Finnish data to the US suggests that routine childhood vaccination could prevent at least 10,000 cases of serious invasive disease annually.[1]

The recently licensed Hib polysaccharide vaccine represents an important advance in our efforts to prevent serious infections caused by encapsulated bacteria in children.[2] This vaccine is safe and effective in children two years of age or older. Its use in children two to five years of age in this country will prevent an estimated 1,000 to 2,000 cases of meningitis per year and most cases of epiglottitis. Although safe, this vaccine is not routinely recommended for children older than five or six years of age, since the risk of disease then is very low.

It is likely that the new Hib polysaccharide-protein conjugate vaccines will prove to be more effective than the plain polysaccharide vaccines in conferring protection on children below two years of age, the group at greatest risk of Hib disease. Preliminary data indicate that a prototype Hib polysaccharide, outer membrane protein conjugate vaccine is immunogenic in infants as young as two to six months of age. However, to achieve universal vaccine efficacy in this age group, the vaccines currently under evaluation may require further modification, such as the inclusion of certain Hib noncapsular antigens.[3]

A vaccine consisting of the polysaccharide (PS) capsule of *Haemophilus influenzae* type b has recently been licensed in the United States. This vaccine is safe and effective in preventing invasive Hib disease in children two years of age and older, but it is ineffective in younger children, the group at greatest risk of disease. The PS vaccine also may be ineffective in preventing disease in certain subgroups of the population that are genetically at increased risk of disease and show impaired antibody responses to immunization. Thus, new strategies need to be considered.[3]

Currently, several new Hib polysaccharide-protein conjugate vaccines are being evaluated. These vaccines differ in their method of preparation, carrier protein, and polysaccharide size. In contrast to the plain Hib PS vaccine, conjugate vaccines are immunogenic in infants and elicit boostable increases in antibody to polysaccharide upon reinjection of vaccine. However, some infants less than six months of age do not respond.[3]

To confer protection on all infants, it may be necessary to modify further the conjugate vaccines. One approach is to use outer membrane proteins (OMPs) as vaccine components. Five major OMPs have been purified from Hib, and three of these contain antigens capable of eliciting strain-specific protective antibodies in experimental animals.[3]

It seems that PS-protein conjugate vaccines hold enormous promise for the prevention of Hib disease in infants, but further work is needed to define the optimal carrier protein, polysaccharide size, and method of coupling.[3] Information is also needed on whether genetic factors influence responses to these vaccines.

The past 35 years have witnessed important advances in the treatment of Hib disease and the recent licensure of a polysaccharide vaccine for prevention of Hib disease in older children. Although considerable work remains to be done before the new conjugate vaccines are ready for licensure in the United States, the prospects for preventing Hib disease in infants by immunization are excellent. It is not unreasonable to expect that most, if not all, of the remaining morbidity and mortality that is associated with Hib diseases in infants will one day be eliminated.[3]

The article by Penney[4] in this issue of the *Journal* summarizes recent advances in the development of new vaccines for prevention of *Haemophilus influenzae* type b meningitis in children. Within the past year at least three capsular polysaccharide vaccines (Praxis, Lederle, and Connaught) have been marketed. However, none of these vaccines is effective among children under one year of age, the age of highest risk of meningitis. Furthermore, it was recently noted that meningitis may occur within 24 hours after receiving this vaccine. Thus, a number of companies are rushing to bring to market a second generation of semisynthetic vaccines which will protect these young children.

It is important that physicians who care for children be aware of the nature of these vaccines and be able to assess their usefulness in preventing serious infections.

The article by Penney[4] discusses the chemical differences between two types of vaccines: the capsular polysaccharide type and the semisynthetic type. It also discusses the optimal molecular design of such vaccines, and refers to the newest vaccines now under development. A hypothesis is presented which the author believes will allow development of effective vaccines against the three principal causes of early childhood meningitis: *Haemophilus influenzae*, *Neisseria meningitidis*, and *Streptococcus pneumoniae*.

CARL POCHEDLY, MD
Consulting Editor

Department of Pediatrics
Health Sciences Center
University of Texas at San Antonio
San Antonio, TX 78284

1. Katz SL: b-CAPSA-I vaccine: an overview clinical monograph. Evansville, Ind, Mead Johnson and Co, 1985.
2. Marwick C: FDA soon to license H influenzae vaccine [news]. *JAMA* 1985; 253:1232-1233.
3. Granoff DM, Munson RS: Prospects for prevention of Haemophilus influenzae type b disease by immunization. *J Infect Dis* 1986; 153:448-460.
4. Penney CL: Chemical perspectives in the design of a pediatric meningitis vaccine. *NY State J Med* 1987; 87:226-229.

Indoor air pollution

If one considers the term "air pollutants" to mean unwanted substances in the air, then the indoor air pollutants that affect health are the unwanted substances in indoor air that cause morbidity and mortality among people.[1] In the general order of the magnitude of their effect upon people's health, these substances arise from: the presence of people indoors, the activities of people indoors, the presence of combustion products in indoor air, the presence of animals indoors, and the effects of the building itself as well as its furnishings.

The presence of people indoors is responsible for the vast number of infectious diseases, including the common respiratory ones which are caused by bacteria and viruses that are disseminated by airborne droplets or aerosols.[2,3] The activities of people indoors—which include manufacturing, commercial, and clerical activities; household activities that include cooking, cleaning, household repairs, and hobbies; and tobacco smoking—are responsible for another large group of illnesses due to toxic and allergic causes. Combustion products of indoor fires, accidental or intentional, or from the use of various forms of heating apparatuses or combustion engines, are responsible for another large group of illnesses and are a major cause of deaths indoors. The presence of animals indoors, not simply cats, dogs, and birds as pets, but also insects such as mites, is responsible for a number of infectious and allergic conditions, and the structure of the building itself—its wallboard, paint, insulating materials, and the functioning of its air conditioners and dehumidifiers—also causes a relatively small number of illnesses and deaths annually.

The paper by McCunney[4] in this issue of the *Journal* deals only with the fifth category of indoor pollutants. This is a category which is of intense interest to people who build and operate buildings, because illnesses such as legionnaires' disease, and the possible effects that radon or asbestos in building structures may have on the health of building occupants, are matters of intense public concern, and sources of potential liability for the building owner or operator. However, the illnesses that arise from these sources represent only a minute proportion of the diseases that arise from "pollutants" in indoor air. Indeed, there is even a question as to whether or not radon arising from building structures, or asbestos statically confined within the walls of buildings, have ever caused any clinically recognizable illnesses or deaths.

The paper by McCunney[4] contains information that can be useful for industrial hygienists, toxicologists, building owners and operators, and, to a limited extent, medical practitioners. The author points out that radon appears to be a hazard to health among workers in uranium mines who are exposed to relatively high concentrations of this radioactive gas in association with dusts, especially if they smoke; and that asbestos appears to be a hazard to the health of unprotected men who work for long periods installing asbestos insulation or who work in other places where they are exposed to a high level of asbestos dust, especially if they also smoke cigarettes. However, neither of these substances have been shown to be causes of illness or death when they are confined statically in the walls of buildings in the extremely low concentrations to which people are exposed to them under these circumstances.

The author makes clear that the phenomenon that he has called "mass hysteria" does not appear to be, strictly speaking, the result of a toxicological effect of indoor air pollutants, but seems to arise rather from a psychological reaction to perceived odors, or from a belief that a pollutant is present in the air. This is a problem for those who have to evaluate whether a mass outbreak of symptomatic illness is a result of a substance present in indoor air but is not, strictly speaking, a result of the presence of that substance *per se*, but of the perception of the substance as being toxic or dangerous to those who breathe it. The paper by McCunney[4] provides a broad overview of the role that the construction and ventilation of buildings plays in indoor air pollution. It is an area that will require constant and responsible attention given what we already know.

LAWRENCE E. HINKLE, JR, MD
Professor, Department of Medicine

Chief, Division of Human Ecology
Cornell University Medical College
New York, NY 10021

1. Hinkle LE, Murray SH: The importance of the quality of indoor air. *Bull NY Acad Med* 1981; 57:827-844.
2. Couch RB: Viruses and indoor air pollution. *Bull NY Acad Med* 1981; 57:907-921.
3. Imperato PJ: Legionellosis and the indoor environment. *Bull NY Acad Med* 1981; 57:922-935.
4. McCunney RJ: The role of building construction and ventilation in indoor air pollution: Review of a recurring problem. *NY State J Med* 1987; 87:203-209.

Epithelioid sarcoma

Epithelioid sarcoma occurs predominantly in the extremities, often in the hand or foot in association with aponeurotic structures. It has a tendency to metastasize to the regional lymph nodes in about 30% of cases.[1] Histologically it is characterized by short oval and round epithelioid cells, with finely granular cytoplasm arranged in cords and clusters.[2] The long-term survival rate is similar to that of most other soft tissue sarcomas.

Most textbooks still treat epithelioid sarcoma as a distinct entity.[1,2] However, some authors[3,4] consider this tumor to be a variant of synovial sarcoma. In the case report appearing in this issue of the *Journal*, Dr Steinherz et al[5] have espoused this latter view.

Apart from the intricacies of the pathologic diagnosis, the important thing from the clinical point of view is early diagnosis. Epithelioid sarcoma arises most frequently in the lower extremity,[6] but the upper extremity and the skeletal muscles of the abdominal wall and trunk may be affected. The appearance of a swelling or pain may draw attention to this neoplasm. Soft tissue sarcomas comprise about 1% of all cancers in patients admitted to general hospitals.[1] Approximately 4,500 new cases and 1,600 deaths are reported in the United States each year. The relative incidence of synovial sarcomas in various series has ranged from 0.8% to 14.1%.[1] As with the other soft tissue sarcomas, survival in synovial sarcomas varies according to the size of the tumor at the time of diagnosis, those individuals with smaller tumors (less than 5 cm in diameter) having a better prognosis.[6,7] Survival rates also vary according to the histologic subtype. Monophasic spindle cell sarcomas have the worst prognosis, with a five-year survival rate of about 30%, while that of biphasic sarcomas is about 54%.[8] As a group, synovial sarcomas are among those with the worst prognosis, and are histologically classified as Grade III. In the current system of staging sarcomas, the grade of the tumor is the chief prognostic determinant.[9] Usually three grades are recognized: Grade I represents the well differentiated, Grade II the medium, and Grade III the poorly differentiated sarcomas.

With early diagnosis a higher chance of cure and limb salvage may be expected. A swelling that cannot be explained as due to a benign cause or condition, or one that does not recede quickly, should be subjected to biopsy. Needle biopsy may occasionally yield sufficient diagnostic material. More often diagnosis requires an excisional biopsy for the occasional small lesion, and an incisional biopsy for the majority of the lesions.

In the past, 40–50% of patients with extremity soft tissue sarcomas were managed with amputation.[10,11] The rate of local recurrence in our institute was then 65% after local excision, 36% after wide excision, and 8% after amputation.[10] Since 1977 the rate of amputations in our institute has dropped to 5%.[12] About half of 121 patients with extremity sarcomas were given adjuvant postoperative radiation due to narrow surgical margins. The five-year local recurrence rate was 6% after the combination of surgery and radiation, and 10% after surgery alone.[13] The results of conservative resection plus adjuvant radiation, reported from the National Cancer Institute,[14] and those of preoperative intra-arterial infusion of doxorubicin hydrochloride plus radiation followed by surgery, reported from UCLA, are quite satisfactory.[15]

Soft tissue sarcomas primarily involve the lungs when they metastasize through the hematogenous route.[16] This biologic peculiarity of soft tissue sarcomas constitutes the rationale for the surgical removal of pulmonary metastases, a policy which, along with chemotherapy, has provided five-year survival rates in the range of 20–30%.[17]

Although the various histologic subtypes of soft tissue sarcoma exhibit different responses to chemotherapeutic agents, in reporting results they are grouped together due to the very small number of each subtype. The objective response to doxorubicin has ranged from 13% to 34% in various reports.[1] Combinations of this drug with other agents, such as dimethyl triazeno imidazole carboxamide (dacarbazine), have yielded higher response rates. Preliminary evidence from the National Cancer Institute suggests that adjuvant chemotherapy in high-grade sarcomas reduces the incidence of recurrences.[1] In a group of 43 patients with high-grade sarcomas treated at our institute with the combination of adriamycin and dacarbazine, the five-year recurrence rate was 47%, somewhat lower than the historically reported rate. What is more striking, however, is that the recurrences in the treated group were late. Although the recurrence rate in the low-grade tumors not treated with chemotherapy was 27% and their cumulative five-year survival rate was 59%, the recurrence rate of the high-grade tumors treated with chemotherapy was 47% and their five-year survival rate was 61%.[12]

Other than the lungs, soft tissue sarcomas may involve the liver, bones, brain, or subcutaneous tissue. Metastases to the heart are rare. In one report based on autopsies of patients who died from metastatic soft tissue sarcoma, there was involvement of the heart in 25% of cases.[18] Clinical evidence of involvement of the heart in vivo in the presence of other metastatic lesions is occasionally seen. Involvement of the heart, clinically detectable as the only initial manifestation of metastatic disease, is rare, as pointed out in the article by Steinherz et al.[5] That report serves to underline three points: Selective combination of modalities is currently practiced in the management of soft tissue sarcomas and has resulted in high limb salvage rates with satisfactory local control, as well as improved survival rates. Increased awareness of the existence of these tumors and their metastatic patterns, including the rarer ones, may help in the earlier diagnosis of the primary tumor and the metastatic recurrence with earlier institution of treatment. Some cases reported in the literature illustrate a reduction in the rate of metastatic recurrence of high-grade sarcomas with adjuvant chemotherapy and in those who relapse, a delay in the appearance of recurrence.[5] This observation suggests that, in order to assess accurately the impact of the combination of modalities in the management of soft tissue sarcomas, the ten-year results should be awaited.

CONSTANTINE P. KARAKOUSIS, MD, PhD
Clinical Professor of Surgery
SUNY Health Science Center
at Buffalo

Chief, Soft Tissue-Melanoma
Bone Service
Roswell Memorial Institute
Buffalo, NY 14263

1. Rosenberg SA, Suit HD, Baker LH: Sarcomas of soft tissues, in DeVita VT Jr, Hellman S, Rosenberg SA (eds): *Cancer: Principles and Practice of Oncology*, ed 2. Philadelphia, JB Lippincott Co, 1985, pp 1243–1292.
2. Hajdu SI: Differential diagnosis of soft tissue and bone tumors. Philadelphia, Lea & Febiger, 1986.
3. Miettinen M, Lehto VP, Vartio T, et al: Epithelioid sarcoma. Ultrastructural and immunohistologic features suggesting a synovial origin. *Arch Pathol Lab Med* 1982; 106:620–623.
4. Farris KB, Reed RJ: Monophasic, glandular, synovial sarcomas and carcinomas of the soft tissues. *Arch Pathol Lab Med* 1982; 106:129–132.
5. Steinherz LJ, Rosen G, Steinherz PG, et al: Isolated cardiac metastatic recurrence of epithelioid sarcoma after two and a half disease-free years. *NY State J Med* 1987; 87:231–233.
6. Wright PH, Sim FH, Soule EH, et al: Synovial sarcoma. *J Bone Joint Surg (Am)* 1982; 64:112–122.
7. Zito RA: Synovial sarcoma: An Australian series of 48 cases. *Pathology* 1984; 16:45–52.
8. Krall RA, Kostianovsky M, Patchefsky AS: Light and electron microscopic studies of monophasic synovial sarcoma. *Lab Invest* 1980; 42:130.
9. Russell WO, Cohen J, Enzinger F, et al: A clinical and pathological staging system for soft tissue sarcomas. *Cancer* 1977; 40:1562–1570.
10. Abbas JS, Holyoke ED, Moore R, et al: The surgical treatment and outcome of soft-tissue sarcoma. *Arch Surg* 1981; 116:765–769.
11. Shiu MH, Castro EB, Hajdu SI, et al: Surgical treatment of 297 soft tissue sarcomas of the lower extremity. *Ann Surg* 1975; 182:597–602.
12. Karakousis CP, Emrich LJ, Rao U, et al: Feasibility of limb salvage and survival in soft tissue sarcomas. *Cancer* 1986; 57:484–491.
13. Karakousis CP: Analysis of failures in the surgery of soft tissue tumors, in *Abstracts of Lectures, Symposia and Free Communications*. Budapest, Hungary, 14th International Cancer Congress (August 21–27, 1986), vol 1, p 25.
14. Rosenberg SA, Tepper J, Glatstein E, et al: The treatment of soft-tissue sarcomas of the extremities: Prospective randomized evaluation of (1) limb-sparing surgery plus radiation therapy compared with amputation and (2) the role of adjuvant chemotherapy. *Ann Surg* 1982; 196:305–315.
15. Eilber FR, Mirra JJ, Grant TT, et al: Is amputation necessary for sarcomas? A seven-year experience with limb salvage. *Ann Surg* 1980; 192:431–438.
16. Vezeridis MP, Moore R, Karakousis CP: Metastatic patterns in soft-tissue sarcomas. *Arch Surg* 1983; 118:915–918.
17. Huang MN, Edgerton F, Takita H, et al: Lung resection for metastatic sarcoma. *Am J Surg* 1978; 135:804–806.
18. Hallahan D, Vogelzong NJ, Simon M: A high frequency of symptomatic cardiac metastases in metastatic soft tissue sarcoma. *Proc Am Soc Clin Oncol* 1985; 4:140.

RESEARCH PAPER

Nosocomial pneumonias in a community teaching hospital

DAVID S. PRATT, MD; LORANN STALLONES, PHD; NANCY JOHNSON, RN; LINDA CHIARELLO, RN; JOHN J. MAY, MD

ABSTRACT. Nosocomial pneumonia continues to be a serious complication of hospitalization. Small hospitals with relatively low patient volumes have seldom reported their rates of pneumonias. We reviewed one year's experience in a 183-bed facility to evaluate the rate of pneumonia.

Sixty-six episodes of pneumonia occurred in 63 patients. Twenty-two (35%) patients with pneumonia died. Men were more often affected than women. While many uncommon operations were performed, only craniotomy was associated with a higher than expected infection rate (13 episodes per 100 operations, 13%).

The rate of occurrence of nosocomial pneumonia in this institution is slightly higher than that reported elsewhere (1.0% of admissions). Fatality rates were also higher. Less common procedures did not result in more morbidity than is reported from larger institutions in the United States. Differences in morbidity from national means were not statistically significant.

(NY State J Med 1987; 87:199–202)

When illness necessitates hospitalization, patients and physicians alike assume that the hospital will provide a safe setting for medical care. At present, there is ample proof that hospitalization, while generally safe, does have nosocomial and iatrogenic risks.[1,2] Pneumonia that develops while a patient is in the hospital represents one such danger. Nosocomial pulmonary infection develops in 0.4–5% of all persons who enter the hospital.[3] The gravity of this complication is supported by fatality rates approaching 50% in some reports.[4-6] The majority of papers in the literature that address nosocomial pneumonias draw on pooled information from a number of hospitals or from individual, large urban teaching facilities.[7,8] The study reported here was carried out in a small community teaching hospital that offers a range of medical and surgical services. The study was designed to describe the distribution of nosocomial pneumonia episodes in such a hospital and to assess the comparability of this experience with information from large national surveys. In light of recent reports suggesting that low patient volume had an adverse impact on the outcome of medical and surgical hospital stays,[9,10] we also addressed the issue of surgical nosocomial pneumonias. This retrospective survey is based on one year's experience with such pneumonias.

PATIENTS AND METHODS

The hospital is a 183-bed facility in rural New York which serves as a secondary and tertiary facility for a population of about 100,000 persons. There are training programs in medicine, surgery, and obstetrics and gynecology. All basic and tertiary services, including cardiac catheterization, are provided except for cardiac surgery, burn care, and organ transplantation.

The charts of all patients identified through the hospital infection control program as having had a lower respiratory tract infection during a one-year period (1980–1981) were reviewed. Lower respiratory tract infections include bronchitis, empyema, and pneumonia. The hospital's surveillance system is an active one, using nurse-specialists to search out all nosocomial infections. Cases are sought by reviewing microbiologic reports, interviewing head nurses, and going over the nurses' medication schedules. This experience and that of others show this method to be superior to reviewing ICD-A-coded discharges.[11] To be included in the study, patients had to have a positive chest roentgenogram (with new or progressive infiltrates) and at least one of the following: leukocytosis (white blood cell count (WBC) $\geqq$ 10,000/mm^3), a fever (temperature $> 38°C$), purulent sputum, or pathogenic organisms seen in or isolated from the sputum.

At the time the diagnosis of pneumonia was made, the following data were collected: temperature, white blood cell count, chest film findings, sputum characteristics, and Gram's stain and culture results. Additional information was obtained concerning underlying illnesses and outcome.

Information collected retrospectively from the charts included age, sex, admission and discharge dates, surgical procedure (date and type), and the use of assisted mechanical ventilation (AMV) and its indications and duration.

Patients were classified as immunocompromised if they met any of the following criteria: congenital immune system defects, profound neutropenia (WBC $\leqq$ 1,500/mm^3), immunosuppressive therapy in progress (chemotherapy or radiation), immunosuppressive malignancy or infection, malnutrition, or diabetes mellitus in poor control. (No patients with acquired immunodeficiency syndrome (AIDS) were treated during the study period.)

Data on total admissions, total number of patients on ventilators, and admissions by service were all obtained for the study period. All operations performed during that time were identified, reviewed, and divided into subspecialty categories. The frequency of highly specialized procedures such as vascular surgery, thoractomy, and craniotomies was also recorded.

The cases reported for general medicine include only internal

---

From the Departments of Medicine (Drs Pratt and May), Medical Research (Dr Stallones), and Infection Control (Ms Johnson and Ms Chiarello), Mary Imogene Bassett Hospital, Cooperstown, NY.

Address correspondence to Dr Pratt, Department of Medicine, Mary Imogene Bassett Hospital, Cooperstown, NY 13326.

TABLE I. Admissions, Cases, and Rates for Nosocomial Infection by Service

	Admissions	Cases	Rate/100 Admissions
Medicine	2,244	30	1.3
Surgery*	1,822	33	1.8
Other†	1,996	0	—
Totals	6,062	63	1.0

* Includes urology, neurosurgery, ENT, ophthalmology, general surgery.
† Includes pediatrics, psychiatry, obstetrics and gynecology.

TABLE II. Age and Sex Distribution of Patients with Nosocomial Pneumonia

	All Cases	Surgical Cases	Medical Cases
Men	66 (± 17.8)* (n = 45)	69 (± 15.3) (n = 23)	63 (± 19.8) (n = 22)
Women	73(± 15.9) (n = 18)	72.7 (± 18.9) (n = 10)	74 (± 12.2) (n = 8)
Ratio M:W	2.5:1	2.3:1	2.75:1

* Mean age (standard deviation of mean).

medicine admissions; likewise, surgical cases do not include obstetrics and gynecology, but do include other surgical specialties. In comparing these data to those of other groups, these distinctions should be borne in mind.

Statistical comparisons of infection rates were performed using the chi-square analysis.

RESULTS

During the study period, 6,062 patients were admitted to the hospital (Table I). The hospital infection control surveillance system identified 99 patients who had acquired a lower respiratory tract infection. When these cases were reviewed using the stated criteria, 66 episodes of nosocomial pneumonia were identified in 63 patients. There were 36 episodes that did not meet the working definition, as stated above. The majority of cases that were excluded either did not have pneumonic infiltrates (49%), or the pneumonia was acquired in the community or at another hospital (30%). Twelve percent had no infiltrates at all, and the rest (9%) lacked the required clinical signs.

The overall rate for nosocomial pneumonia was 63 cases/6,062 admissions (1.0%) (Table I). The average age of the affected patients was 68 years, with a range from 20 to 95 years (Fig 1). Age distributions for men and women were similar. The attack rate for men was 15.5/1,000 and for women was 5.7/1,000; this difference is significant ($p < 0.001$).

Twenty-two (35%) of the patients died during the observation period of the following causes: cardiac dysfunction (seven patients), respiratory arrest (five patients), pneumonia (five patients), cancer (three patients), brain trauma (one patient), and perforated duodenum (one patient).

Thirty-three (52%) of the 63 patients sustained respiratory tract infections in the immediate postoperative period (up to ten days after operation). These 33 patients represent an infection rate of 1.8/100 admissions to surgery. The men in this group were slightly younger than the women and outnumbered them by 2.3 to 1 (Table II). Twenty-two (66.7%) of these patients with immediate postoperative pneumonia were intubated beyond their stay in the recovery room and required assisted mechanical ventilation. All were cared for in the intensive care unit after surgery. Eight (24.2%) patients were immunosuppressed.

The attack rates of nosocomial infections in patients who underwent surgery are shown in Figure 2. General surgery produced the largest number of nosocomial pneumonias, but because of the number of operations performed, this category had a low overall infection rate (1.5%). Conversely, only 31 thoracic surgical operations were performed during the study period, with two nosocomial pneumonias developing, giving an infection rate of 6.4%. Figure 2 also shows that neurosurgical and orthopedic procedures both had attack rates of greater than 3%. The single most common operation associated with nosocomial pneumonia in this study was craniotomy. Of the 33 affected surgical patients, nosocomial pneumonia developed in six patients during the postoperative period. During the study period, a total of 47 craniotomies were performed. This gives a pneumonia rate of 13% following craniotomy. The pneumonia rate in patients undergoing craniotomy was significantly greater than that for all others in the study ($p < 0.001$). Craniotomies contributed 18% of all the nosocomial pneumonias in the surgical series. If craniotomies were removed from the surgical series, the overall rate of nosocomial pneumonias on the surgical service would drop to 1.5%.

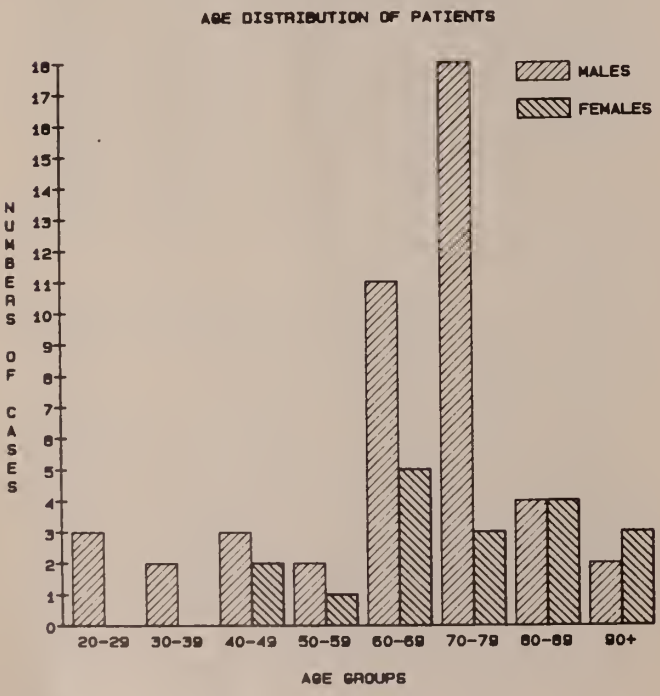


FIGURE 1. The age and sex distribution of 63 medical and surgical patients with nosocomial pneumonias.

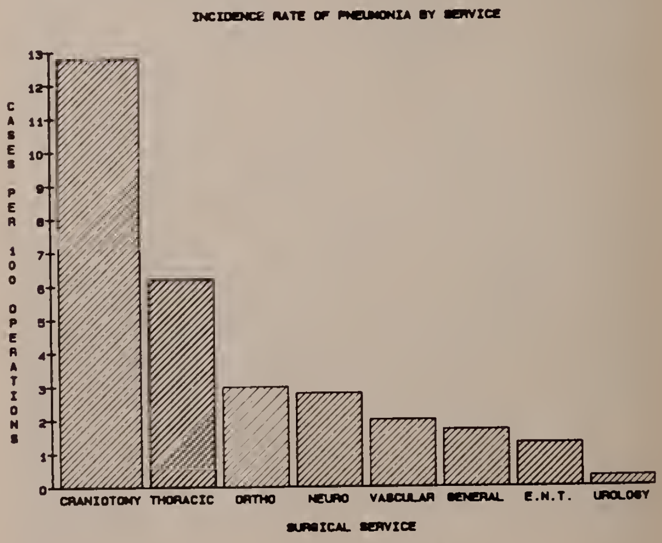


FIGURE 2. The attack rates of nosocomial pneumonia for surgical patients. Note that the rate for craniotomy alone is contrasted with rates for all thoracic, orthopedic, neurosurgical, vascular, general surgical, otolaryngologic, and urologic procedures.

There were eight fatal episodes of nosocomial pneumonia in the surgical group (24.2%). Nearly 90% of these fatal cases involved intubation and a stay in the intensive care unit, suggesting serious surgical illness. Of note is the fact that only two of the deaths in the surgical series occurred in immunocompromised patients.

Thirty (47.6%) of the affected patients were on the medical service. These cases represent an attack rate of 1.3/100 (1.3%). Table II shows that the men in this group were younger than the women and outnumbered them by 2.75 to 1. Ten of these patients with pneumonia, or 33%, were cared for in the intensive care unit; eight, 27%, were immunosuppressed; and seven patients, 23%, needed AMV. When the medical patients were evaluated for underlying problems that might predispose them to acquiring pneumonia, 26% were found to have had either a cerebrovascular accident or another medical problem that impaired their level of consciousness. Thirty-three percent had evidence of significant cardiac compromise presenting as congestive heart failure, myocardial infarction, or coronary artery disease requiring medication. Malignancy as an underlying problem was present in 16% of the medical patients in this series.

Thirteen of the medical patients with a hospital-acquired pneumonia had a fatal outcome (43.3%). Among those who died, seven patients (53.8%) were immunosuppressed, four had endotracheal tubes (30.7%), and three (23%) were maintained on AMV. While the general medicine division had more pneumonia deaths than the surgical division, the difference was not statistically significant (p = 0.19).

Pathogenic organisms isolated from the patients with pneumonia and the results of diagnostic Gram's stains are shown in Table III. Of the total of 66 episodes of pneumonia, 55 cases (83%) were cultured, and seven had Gram's stains only (11%), while the remaining four (6%) had neither culture nor Gram's stain. Gram-negative organisms were predominant or were found exclusively in 26 episodes (39%). Gram-positive organisms were felt to have caused the pneumonia in 21 instances (32%). The remaining 19 cultures, or 29%, either were untested, showed normal flora, or showed no growth. Among the 22 patients who died, nine (41%) had cultures with only a gram-negative organism, two (9%) had cultures with only a gram-positive organism, and four had a combination of both. Normal mouth flora were found in five of the patients who died. Two fatal cases had no culture results.

Thirty-one patients with nosocomial pneumonia were receiving or had been receiving mechanical ventilation (49.2%). During the study period a total of 134 patients at this hospital went on AMV, giving an infection rate of 23% for patients needing respiratory support. This amounts to about 20 times the infection rate for all patients. This increased rate for patients on ventilators was significant ($p < 0.001$). Gram-negative organisms outnumbered gram-positive organisms by three to one in patients with pneumonia on AMV. Patients who did not need AMV had an equal number of gram-positive and gram-negative organisms isolated during their illness. Although this difference in frequency of gram-negative isolates is striking, it was not statistically significant at the 0.05 level. Men outnumbered women on assisted mechanical ventilation by 3.4 to 1; the age distribution, however, was not different from that of the population as a whole. The case fatality rate of those on assisted ventilation was 32%. This death rate is comparable to that of the total population with nosocomial pneumonia.

TABLE III. Principal Pathogenic Organism Isolated from Sputum* of Patients with Nosocomial Pneumonia (June 1–May 31)

	No. of Cases with Isolates	%
Klebsiella†	7	11
Streptococcus	7	11
Staphylococcus	6	9
Haemophilus influenzae†	5	8
β hemolytic *Streptococcus*	4	6
Serratia†	3	5
Neisseria†	2	3
Normal mouth flora	12	18
Multiple organisms	16	23
No culture	4	6
Totals	66	100%

* All sputum samples were judged adequate either by cellularity (few or no epithelial cells) or by the method in which they were obtained (ie, suctioned from endotracheal tube).

† Gram-negative organisms.

DISCUSSION

The information presented above shows that the overall pneumonia rate (1.0%) was higher than that described in other series.[1,7] These reported rates generally range from a low of 0.4% to a high of 0.94%.[12,13] The large, well-organized SENIC study suggests that the mean rate in the United States is about 0.8%.[1]

When compared to the SENIC mean, the rate found in this report translates into about 15 "excess" cases of pneumonia. While impressive, this difference is not statistically significant (p = 0.18). Direct comparison of the SENIC series (388 hospitals, 169,000 patients) with this one-year review fails to take into account yearly variation around the true mean. While we are pleased with this hospital's strong surveillance efforts, and there are a large number of acutely ill transfer patients, we do not find evidence for true excess morbidity in this series.

Surgical patients appeared to have required more intensive attention than medical patients. This can be seen by comparing the proportion of surgical patients needing AMV (66%) to the proportion of medical patients needing AMV (23%). Surgical patients were cared for in the intensive care unit almost twice as often as medical patients. Since the proportions for immunocompromise were about equal for both groups, one can infer that the surgical patients were "sicker" after surgery and required a more intensive level of care. These intensity factors are reflected in the SENIC data as well.[7]

Others have reported that nosocomial pneumonia appears to be the most common, fatal, hospital-acquired infection.[14] In this series, nearly 35% of those patients with nosocomial pneumonia died. Death attributable to these infections and respiratory compromise amounted to 48% of the total. These case fatality rates are similar to those reported by Cross and Roup[8] from a large military hospital.

Assisted mechanical ventilation is clearly associated with an increased risk of pneumonia. When we reviewed fatal pneumonias, the death rate was 32% for patients on AMV. While this fatality rate is not greater than that for the group as a whole, pneumonias were clearly more common in the AMV group.

This study shows that while pneumonia is more likely to develop in surgical patients, a fatal outcome is more common among medical patients. The patients in fatal medical cases were more often immunocompromised than those in surgical cases, whereas surgical cases were most often associated with the use of the intensive care unit and assisted mechanical ventilation. The fatal pneumonias were more often associated with gram-negative than gram-positive organisms, an experience shared by Stevens et al,[15] who found poor outcomes for patients with gram-negative pneumonias in intensive care units. Stevens and coworkers further found that *Pseudomonas* pneumonia had a particularly poor prognosis, with a case fatality rate greater than 70%. We experienced three episodes of *Pseudomonas* pneumonia, with one death. A recent report of fatal nosocomial pneumonias points out that cancer was often associated with fatal cases.[14] Of the 21 deaths reported here, seven had an active malignancy at the time of death (33.3%). This rate is comparable to that reported by Gross et al[14] and supports the contention that patients with cancer who acquire nosocomial pneumonia may be at increased risk of death.[14]

Perhaps the most interesting results were from the neurosurgical service. In the period under study, 47 craniotomies were performed, and pneumonia developed in 13% of the patients. This high attack rate reflects not only the effects of the operation, which is often long, requiring a great deal of anesthesia, but also the need for assisted mechanical ventilation in the postoperative period. Poor breathing drives coupled with sustained mechanical ventilation make these patients prone to pooling of secretions and atelectasis. If the craniotomy-associated pneumonias were removed from the neurosurgical group, there would have been only one case of nosocomial pneumonia in the neurosurgical service during the study year. While pneumonia complicating altered consciousness is well recognized,[16] little additional data on the risk of craniotomy per se are available to support these findings.

We conclude from this study that this community teaching hospital had a nosocomial pneumonia rate roughly comparable to that of the nation at large during the study period. Despite improved antibiotic therapy, assisted mechanical ventilation, and modern monitoring technology, pneumonia rates remain unacceptably high. Vigilance for increased risk and scrupulous attention to asepsis remain cornerstones of good care for all medical and surgical patients. In a recent paper,[17] one of the authors pointed out that 16% of pneumococcal bacteremias occurred in patients with nosocomial pneumonia. This group had a 65% death rate. The availability of an efficacious pneumococcal polysaccharide vaccine makes it possible to reduce the morbidity and mortality of some nosocomial pneumonias through immunization.

Acknowledgments. The authors thank the medical records department, the microbiology laboratory, and the members of the respiratory care staff for their help. In addition, we appreciated the help of G. R. Thompson, MD, and the word processing staff.

REFERENCES

1. Haley RW, Hooton TM, Culver DH, et al: Nosocomial infections in US hospitals, 1975-1976: Estimated frequency by selected characteristics of patients. *Am J Med* 1981; 70:947-959.
2. Foote GA, Schabel SI, Hodges M: Pulmonary complications of the flow-directed balloon-tipped catheter. *N Engl J Med* 1974: 290:927-931.
3. Pierce AK, Sanford JP: Aerobic gram-negative bacillary pneumonias. *Am Rev Respir Dis* 1974; 110:647-658.
4. Graybill JR, Marshall LW, Charache P, et al: Nosocomial pneumonia: A continuing major problem. *Am Rev Respir Dis* 1973; 108:1130-1140.
5. Martin LE, Asher EF, Casey JM, et al: Postoperative pneumonia: Determinants of mortality. *Arch Surg* 1974; 119:379-383.
6. Berger R, Arango L: Etiologic diagnosis of bacterial nosocomial pneumonia in seriously ill patients. *Crit Care Med* 1985; 13:833-836.
7. Hooton TM, Haley RW, Culver DH, et al: The joint associations of multiple risk factors with the occurrence of nosocomial infection. *Am J Med* 1981; 70:960-970.
8. Cross AS, Roup B: Role of respiratory assistance devices in endemic nosocomial pneumonia. *Am J Med* 1981; 70:681-685.
9. Kennedy RH, Kennedy MA, Frye RL, et al: Use of the cardiac-catheterization laboratory in a defined population. *N Engl J Med* 1980; 303:1273-1277.
10. Luft HS, Bunker JP, Enthoven AC: Should operations be regionalized? The empirical relation between surgical volume and mortality. *N Engl J Med* 1979; 301:1364-1369.
11. Kennedy GT, Stern MP, Crawford MH: Miscoding of hospital discharges as acute myocardial infarction: Implications for surveillance programs aimed at elucidating trends in coronary artery disease. *Am J Cardiol* 1984; 53:1000-1002.
12. Centers for Disease Control: National Nosocomial Infection Study Report, Annual Summary 1978, issued March 1981.
13. Wenzel RP, Osterman CA, Hunting KJ, et al: Hospital-acquired infections. I. Surveillance in a university hospital. *Am J Epidemiol* 1976; 104:645-651.
14. Gross PA, Neu HC, Aswapokee P, et al: Deaths from nosocomial infections: Experience in a university hospital and a community hospital. *Am J Med* 1980; 68:219-223.
15. Stevens RM, Teres D, Skillman JJ, et al: Pneumonia in an intensive care unit. A 30-month experience. *Arch Intern Med* 1974; 134:106-110.
16. Huxley EJ, Viroslav J, Gray WR, et al: Pharyngeal aspiration in normal adults and patients with depressed consciousness. *Am J Med* 1978; 64:564-568.
17. Federson DS, Chiarello LA: Previous hospital care and pneumococcal bacteremia: Importance for pneumococcal immunization. *Arch Intern Med* 1983; 143:885-889.

REVIEW ARTICLE

The role of building construction and ventilation in indoor air pollution

Review of a recurring problem

ROBERT J. MCCUNNEY, MD

Air pollutants in homes and offices continue to arouse concern. Tobacco smoke, formaldehyde, and radon are among many materials that have received attention in both the scientific and lay literature. Energy conservation measures such as improved insulation, reduced air exchange, and fuel switching have resulted in some buildings becoming so air tight that physical comfort and health are now critically dependent on effective air handling systems. Ventilation that is appropriate to the size of a room, the number of inhabitants, and potential contaminants is essential to prevent unfavorable reactions among susceptible people.

The purpose of this report is to review the common types of health effects that can occur from indoor air pollutants in both home and office environments, the source of such contaminants, and the control of these pollutants. Greater attention will be paid to the more serious afflictions that affect people in indoor environments.

TYPES OF BUILDING-RELATED HEALTH EFFECTS

Many adverse health effects have been attributed to poor indoor air quality, including hypersensitivity pneumonitis, infections, a variety of nonspecific reactions to building materials, and epidemic psychogenic illness.

Hypersensitivity Pneumonitis. Hypersensitivity pneumonitis (HP) refers to interstitial lung disease caused by organic dusts or by aerosols that originate in air handling systems. The most common agents responsible for HP are thermophilic organisms of the micropolyspora and thermoactinomyces group.[1] Symptoms usually occur within four to six hours after exposure to the offending agent and consist of fever, chills, cough, and dyspnea. The chest film, which can be normal, most often shows a diffuse parenchymal infiltrate. The most specific test for HP is a bronchial challenge with the antigen or the implicated water, although such testing is not often practical and can be hazardous. A lung biopsy is usually not necessary. Symptoms should develop within four to six hours after inhalation of the antigen to support a diagnosis of acute hypersensitivity pneumonitis. Gallium scans often demonstrate inflammatory changes; bronchoalveolar lavage can indicate signs of alveolitis. Eosinophilia is usually not present, and atopic individuals do not appear to be at increased risk.

Hypersensivity pneumonitis specifically associated with air conditioning systems was first described in 1970.[2] Among 27 office workers, four became ill secondary to thermophilic actinomyces originating in an air conditioner. Two people experienced acute reactions of fever, chills, dyspnea, and cough, whereas two others developed chronic effects such as cough and shortness of breath, but without fever or systemic illness. Chest film abnormalities consisted of a diffuse nodular infiltrate. Pulmonary function testing revealed a restrictive defect, with impaired diffusion capacity—abnormalities that resolved after avoidance of exposure to the antigen. None of the affected workers were atopic; all had serum precipitins against extracts of the thermophilic actinomyces obtained from the air conditioner.

Following this report, a woman was described in whom pulmonary symptoms developed secondary to an actinomycete (micropolysporo genus) located in the heating and humidification system in her home.[3]

In a much larger outbreak, 48 of 4,023 office workers acquired hypersensitivity pneumonitis because of a contaminated open spray air cooling system.[4] Chest films, diffusion capacity measurements, serum precipitins, and single spirometry, however, proved to be of little diagnostic value. Instead, a history of respiratory and/or systemic symptoms was the most sensitive screening parameter. The most useful diagnostic test was the measurement of forced vital capacity (FVC) before and after work. Affected workers improved after they were removed from the office complex. The prevalence of precipitins was similar in the symptomatic and asymptomatic group, a finding often noted in outbreaks of hypersensitivity pneumonitis.

Extrinsic allergic alveolitis is another term used to define the same symptom complex. In a stationery factory,

---

Dr McCunney is Assistant Professor of Public Health, Boston University School of Public Health, and Medical Director of Goddard Occupational Health Services.

Address correspondence to Dr McCunney, Corporate Medical Director, Cabot Corporation, 950 Winter St, Waltham, MA 02254.

Presented at the Annual Assembly of the Medical Society of the State of New York, June 7, 1985, and at an update on occupational medicine held at the University of Massachusetts Medical Center, March 19, 1986.

some workers became ill as a result of inhalation of a water aerosol contaminated by microorganisms.[5] No specific organism was incriminated, however. Of the 560 people in the factory, 24 experienced a range of symptoms, including chest tightness, chills, and shortness of breath at rest. No chest film abnormalities were noted, although the illness was reproduced by inhalation challenge. All of the affected workers had serum precipitins to contaminated water. Tolerance developed rapidly, but was lost after an absence from the factory of a day or two—a reaction similar to that of byssinosis and metal fume fever.

Extrinsic allergic alveolitis also occurred in nine of 350 workers at the printing floor of a factory.[6] Although a specific agent was not identified, air conditioning units were contaminated with microorganisms. Serum precipitin levels were similar in both symptomatic and asymptomatic people. After the cold water humidification system was switched to steam, symptoms resolved.

A forced-air heating-cooling unit was held accountable for two of 14 clerical workers developing hypersensitivity pneumonitis.[7] In this situation as well, the specific agent was not identified, but pencillium molds were cultured from the air handling system.

Although hypersensitivity pneumonitis is recognized as having occurred in cooling, heating, or humidifying systems in offices, factories, homes, and automobiles,[8-11] its diagnosis can be difficult. In chronic cases, symptoms may develop up to three years from the time of first exposure,[12] serum precipitin levels may not be different than controls, and the offending agent may not be identified. To assist the clinician, guidelines for the uniform diagnosis of this disorder have been proposed (Table I).[7]

Immunocompromised Hosts. Since immunocompromised people have a greater risk of contracting many illnesses, it is not surprising that aspergillosis developed in ten immunocompromised patients because of a contaminated window air conditioner in the hospital.[13] The diagnosis was based on fever, unexplained infiltrates on the chest films, and nasal cultures that showed growth of *Aspergillus*. Dust from a local construction site had apparently lodged in the unit's filter. Any undisturbed surface where dust and lint accumulate can serve as a source of viable *Aspergillus* spores.

In two medical wards, 20 of 24 immunocompromised patients became septic as a result of *Actinobacillus* that originated in cold air humidifiers.[14] The organism was isolated from intravenous catheters and all six humidifiers.

Humidifier Fever. Humidifier fever refers to a self-limited illness that consists of fever, chills, headache, and cough, secondary to contaminated air handling systems. Tolerance seems to develop, but is lost just as readily. Diagnostic studies are nonspecific. Treatment is symptomatic since the offending agent is rarely identified. In contrast to hypersensitivity pneumonitis, which has a low attack rate (less than 1%), humidifier fever generally affects a larger proportion of those exposed (25–40%). The diagnosis, which can be difficult because of its similarities to acute hypersensitivity pneumonitis, is often based on the study of groups of people. After changes are instituted in some aspect of the ventilation system, symptoms and other health complaints usually resolve.

In a rayon processing factory, 20 of 50 office workers suffered fever and malaise that was attributed to a humidifier that disseminated microbial spores and cysts.[15] Although chest films revealed no abnormalities, pulmonary function studies suggested mild airway obstruction. After the humidifier was cleaned and a new pre-filter installed, no new cases developed. Antigens produced by microbial action on the rayon fiber were considered responsible.

A similar outbreak of humidifier fever occurred in 26 of 50 people employed in one area of a large industrial complex.[16] A water humidifier system was held accountable for fever, chills, headache, cough, and dyspnea. After the humidification system was removed, symptoms resolved. Although the agent that causes humidifier fever has yet to be identified, protozoa are considered responsible; thermophilic vulgaris, *T Candidas*, thermotolerant bacteria, penicillium species, and *Naegleria gruberi* have also been implicated.[1]

Other outbreaks of respiratory illness have been reported in office workers.[17] Among 325 workers, 40% experienced headaches, myalgias, fever, chills, and cough soon after the heating and air conditioning system was started. Numerous fungi and mites were isolated from the unit, but no single agent was held responsible. Serologic testing of precipitins was inconclusive, but diffusion capacity measurements were lower in affected workers than in controls.

Infections. The most notable infection resulting from indoor air pollution is that caused by strains of *Legionella pneumophila*, namely legionnaires' disease and Pontiac fever. Legionnaires' disease is a multisystemic disorder that can affect the lungs, liver, kidney, gastrointestinal tract, and central nervous system. The attack rate ranges from 0.1–5%; 15–20% of those infected die from the illness.[18] Person-to-person spread does not appear to occur; the only proven route of transmission is through contaminated aerosols that usually originate in air handling systems. *Legionella pneumophila* survive extremely well in water; in fact, organisms have remained viable in tap water for as long as a year.[19]

Legionnaires' disease is diagnosed by the isolation of the agent from the blood, lung tissue, respiratory secre-

TABLE I. Guidelines for the Diagnosis of Hypersensitivity Pneumonitis (HP)*

1. Symptoms and signs of HP that occur consistently within several hours of exposure to airborne, respirable-size antigens. Relief of symptoms following removal from exposure.
2. A greater prevalence of positive humoral and/or cell-mediated immunological responses among cases of HP than among suitably selected controls to antigens collected by air sampling from the environments occupied by cases and controls.
3. A greater prevalence of objective responses (eg, abnormal pulmonary function) to environmental or workplace inhalation challenge among informed, consenting volunteer cases than among suitably selected, informed, and consenting volunteer controls.
4. Demonstration of a T-lymphocyte-predominant alveolitis by bronchoalveolar lavage.
5. Lung biopsy evidence of granulomatous inflammation of parenchymal, bronchiolar, and alveolar areas. These areas of the lung may also be identified by staining with specific immunofluorescent antibodies directed against the suspected airborne antigens.

* Adapted from Bernstein et al.[7]

tions, or pleural fluid, or by the demonstration of a significant rise in serum antibody during convalescence. Three types of microbiologic tests are available, including direct fluorescent antibody testing, serologic testing, and culture. Although the direct fluorescent antibody test is highly sensitive (90%), its specificity suffers from false positive reactions due to *Bacteriodes fragilis*, *Pseudomonas fluorescens*, and *Pseudomonas alkalgenes.* Treatment with erythromycin reduces both the fatality and morbidity of the disease.

Legionnaires' disease was first reported in 1977, following an epidemic of 182 cases (25 fatal) that occurred at a convention in Philadelphia.[20] The disease, however, has been shown by retrospective review to have originally occurred in 1947. Other outbreaks have occurred,[21] including an episode at a Memphis hospital in which 39 patients contracted the illness as a result of a contaminated auxiliary air conditioning cooling tower.[22] Although the device had been used for only one month, its water had not received appropriate chemical treatment.

Hospital potable water has also been implicated in the transmission of *L pneumophila.* In a number of settings, acquisition of the disease has ceased or been reduced following treatment of the water system.[23-27] In one hospital, however, an initial investigation suggested potable water as the source, but a cooling tower was later held accountable.[28] The authors emphasized the importance of a thorough environmental/epidemiologic investigation before the institution of certain remedies, such as higher water temperatures (because of the potential for burns) and hyperchlorination (because of the risk of corroding the plumbing system). A major complication in diagnosing legionnaires' disease is the ubiquitous nature of the organism. *Legionella pneumophila* is regularly isolated in cooling towers and evaporative condensers, in the absence of illness.[29-32]

Pontiac fever, also caused by *L pneumophila*, is characterized by the acute onset of fever, chills, and myalgias—without pulmonary involvement. Originally described in 1968, the disorder is usually due to contaminated air conditioning systems.[33] The incubation period averages about 36 hours, in contrast to that of legionnaires' disease, which is usually seven to ten days. Since Pontiac fever is a self-limited infection of two to seven days' duration, treatment is rarely necessary. Its pathophysiology remains unclear—no differences have been noted between the strains of *L pneumophila* that cause legionnaires' disease and those that cause Pontiac fever.

Pontiac fever has also been associated with whirlpool use.[34] Among 23 members of a church group, 14 developed fever, myalgias, and headache within 4–38 hours after using the same whirlpool at a health club. These symptoms were similar to previously described outbreaks of Pontiac fever.[33,35-37] A blocked vent on the roof adjacent to the air conditioning unit was noted.

Specimens taken from the condensate pan yielded *L pneumophila* (serogroup 3). Evidence supporting the diagnosis included a high proportion of seroconversion to *L pneumophila* among six patients, high convalescent titers to the organism, and isolation of the organism from the whirlpool. *L pneumophila* has apparently never been isolated from patients with Pontiac fever. Diagnosis is usually made retrospectively with the use of microbial sampling and epidemiologic review.

Nonspecific Reactions. Dermatitis and mucous membrane irritation have been caused by carbonless copy paper,[38] fiberglass, and mineral wood.[39-41] Itching of exposed skin, which is relieved by showering, is a characteristic finding. Detergent residues left in carpets and formaldehyde have also led to mucous membrane irritation, sore throat, and conjunctivitis. Headache and fatigue, the most common symptoms associated with indoor health complaints, are most often associated with inadequate ventilation.

Allergic reactions have also been provoked after exposure to contaminated air conditioners in automobiles.[42] Among 224 people with atopic-type allergies (dust, pollen), 18% showed decrements in peak expiratory flow rate and exacerbation of allergic symptoms on contact with a variety of actinomyces cultured from air conditioners. Respiratory-type allergic reactions have been initiated by inhalation of fungi that contaminate other types of air conditioners.[43,44]

Epidemic Psychogenic Illness. Epidemic psychogenic illness, also known as mass hysteria, refers to a constellation of symptoms that develop in a group of people who are under some type of physical or emotional stress. Concern almost invariably focuses on an apparent toxic agent. Consistent features are outlined in Table II.[45] The following examples demonstrate common elements of the disorder.

In an evaluation of 29 school children in whom nausea, abdominal pain, and shortness of breath developed, m-butylbenzene sulfonamide was detected in the urine of many of those who claimed to be ill. This substance, a phenol alcohol derivative found in pesticides, was thought to be the cause of the disorder, until it was also detected in the urine of healthy volunteers.[46] The toxic substance proved to be an artifact secondary to the plastic urine container, since samples taken in glass containers showed no evidence of the compound. Keeping affected persons together and continuing the assembly despite persisting illness, in conjunction with attention focused on ambulances and fire engines, appeared to enhance the symptoms. In similar settings, concern has inadvertently been focused on other red herrings, including toxic gases,[47] food,[46] and nuclear fallout.[48]

In an electronics component manufacturing plant, more than 46% of employees reported lightheadedness, headache, and fatigue.[49] Diesel fumes that entered the production area were considered responsible, until an investigation conducted by the National Institute for Occu-

TABLE II. Mass Hysteria: Characteristics

Absence of laboratory results and physical findings from a specific organic cause
Preponderance of illness in girls or women
Benign morbidity, often with rapid spread followed by rapid remission of symptoms
Apparent transmission of illness by sight or sound
Presence of hyperventilation or syncope
Relapse of illness in the setting of the original outbreak
Evidence of unusual physical or psychological stress

pational Safety and Health led to the assessment of mass psychogenic illness. Only female production workers were affected; nearly 60% became ill after hearing of symptoms experienced by other workers. The investigators acknowledged that psychogenic illness does not imply a psychiatric disorder, or that the symptoms were imaginary. In contrast, the syndrome was described as representing a normal psychophysiologic response to a stressful environment.

The term "mass hysteria" has been claimed to be both a pejorative term towards women and an excuse for incomplete investigation of low levels of environmental pollutants.[50] The basis for the authors' contention, however, at least that referring to low level contamination, was later refuted.[51] Epidemic psychogenic illness has been proposed as a more appropriate term.[52]

SOURCES OF INDOOR POLLUTANTS

Indoor pollutants can arise both outside and inside any building. Within a building, contaminants can be generated from building materials, furnishings, insulation, maintenance procedures, and occupants, both human and animal. Outside the building, car and diesel exhaust from a parking lot or loading dock can be the source of health complaints. Nearby construction and demolition can generate hazards such as pathogenic organisms and asbestos, among others. Fungi, such as *Aspergillus*, can be found in decaying organic matter; these opportunistic organisms are ubiquitous and are among the major organisms in the environment.[53]

Sources of organic contaminants in closed office spaces[54] can be categorized as follows: new building materials, aged building materials, wet process copiers, tobacco smoke, building maintenance products. New building materials are a source of organic contaminants because they often contain residual solvents and other compounds left after the process of manufacture and installation.

A committee of the National Research Council[55] addressed the types of indoor pollutants, their respective health effects, means of control, and areas ripe for further investigation. The major agents of concern included radon, formaldehyde, asbestos, tobacco smoke, indoor combustion, microorganisms/allergens, and, possibly, moisture.

Radon. Radon is a gas that diffuses out of radium bearing materials and dissociates initially into polonium, then into bismuth and lead particles (so-called radon daughters). Inhalation of these radon daughters is thought to account for the increased rate of lung cancer among uranium miners.[56] Radon, however, is a natural radioactive gas that contributes to much of the background radiation in the environment. It is emitted by any material that contains radium 226, including concrete, brick, stone, and soil.[57] The actual impact on one's health secondary to low-level, long-term exposure to radon is unclear, although the agent has been considered responsible for a large proportion of lung cancer deaths not due to cigarette smoking.[58,59]

Formaldehyde. This chemical is contained in numerous products, including cigarettes, drapes, carpeting, wallboard, cosmetics, shampoos, and even toothpaste. Its primary action as a toxin is as an irritant to mucous membranes, especially those of the eyes, nose, and throat. Asthma and chest discomfort can also occur.[60] The major potential hazard that has been attributed to formaldehyde is risk of malignancy of the nasal cavities. After an animal study demonstrated a dose-related increase in nasal cancer, further studies were warranted. A large-scale epidemiologic study (25,000 people) conducted by the National Cancer Institute, however, failed to demonstrate this finding.

In homes insulated with urea foam formaldehyde—often at levels much lower than recommended occupational limits—headaches, eye irritation, rashes, and nausea have been reported.[61] Formaldehyde can also be emitted from consumer goods, even after installation, especially particle board. In one energy efficient home, formaldehyde levels increased from 120 $\mu g/m^3$ to 240 $\mu g/m^3$ when furniture was added.[54]

Asbestos. The health effects of asbestos have been reviewed in detail elsewhere.[62,63] The main concern regarding low-level exposure to this versatile substance is an increased risk of lung cancer and mesothelioma. In situations in which asbestos is in bonded form and is not capable of becoming airborne, risk is minimal. Asbestos insulation in schools, public and commercial buildings, as well as hospitals, continues to create controversy as to whether it should be removed, covered, or ignored.

Tobacco. Contaminants secondary to cigarette smoking are the subject of considerable controversy. At debate is whether close contacts of smokers are at increased risk of suffering lung cancer or obstructive airway disease. Studies in Japan, Greece, and New Orleans have indicated increased rates of lung cancer among nonsmoking spouses of heavy smokers.[64–66] Non-smokers have also demonstrated increased levels of urinary cotinine when in promixity of smokers.[67]

Because of potential effects from secondary or passive tobacco smoke, some corporations have established policies that prohibit or restrict smoking to certain areas. Various incentives to stop smoking are used, including the offering of smoking cessation programs. The legal system has become involved in various dimensions of the problem, including third party liability and workers' compensation claims. Tobacco smoke has been ruled a toxic substance by a New Jersey federal court. An employer, in turn, was forced to take alternative measures for an employee with medical evidence of extreme sensitivity to tobacco smoke.

According to the American Society of Heating and Refrigeration Engineers, tobacco smoke is one of the most difficult contaminants to control at its source.[68] In fact, the organization recommends a substantial increase in ventilation for rooms where smoking is permitted (20 cubic feet per minute per person versus 5 cubic feet per minute per person).

Indoor Combustion. Kerosene heaters and wood stoves are the source of numerous products of combustion including carbon monoxide, carbon dioxide, oxides of nitrogen, and particulates.

Miscellaneous. Pesticides, floor cleaners, and detergents have all been associated with various health complaints. Organophosphate poisoning has occurred in office workers secondary to chlorpyrifos and methyl carbonic acid.[69]

INVESTIGATIONS BY THE NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH

The National Institute for Occupational Safety and Health (NIOSH) reported the results of 356 investigations of indoor air complaints (not including asbestos-related problems) from 1971 to 1985 (Table III). Nearly half of the problems appeared to be due to inadequate ventilation.

Prior to 1978, only six indoor air quality investigations were conducted. The agency attributed the increase in the number of requests to both improved energy efficiency and greater awareness of toxic substances in home and office environments. The NIOSH experience may not be reflective of the true state of the problem, because the agency is usually consulted only when initial attempts to solve a problem have failed or when complaints persist after initial corrections are made.

Other NIOSH investigations have uncovered problems such as methyl alcohol from spirit duplicators,[70] trinitrofluorenone from laser printers,[71] sulfur dioxide from a heating system,[72] and the development of dermatitis from amines in the heating system[73,74] and chlordane from the use of termite control agents[75,76] (Table IV).

INVESTIGATING A SUSPECTED CASE OF INDOOR POLLUTION

When faced with an outbreak of illness in an indoor setting, it is often instructive to meet with people knowledgeable about the historical aspects of the problem. Methods used in a good medical history can yield information regarding the onset, duration, frequency, and type of health concerns, and whether changes have taken place outside or inside the building. A walk through the facility is essential to assess potential sources of contaminants.

Ultimately, an industrial hygienist is needed to review the ventilation system and to conduct appropriate air sampling. Attention is paid to the percentage of fresh air, the type, if any, of anticorrosive substances in the heating system, and the frequency of ventilation maintenance, especially the cleaning of filters. The astute hygienist determines the number of room air changes per hour, the humidity, and the condition of filters, ducts, and heating equipment. A thorough building audit also addresses the type of insulation, the use of mechanical equipment such as copying machines, and house-cleaning procedures, especially the use of insecticides and cleaning solvents.

After the initial review, attention can be directed toward the potential problem. A questionnaire can then be administered to help assess the need for further diagnostic studies. Some of the people in the affected environment may feel more comfortable, however, if offered the opportunity to discuss their concerns in private with a trained health professional. For those people who have already been evaluated by a physician for health effects that may be related to the indoor environment, review of the medical records is appropriate and can aid in determining the need for microbial analysis of ventilation equipment. In outbreaks of illness, it may be beneficial to encourage individual medical evaluations for those interested. Each situation needs to be assessed on its own merits, however, because levels of resources and unrest vary considerably.

TABLE III. Completed NIOSH Indoor Air Quality Investigations by Problem Type (through December 1985)

Problem Type	Number Completed	Percent Completed
Contamination (inside)	67	19
Contamination (outside)	38	11
Contamination (building fabric)	14	4
Contamination (biological)	19	5
Inadequate ventilation	179	50
Unknown	39	11
Total	356	

TABLE IV. NIOSH Investigations Into Indoor Air Quality

Type	Source
Inside contamination	Copying machines, pesticides, boiler additives, cleaning agents, tobacco smoke, combustion sources
Outside contamination	Motor vehicle exhaust, boiler gases, exhausted air, construction, gasoline spill
Building fabric	Urea formaldehyde foam, particle board, glues/adhesives, fibrous glass, caulking
Biological contamination	Bacteria, fungi, protozoa, microbial products
Inadequate ventilation	Some abnormality in volume or percentage of fresh air, air distribution, temperature, humidity, or filtration system

Most investigations will have led to the cause of the problem at this stage, although others will require further diagnostic work, such as culturing air handling systems for fungi and other microorganisms. Although a standard approach for determining the need for sampling for airborne microbial organisms appears lacking, the literature on sampling techniques has been reviewed.[77] Guidelines to prevent outbreaks of illness due to contaminated air handling systems have been proposed by the Centers for Disease Control[17] (Table V).

CONCLUSIONS

The indoor environment can be the cause of a range of illnesses and adverse health effects. New materials, energy efficiency measures, and inadequate ventilation have all been held accountable for conditions ranging from fatal pulmonary illness to nonspecific symptoms such as headache and fatigue. Health problems secondary to indoor air contaminants can be prevented, however, through efficient ventilation systems that are properly maintained and by careful attention to the health risks associated with certain materials and processes. Most investigations of indoor air complaints that include a review of the affected area, an assessment of the health concerns, and an industrial hygiene audit will uncover the problem. More attention ought to be directed to the indoor environment as a potential source of health problems that are often overlooked as psychosomatic.

The ideal approach to preventing the adverse effects of

TABLE V. Engineering Measures Thought to Prevent the Occurrence of Building-Associated Microbial Contamination

1. Prompt repair of all external and internal leaks.
2. Maintenance of relative humidity below 70%. (At higher levels of humidity, germination and proliferation of fungal spores is enhanced.)
3. Prevention of accumulation of stagnant water under cooling-deck coils of air-handling units (through proper inclination and continuous drainage of drain pans).
4. The use of steam, rather than recirculated water, as a water source for humidifiers in HVAC systems. (Steam sources should not be contaminated with volatile amines.)
5. Replacement of filters in air-handling units at regular intervals. (Filters ideally should have at least a moderate efficiency rating (50% or more) as measured by the atmospheric-dust spot test and be of the extended-surface type; prefilters (eg, roll type) are best used before air passage over the higher-efficiency filters.)
6. Removal, rather than disinfection, of carpets, upholstery, ceiling tiles, and other porous furnishings that may have been grossly contaminated by a potentially harmful substance.
7. Supply of outdoor air into ventilation systems at minimum rates per occupant of at least 20 cubic feet per minute in smoking areas and at least 5 cubic feet per minute in nonsmoking areas.

indoor pollution is through proper ventilation. Although the most effective ventilation criteria are a matter of debate, it is clear that inadequate ventilation, in the absence of any other source of contamination, can cause systemic health effects, especially fatigue. Although symptoms associated with poorly ventilated buildings are indeed troublesome, it is uncertain whether long-term health effects can develop. Preventive maintenance of air handling systems is essential.

REFERENCES

1. Wyngaarden JB, Smith LH: Interstitial lung disease of known etiology, in Wyngaarden JB, Smith LH (eds): *Cecil Textbook of Medicine*, ed 17. Philadelphia, WB Saunders, 1985, pp 417–419.
2. Banaszak EF, Thiede WH, Fink JN: Hypersensitivity pneumonitis due to contamination of an air conditioner. *N Engl J Med* 1970; 283:271–276.
3. Fink JN, Banaszak EF, Thiede WH, et al: Interstitial pneumonitis due to hypersensitivity to an organism contaminating a heating system. *N Engl J Med* 1971; 74:80–83.
4. Arnow PM, Find JN, Schlueter DP, et al: Early detection of hypersensitivity pneumonitis in office workers. *Am J Med* 1978; 64:236–242.
5. Friend JA, Gaddie J, Palmer KN, et al: Extrinsic allergic alveolitis and contaminated cooling-water in a factory machine. *Lancet* 1977; 1:297–300.
6. Pickering CA, Moore WK, Lacey J, et al: Investigation of a respiratory disease associated with an air conditioning system. *Clin Allergy* 1976; 6:109–118.
7. Bernstein RS, Sorenson WG, Garabrant D, et al: Exposures to respirable, airborne, penicillium from a contaminated ventilation system: Clinical, environmental and epidemiological aspects. *Am Ind Hyg Assoc J* 1983; 44:161–169.
8. Fink JN, Banaszak EF, Baroriak JJ, et al: Interstitial lung disease due to contamination of forced air systems. *Ann Intern Med* 1976; 84:406–413.
9. Scully RE: Weekly clinicopathological exercises (Hypersensitivity pneumonitis due to thermophilic actinomycetes). *N Engl J Med* 1979; 301:1168–1174.
10. Solley GO, Hyatt RE: Hypersensitivity pneumonitis induced by Penicillium species. *J Allergy Clin Immunol* 1980; 65:65–70.
11. Kumar P, Marier R, Leech SH: Hypersensitivity pneumonia due to contamination of a car air conditioner [letter]. *N Engl J Med* 1981; 305:1531–1532.
12. Van Assendelft A, Forsén KO, Keskinen H, et al: Humidifier-associated extrinsic allergic alveolitis. *Scan J Work Environ Health* 1979; 5:35–41.
13. Lentino JR, Rosenkranz MA, Michaels JA, et al: Nosocomial aspergillosis: A retrospective review of airborne disease secondary to road construction and contaminated air conditioners. *Am J Epidemiol* 1982; 116:430–437.
14. Edwards JH: Microbial and immunological investigations and remedial action after an outbreak of humidifier fever. *Br J Ind Med* 1980; 37:55–62.
15. Smith PW, Massanari RM: Room humidifiers as the source of actinobacter infections. *JAMA* 1977; 237:795–797.
16. Ganier M, Lieberman P, Fink J, et al: Humidifier lung. An outbreak in office workers. *Chest* 1980; 77:183–187.
17. Outbreaks of respiratory illness among employees in large office buildings—Tennessee, District of Columbia. *MMWR* 1984; 33:506–513.
18. Fraser DW: Legionellosis, in Wyngaarden JB, Smith LH (eds): *Cecil Textbook of Medicine*, ed 17. Philadelphia, WB Saunders, 1985, pp 1516–1519.
19. Skaliy P, McEachern HV: Survival of the Legionnaires' disease bacterium in water. *Ann Intern Med* 1979; 90:662–663.
20. Fraser DW, Tsai TF, Orenstein W, et al: Legionnaires' disease; Description of an epidemic of pneumonia. *N Engl J Med* 1977; 297:1189–1197.
21. Broome CV, Fraser DW: Epidemiologic aspects of legionellosis. *Epidemiol Rev* 1979; 1:1–16.
22. Dondero TJ, Rendtorff RC, Mallison GF, et al: An outbreak of Legionnaires' disease associated with a contaminated air-conditioning cooling tower. *N Engl J Med* 1980; 302:365–370.
23. Fisher-Hoch SP, Bartlett CL, Tobin JO, et al: Investigation and control of an outbreak of legionnaires' disease in a district general hospital. *Lancet* 1981; 1:932–936.
24. Shands KN, Ho JL, Meyer RD, et al: Potable water as a source of Legionnaires' disease. *JAMA* 1985; 253:1412–1416.
25. Helms C, Massanari R, Zeitler R, et al: Legionnaires' disease associated with a hospital water supply system: A cluster of 24 nosocomial cases. *Ann Intern Med* 1983; 99:172–178.
26. Stout J, Yu VL, Vickers RM, et al: Ubiquitousness of Legionella pneumophila in the water supply of a hospital with endemic Legionnaires' disease. *N Engl J Med* 1982; 306:466–468.
27. Best M, Yu VL, Stout J, et al: Legionellaceae in the hospital water-supply: Epidemiological link with disease and evaluation of a method for control of nosocomial legionnaires' disease and Pittsburgh pneumonia. *Lancet* 1983; 2:307–310.
28. Garbe PL, Davis BJ, Weisfeld JS, et al: Nosocomial Legionnaire's disease. Epidemiologic demonstration of cooling towers as a source. *JAMA* 1985; 254:521–524.
29. Politi BD, Fraser DW, Mallison GF, et al: A major focus of Legionnaires' disease in Bloomington, Indiana. *Ann Intern Med* 1979; 90:587–591.
30. Morris GK, Patton CM, Feeley JC, et al: Isolation of the Legionnaires' disease bacterium from environmental samples. *Ann Intern Med* 1979; 90:664–666.
31. Filermann CB, Cherry WB, Orrison LH, et al: Isolation of Legionnaires' disease bacteria from nonepidemic related aquatic habitats. *Journal Appl Environ Microbiol* 1979; 37:239–242.
32. Kurtz JB, Bartlett CL, Newton UA, et al: Legionella pneumophila in cooling tower systems: Report of a survey of cooling towers in London and a pilot trial of selected biocides. *J Hyg* 1982; 88:369–381.
33. Glick TH, Gregg MB, Berman B, et al: Pontiac fever: An epidemic of unknown etiology in a health department. I. Clinical and epidemiologic aspects. *Am J Epidemiol* 1978; 107:149–160.
34. Mangione EJ, Remis RS, Tait KA, et al: An outbreak of Pontiac fever related to whirlpool use, Michigan 1982. *JAMA* 1982; 253:535–539.
35. Fraser DW, Deubner DC, Hill DL, et al: Nonpneumonic, short-incubation-period Legionellosis (Pontiac fever) in men who cleaned a steam turbine condenser. *Science* 1979; 205:690–691.
36. Spitalny K, Vogt R, Witherell L, et al: Legionnaires' disease and Pontiac fever associated with a whirlpool. Presented at the 22nd Interscience Conference on Anti-microbial Agents and Chemotherapy, Miami Beach, Fla, November 1982.
37. Herwaldt LA, Gorman GW, McGrath T, et al: A new Legionella species, Legionella feeleii species nova, causes Pontiac fever in an automobile plant. *Ann Intern Med* 1984; 100:333–338.
38. Marks JG Jr: Allergic contact dermatitis from carbonless copy paper. *JAMA* 1981; 245:2331–2332.
39. Chrostek WJ: *Town Center Associated Building*. Report No. HHE 79-77-605, Cincinnati, National Institute for Occupational Safety and Health, July 1979.
40. Baker D, Fannick N: *Blue Cross of Northeastern New York, Inc.* Slingerlands, New York. HETA 81-058-1037. Cincinnati, National Institute for Occupational Safety and Health, January 1982.
41. Dermatitis of the scalp associated with the installation of ceramic wool fiber (KAO-wool) insulation in a nuclear power station—Ohio. *MMWR* 1981; 30:446–467.
42. Kumar P, Marier R, Leech SH: Respiratory allergies related to automobile air conditioners. *N Engl J Med* 1984; 311:1619–1621.
43. Graveson S: Fungi as a cause of allergic disease. *Allergy* 1979; 34:135–154.
44. Salvaggio JE, Aukrust L: Mold-induced asthma. *J Allergy Clin Immunol* 1981; 68:327–346.
45. Small GW, Borus JF: Outbreak of illness in a school chorus. Toxic poisoning or mass hysteria? *N Engl J Med* 1983; 308:632–635.
46. Pfeiffer PH: Mass hysteria masquerading as food poisoning. *J Maine Med Assoc* 1964; 55:27.
47. Stahl SM, Lebedun M: Mystery gas: An analysis of mass hysteria. *J Health Soc Behav* 1974; 15:44–50.
48. Medalia NZ, Larsen ON: Diffusion and belief in a collective delusion: The Seattle windshield pitting epidemic. *Am Sociol Rev* 1958; 23:180–186.
49. Epidemic psychogenic illness in an industrial setting—Pennsylvania. *MMWR* 1983; 32:287–294.
50. Faust HS, Brilliant LB: Is the diagnosis of 'mass hysteria' an excuse for incomplete investigation of low-level environmental contamination? *J Occup Med* 1981; 23:22–26.
51. Levine RJ: Is the presence of low-level environmental contamination a sufficient excuse for not diagnosing mass hysteria? [letter]. *J Occup Med* 1981; 23:597–599.
52. Alexander RW, Fedoruk MJ: Epidemic psychogenic illness in a telephone operators' building. *J Occup Med* 1986; 28:42–45.
53. Richard JL: Fungi as industrial hazards. *Occup Health Saf* 1979; 48:43–48.
54. Hollowell CD, Miksch RR: Sources and concentrations of organic compounds in indoor environments. *Bull NY Acad Med* 1981; 57:962–977.
55. National Research Council. *Indoor Pollutants*. Washington, DC, National Academy Press, 1981.

56. Archer VEW, Wagoner JK, Lundin FE Jr: Uranium mining and cigarette smoking effects on man. *J Occup Med* 1973, 15:204–211.

57. Budiansky S: Indoor air pollution *Env Sci Tech* 1980; 14 1023–1027

58. Radford EP, Renard KG: Lung cancer in Swedish iron miners exposed to low doses of radon daughters. *N Engl J Med* 1984; 310:1485–1494.

59 Harley NH: Radon and lung cancer in mines and homes [editorial] *N Engl J Med* 1984; 310:1525–1527

60 National Research Council *Formaldehyde and Other Aldehydes* Washington, DC, National Academy Press, 1981.

61. Harris JC, Rumack BH, Aldrich FD, et al: Toxicology of urea-formaldehyde and polyurethane foam insulation. *JAMA* 1981, 245:243–246.

62. Becklake MR: Asbestos-related diseases of the lung and other organs. Their epidemiology and implications for clinical practice. *Am Rev Respir Dis* 1976; 114:187–227.

63. Selikoff IJ, Lee DH: *Asbestos and Disease.* New York, Academic Press, 1978.

64 Hirayama T: Non-smoking wives of heavy smokers have a higher risk of lung cancer: A study from Japan. *Br Med J* 1981; 282:183–185.

65. Trichopoulos D, Kalandidi A, Sparros L, et al Lung cancer and passive smoking. *Intl J Cancer* 1981; 27:1–4

66. Correa P, Pickle LW, Fontham E. Passive smoking and lung cancer *Lancet* 1983; 2:595–597

67 Matsukura S, Taminato T, Kitano N, et al: Effects of environmental tobacco smoke on urinary cotinine excretion in nonsmokers. Evidence of passive smoking. *N Engl J Med* 1984, 311:828–832

68. Ventilation for acceptable indoor air quality. ASHRAE Standard 62-1981

69. Hodgson MJ, Parkinson DK: Diagnosis of organophosphate intoxication [letter] *N Engl J Med* 1985; 313:329.

70. Methyl alcohol toxicity in teachers aides using spirit duplicators – Washington *MMWR* 1980; 29:437–438.

71 National Institute for Occupational Safety and Health Hazard Evaluations and Technical Assistance Report 81 352-1029. Department of Health and Human Services, IBM

72. National Institute for Occupational Safety and Health. Hazard Evaluations and Technical Assistance Report 83-172. Wrappingers Falls School.

73 National Institute for Occupational Safety and Health. Hazard Evaluations and Technical Assistance Report 81-247-958. Boehringer-Ingelheim, Ltd.

74 National Institute for Occupational Safety and Health. Hazard Evaluations and Technical Assistance Report 83-020-1351. Cornell University.

75 National Institute for Occupational Safety and Health. Hazard Evaluations and Technical Assistance Report 83 444. Georgetown University.

76. National Institute for Occupational Safety and Health. Hazard Evaluations and Technical Assistance Report 83-258. Planned Parenthood.

77. Chatigny MA Sampling microbial aerosols, in JA Merchant, BA Boehlecke G Taylor, et al (eds) *Occupational Respiratory Disease Report.* Morgantown, WV, US Dept Health and Human Services (NIOSH). 1981, vol 1, pp 117–163

See also pp 196–197.

FROM THE LIBRARY

VALUE OF HOME VISITS IN THE TREATMENT OF THE ALLERGIC PATIENT

. . . It is not possible to detect a musty, moldy odor by asking questions in the office or to obtain more than a rough idea of the location of the home and the type of living quarters. A few minutes in the home may give more information than an hour of history-taking in the office. A home visit is conducted by thoroughly examining the house from basement to attic and looking at the surrounding grounds. Since most people spend nearly a third of their lifetime in their bedroom, this is the most important room to examine. Beds are checked to see if pillows and mattresses are of the nonallergenic type or have protective zippered encasings. Because of the quantity of organic allergens usually found in the living room, it is advisable to locate the patient's bedroom as far as possible from this area.

The inspection of the cellar is often rewarding. Dampness predisposes to the growth of molds, and a cement floor creates dust which can be overcome by painting with a sealer or rubber-based paint. Often, discarded stuffed furniture is found which the patient neglected to mention during the office visit. The conglomeration of odds and ends which sit undisturbed, gathering dust, in some cellars is amazing. Individuals may be classified as "throwers" or "savers," and it is the latter who usually have the worst-looking cellars. Furnace pipes and cold air systems should be cleaned by strong suction each summer and the filters of heating units changed frequently throughout the winter.

The avoidance of animal dander in cases in which a pet may be an important cause is a potentially loaded subject familiar to all allergists. When the danger of inhaling such a potent substance day after day is carefully explained, most patients will gradually yield. When a pet is taken from a child, a harmless replacement should be offered to lessen the emotional impact. Stuffed animals and toys can be replaced by nonallergenic types of foam rubber or synthetic fibers. Stuffed animals, toys, blankets, carpeting, and stuffed furniture should be inspected. We advise elimination of all rugs from the bedroom and strongly urge that all felt rug pads, which usually contain crude cattle hair, be removed from the house and rubber pads substituted. Ordinary cosmetics should be kept away from the bedroom area and replaced by the nonallergenic variety. We urge that wool clothing which needs protection by moth-repellent chemicals be kept in tightly closed plastic bags away from the sleeping area. . . .

These procedures may seem to present a formidable list that necessarily upsets the routine of housekeeping, but they should never be presented in a manner which will overwhelm the patient. With tact and patience the allergist can usually succeed in getting the patient or his family to take the necessary precautions.

We firmly believe that home visits are one of the most valuable contributions to the successful treatment of allergic patients and urge all physicians to employ this procedure more often. The enhancement of the patient's response to allergic therapy will more than justify the additional expenditures of time and energy.

RICHARD D. WISEMAN, MD
JOSEPH R. WISEMAN, MD
(*NY State J Med* 1964; 64:1948–1951)

Arthur Donaldson Smith, MD

Physician, explorer, naturalist, and diplomat—Part III*

Pascal James Imperato, MD

Arthur Donaldson Smith left London on June 1, 1894, bound for Lake Rudolf, and sailed to Aden, where Lieutenant Colonel Sealy, political agent for the Somali Coast at Aden, and other British officials had overseen preliminary preparations. Smith engaged 55 Somali in Aden, each signing a contract drawn up by Sealy. Desertion was a major and understandable problem among expeditions heading into the East African interior, and Smith and Sealy were extremely cautious in their choice of men. It was mass desertion among unseasoned men which had caused the collapse of Chanler's expedition, and Smith was aware of this fact.

On June 29, 1894, Smith and Dodson sailed for Berbera along with their 55 Somali men (Fig 1). With them was Fred Gillett, a sportsman who planned to accompany them. Berbera was the final launching point where they spent ten days getting together 27 more men; 150 days' supply of rice, dates, and ghee (rancid butter); an enormous stock of trading goods; a total of 84 camels; and 100 boxes with copper vessels containing spirits for preserving reptiles, fishes, and batrachia. In addition, they took boxes for collecting birds and insects, a collapsible Berthon boat, cartridges, and numerous other supplies.[1(pp8-9)]

Captain Percy Cox, the acting resident at Berbera, with whom Smith later became close friends, did what he could to help send the expedition off in the best possible condition. Finally, on July 10, 1894, Smith marched out in the late afternoon and headed southwest for Hargeisa, 95 miles away. The country here is hot and dry, a broad plain which eventually rises up into a high plateau. And at the time of Smith's trip it contained concentrations of wildlife hard to believe today. Because Smith was a sportsman and a naturalist, his descriptions of wildlife in all the areas he traversed are detailed and extensive. There were *Oryx beisa* between Berbera and Hargeisa and a number of lions. Indeed, this area of Somalia and that to the southwest (called the Haud and the Ogaden) were renowned for their splendid lion populations and great herds of wildlife. They no longer are.

After spending four days at Hargeisa, Smith moved across the Haud, marching nine hours a day and covering about 24 miles per day.[1(p19)] Two days before leaving Hargeisa, Smith wrote to John Coles, the map curator of the Royal Geographical Society. In his letter he described his trip from Aden to Berbera and to Hargeisa and his concerns about his camels:

> There was no food within three or four days of Berbera; I had to push on to Hargeisa as fast as I could to save what few camels I had from starvation. I came with only 80 camels, all loaded very heavily. I should have had 110 camels for the loads. However, I only lost five camels on the way . . . .[2]

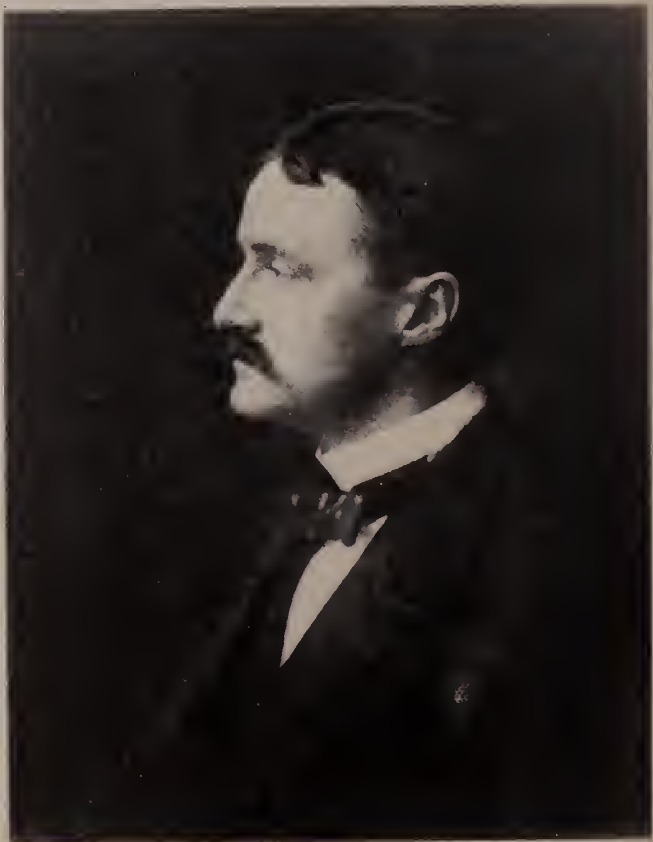


FIGURE 1. Arthur Donaldson Smith, MD, at the time of his first expedition to Lake Rudolf (reproduced from *Through Unknown African Countries*, London and New York, Edward Arnold, 1897).

---

* Parts I and II of this article were published in the February and March 1987 issues, respectively; parts IV and V will appear in subsequent issues. Author's acknowledgments will appear at the end of part V.

Address correspondence to Dr Imperato, Editor, *New York State Journal of Medicine*, 420 Lakeville Rd, Lake Success, NY 11042.

Explorers like Smith were understandably preoccupied with transport animals, for if they died or were too weak to go on, an expedition would collapse.

In his letter he also says: "I have Qualla Idris as my head man . . . "[2] In his book, *Through Unknown African Countries*, Smith refers to Idris as Hadji Idris,[1(p25)] no doubt because he had made the pilgrimage to Mecca. Qualla Idris (Fig 2) was a well-known figure in African exploration, although Smith does not mention this. Clearly, though, Coles knew who Idris was. Von Höhnel says of him:

> . . . [he] had been to America as a boy, and, later was for six years one of Stanley's truest and most faithful followers on the Congo, going eventually with his master to Europe . . . and acted as a guide to the expedition . . . led by the brothers James . . . Qualla spoke Arabic, Hindustani, English and Kiswahili.[3(v1,pp11-12)]

A head man could make or break an expedition, so Smith had used good judgment in hiring Qualla Idris, who was then 32 years old. Qualla Idris was a Somali from Habr-Anwal who had been with Henry Morton Stanley, with the James brothers on their expedition to Somaliland, and with Count Teleki and von Höhnel. The latter speaks of him in glowing terms.[3(v1,pp11-12)] However, Smith was to have problems with Qualla Idris, who threatened to desert him when they were detained by the Ethiopians.[1(p99)] Thus even seasoned men verged on collapse under the burden of hardship.

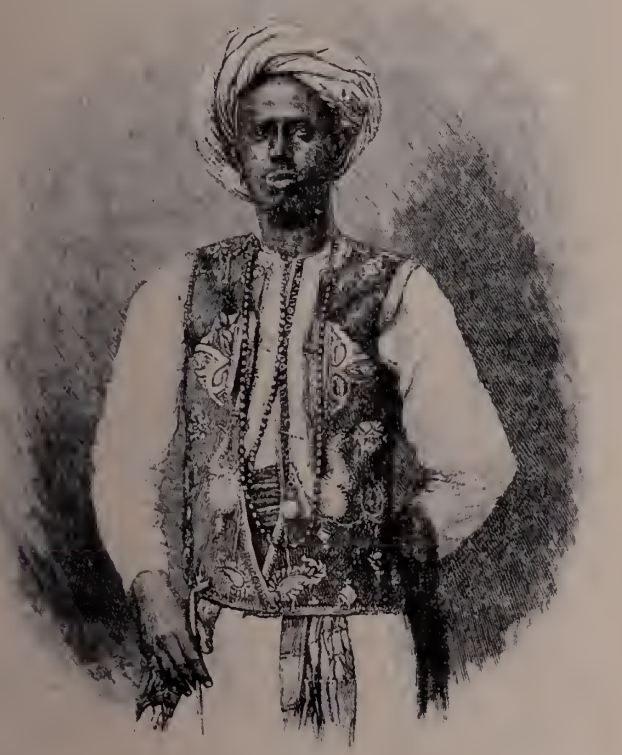


FIGURE 2. Qualla Idris, Smith's head man, who had served as chief of the Somali guard in the Teleki expedition. He later became a trader and drowned when his *dhow* (ship) sank in the Red Sea (reproduced from *Discovery of Lakes Rudolf and Stephanie*, London, Longmans, Green and Co, 1894).

Smith's gunbearer was Karsha,[1(p40)] also an experienced African traveler. He had traveled with Teleki and von Höhnel and with Chanler, who said of him, " . . . [Karsha] was tall, light in color, possessed of really beautiful features . . . and active and lithe as a panther. He was devotion itself, but lacked the qualities of patience and self-control, which are so necessary for a leader."[4(p26)] Patience and self-control were not Chanler's strong points. Yet it is of interest that he recognized their relevance to leadership. Chanler spelled Karsha's name as Karscho. He proved dependable and loyal to Smith.

The first medical problem of the expedition arose before Smith left Berbera. One of his men claimed to have been bitten by a poisonous snake. "I found the man groaning and acting as if he were in his last agonies, but there was not the slightest inflammation in his toe. He had merely been pricked by a thorn, so I gave him a good punching to get him on his feet, and proved to him that he was alright." The judicious use of force coupled with understanding went a long way.

By July 27 the expedition arrived at Milmil, situated on a dry river bed or "tug" of the same name. So far they were moving through country which was secure and frequently traversed by sportsmen of the era. In fact they were unable to hunt between Berbera and Hargeisa as the area was reserved for the Indian garrison at Aden.

But at Milmil, the local Somalis complained to Smith about a man-eating lion. Smith had a zareba (thorn bush enclosure) built and a donkey tethered ten feet away. He and one of his men then hid themselves in the zareba. Not long after sunset, a group of hyenas which had been upsetting both the scene and the donkey suddenly fled in all directions. There was a great thud as a huge male lion jumped on the donkey. Smith fired at the animal from a distance of ten feet. The lion roared and then bounded for the zareba. The weight of his body collapsed the wall on top of Smith and his Somali companion. The wounded lion was within a foot of Smith's face, roaring and moaning in pain. Finally the lion got up and walked to some bushes a few feet away. Smith lay still, waiting, until he heard the barking of a fox which told him the lion was dead. He and his Somali gunbearer spent the night keeping the hyenas away from what turned out to be a magnificant black-maned lion (Fig 3).

Taking on new camels at Milmil, Smith headed due west, not realizing that the Ethiopians had already penetrated this far south. This miscalculation was to bring him to grief.

The country immediately west of Milmil was then unexplored by Europeans. Smith could have given himself a wider margin of safety from the Ethiopians by going southwest. But this area all the way to the Webi Shebeli River (river of leopards) had been traversed by Captain Swayne, by Counts Hojes and Cudenhove, and by the Italians, Ruspoli and Bottego. Smith wanted to traverse as much unknown country as possible, but to do so he had to skirt around patches already explored by others. By the middle of August Smith was at Bodele on a tug called Terfa, 150 miles from Milmil. There he unexpectedly bumped into Captain C. J. Perceval. They exchanged pleasantries and

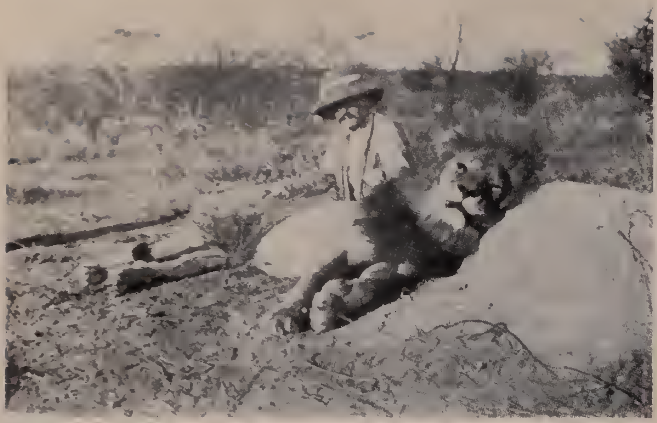


FIGURE 3. Arthur Donaldson Smith, MD, with a man-eating lion killed at Milmil, Somaliland, 1894 (reproduced from *Through Unknown African Countries*, London and New York, Edward Arnold, 1897).

Smith continued west while Perceval went north.

Smith moved into the Erer River Valley and says of it in his book, "The country swarmed with rhinoceroses." [1(p30)] Other forms of wildlife including lions, zebras, and antelope also abounded in the region. Ten days of marching brought Smith to the Webi Shebeli which was in flood and could not be crossed. Going downstream he found the river narrower and had rafts built to ferry the animals, supplies, and men across. But the crossing was difficult because of the swift current and the presence of large numbers of hippos and crocodiles. Gillett's mule was grabbed by a crocodile, but was finally rescued by the Somali. Smith and Gillett called this place "Crocodile Camp." It was situated on the left bank of the Webi Shebeli 50 miles north of the town of Imi.

Once across the river, Smith would have done better had he proceeded southwest. But he learned from two Galla his men had captured as informants that there was a town built of stone to the northwest. He impulsively decided to visit it. "This was most interesting news," he wrote. "Stone houses in this part of Africa!" [1(p43)] This town, Skeikh Hussein, was 160 miles away and, unknown to Smith, solidly in the hands of the Ethiopians.

Smith and his caravan had now been in the field for six weeks. He had thus far avoided any serious problems with local populations. However, from the reports given him by local people, it was clear that the Ethiopians were regularly raiding the area and, in so doing, had reduced people to destitution. This also made local groups suspicious of any outsiders and prone to attack before asking questions.[1(p43)] Smith had a well-armed group of fighters with him and constantly drilled them.

> The Somalis were very fond of being drilled, and it was not long before they learned to obey quickly the various orders given. In case of alarm, each company knew the position it was to take at once, and also where to find the cases of ammunition assigned to it. In ordinary cases there would be eight sentries on duty all night, one to each company.[1(pp35-36)]

SMITH DETAINED BY THE ETHIOPIANS

Smith and his caravan gradually marched up into the highlands, passing through herds of elephants and gazelles. Every Galla they met told the same story. They had been reduced to poverty by the raiding Ethiopians. Common sense should have induced Smith to head south, but the town built of stone was too much of a magnet.[1(p43)] On September 17, 1894, after Smith had named a nearby chain of mountains, the Gillett Mountains, in honor of his friend, he met up with his first Ethiopians. Their cordiality was in large measure due to the fact that Smith's caravan was a superior armed force. After hunting elephant, the caravan marched three days to Sheikh Hussein. The altitude here is 5,000 feet and the scenery is magnificent, consisting of deep canyons and high mountains. Sheikh Hussein was all that Smith expected, a town of stone houses and the elegant stone tombs of local Moslem saints.

There were only a few Ethiopians among the Galla of Sheikh Hussein. But it did not take Smith long to realize that they were not keen on his going west to Lake Rudolf. It also did not take long for all but 15 of his men to rebel against the cold.[1(p57)] He, Dodson, and Gillett managed to put down the rebellion. But Smith still thought the trip worthwhile. He discovered that the Webi Shebeli rose in mountains nearby, a fact unknown at the time. He also made detailed observations on the fauna, noting that there were many lesser kudu in the area. He named the headwaters of the Webi Shebeli after himself and the Web River after Gillett.

After two weeks in the fog and rain of Sheikh Hussein, two Ethiopians appeared, on October 1, 1894, and told Smith that Menelik's governor of the region wanted him to come to Ginea. Known as Ginir today, this town is about 200 miles north of Moyale, which is on the present Ethiopia-Kenya border. What was of more concern to Smith was the size of the governor's army, which had just returned from an expedition in the south. Smith, Dodson, and Gillett put their heads together and decided that only one of them should go to Ginea. Gillett agreed to go with eight men, confident the Ethiopians would not attack with the main Somali force and Dodson and Smith at Sheikh Hussein. During the week Gillett was gone, Dodson and Smith collected natural history specimens and Smith drilled his Somali in preparation for an Ethiopian attack.[1(p63)] Smith wrote:

> I tried to keep my boys cheerful by instituting games of all sorts, and by drilling them night and morning, giving them also much target practice. As there was a possibility of the Abyssinians attacking the camp, these drills were a most important precaution.

Smith not only kept his fighting men in shape, he also distracted them from their worries over an Ethiopian attack.

> I also offered a rupee to any of my boys, who would bring me in any new natural-history specimens; consequently dozens of snakes, lizards, mammals and curious kinds of bugs were being continually brought to my tent. My boy Karsha captured for me . . . an animal fourteen inches long, and covered with long, silvery white fur, resembling *Lophiomys imhausi*.[1(p64)]

The animal, a rare crested tree rat, was later named *Lophiomys smithii*.

A week later a message came from Gillett saying it was alright for the whole caravan to come to Ginea. In three days Smith and Dodson were there. The Ethiopians were courteous, but the governor, Wal-da-Gubbra, said he could not let Smith go on unless Emperor Menelik approved it. So Smith wrote a letter to Menelik on October 17, expecting a reply in nine days. Smith told Menelik that the purpose of his journey was purely scientific. Menelik had, of course, heard this sort of story many times before, as Smith well knew.

To fill in the time the three men made short trips around Ginea. It was during one of these trips that Smith discovered the spectacular caves of the Web River which he called "Wyndlawn" after his summer home near Philadelphia. The name, like that of the Gillett Mountains, did not survive far into the future. These caves, cut by the course of the river through the base of a mountain, are in a sense an underground Grand Canyon.[1(p82)] The caves today are known as the Caves of Sof Omar, after a nearby village.[5]

A LETTER FROM EMPEROR MENELIK

Smith and his group tried to slip out of Ginea but were stopped by the Ethiopians, who were prepared to use force if necessary. Finally, on November 16, over a month since his arrival in the area, Smith received word from Menelik that he must return the way he came. Smith says: "The letter was in Abyssinian, but translated by a Frenchman into French and English, and was enclosed in an envelope bearing a gilt crown." [1(p102)] A copy of the Amharic version of his letter is reproduced on page 103 of Smith's book and a French translation appears on page 455.[1] The French syntax and grammar are extremely poor. An English version of his French text is as follows:

> The Conquering Lion of the Tribe of Judah, Emperor of Ethiopia, etc.
> Salutations
>
> I have received your letter of October 1894 from Shine.
>
> I have learned that you have come for a goal concerning Europe to visit the Galla country situated to the south of Ethiopia. Only the period you have chosen for this trip coincides with that in which I myself am going to leave on an expedition and that means that you wish to cross the same country I wish to subjugate. I fear that in passing by this country you will only find yourself between the Gallas and the men of my army. In case a misfortune befalls you, that would give me much distress. I will be very happy if you would cross these countries when they will be subjugated. I tell you that because when a traveler has an idea, it is better that it succeeds; if he falls into a precipice it is a misfortune for him and for the king of his country.
>
> I am not telling you not to carry out your project, but I hope you will have the patience to wait 2 or 3 years when these countries will be altogether subjugated.
>
> Addis Ababa 29 October 1887
> 7 November 1894

In the spring of 1986, a copy of the original Amharic version of this letter was sent to Addis Ababa in Ethiopia for translation.[6] It reads as follows:

> The Conquering Lion of the Tribe of Judah, Menelik The II,
> Elect of God
> Emperor of Ethiopia
>
> To: Monsieur Arthur Donaldson Smith:
> Greetings!
>
> Praise Be Unto God, I Am Well
>
> I received your letter of October 1894 from *Gine*. Your visit is of mutual interest both to Europe and Ethiopia. Unfortunately, the month you have chosen for your trip coincides with that of mine during which period I myself am going to leave on an expedition to the uncivilized part of the country which happens to be the route you will be travelling through. I am worried that you may be caught in between my army and that of the savages in which case I shall find it rather distressing should anything bad happen to you as a result of this. I shall be happier if your choice would be to visit the civilized part of the country. In suggesting that I am by no means implying that I am disapproving the purpose of your visit: On the contrary, all that I am saying is a successful mission is gratifying but failure of it will not only be a source of disappointment to you but also will have a negative impact on the reputation of the ruler of that country. As it will not take more than 2 or 3 years to civilize this part of the country, it will perhaps be preferable if you could postpone your trip until then.
>
> Done at Addis Ababa the twenty ninth day
> of October 1887 (Ethiopian Calendar)
> November 7, 1894 (Gregorian Calendar)

This translation does not differ much in substance from the French version published by Smith. However, its tone is substantially more diplomatic. An extremely important point is that, in the 1986 translation, Menelik says that he is going to leave on an expedition to the uncivilized part of the country, implying that the area is Ethiopian. The 1894 translation says, "... the Galla country situated to the south of Ethiopia." [1(p455)] The implications of this are significant because Menelik was within his rights to prohibit passage through areas that were Ethiopian. In point of fact, the area was not Ethiopian yet, but Menelik was anxious to make it so as part of his plan of imperial expansion. Wal-da-Gubbra, the local Ethiopian governor at Ginea, told Smith that Menelik owned all the country to Mombasa.[1(p100)]

When examined in Addis Ababa in June 1986, it was noted that Smith's Amharic version bore no signature and no official stamp. This may represent an oversight. However, it also raises the possibility that the letter was written on imperial stationery in either Addis Ababa or in a regional center in the emperor's name and not by Emperor Menelik himself.

SMITH'S EXPEDITION RETREATS

It was while Smith was in the area of Ginea that Qualla Idris came to him, saying, in Smith's words, "... he was disgusted with the expedition, and wished to be allowed to return home by way of Abyssinia. This was a splendid thing for a head man to do, just at the critical point to desert his master, when everything seemed to be going against him!" [1(p99)] Smith convinced Idris to stay but never trusted him after that.

> He returned to his duty in a sullen fashion, but I could never trust him after this ... my two head men Salan Mohammed and Ahamed Aden were most loyal towards me. This Salan was by far the best man I had in the caravan ... Idris hated Salan on account of the latter's loyalty to me; he was too cunning to attempt to interfere with him, as he knew that if it came to an issue between us I should have Salan and a large number of boys to support me.[1(p99)]

Idris knew that they were fairly close to Addis Ababa, which is about 200 miles northwest of where they were. It was fairly safe and easy to travel from there to the coast. The faithful Qualla Idris of Stanley and Teleki had become disgruntled and capable of mutiny in other circumstances. Smith was still determined to get to Lake Rudolf. So he headed far east, back into Somaliland, to fool the Ethiopians, and then went south along the Webi Shebeli River. The land was full of zebra, oryx, gerenuk, and rhino. By Christmas they had been in the field five months. They met up with Prince Boris, a Russian sportsman, and had Christmas dinner with him.

Some of the men were sent back to Berbera on the coast to pick up additional supplies for Smith's new attempt to get to Lake Rudolf. During the week that ensued, Smith recovered as best he could from both malaria and conjunctivitis as his main force moved south to Bari, a trading center.

It was at Bari that Fred Gillett learned that his father had died three months before. Smith writes:

> But now Fred received a shock that would have been hard enough to bear at any time, but which was all the more severe as it came when he was far away in the wilderness . . . Coupled with the sad news were announcements that made it imperative that my friend, who had assisted me so often in my geographical and natural-history work, should return home at once . . . On the third day (January 25) Fred started for the coast, with nine men and ten camels. I sent out at the same time to Berbera eight boxes of natural-history specimens, and my poor sick head man, Ahamed Aden.[1(pp135–136)]

However, Gillett's father's death may have been only part of the reason why he left. Smith and Gillett obviously had some kind of a falling out, as borne out in a letter of February 27, 1896, that Smith wrote to J. Scott Keltie, secretary and editor of publications of the Royal Geographical Society.[7] In this letter, Smith wrote about the proofs he had received for his article that was to be published in the *Geographical Journal.* He said: " . . . please change the sentence 'Mr. Fred Gillett agreed to accompany me with a sporting outfit' to Mr. Fred Gillett asked me to permit him to accompany me with twelve men, to which I agreed on account of companionship." Smith then goes on to tell Keltie, " . . . I regret that you ever asked for Gillett's photographs for the journal but as you spoke to him first I did not like to refuse." Smith then asked that the Gillett photographs be placed in the center of the article and not at the beginning. "I have spent much money, to say nothing of the risks I ran on the expedition, and I think I should not have an article published by the R.G.S. that will disgust me to look back upon." [7] Smith then candidly says: Whatever happened between myself and Gillett has been patched up, and I do not think you would want to bring about rows." [7] Smith then told Keltie that his friends were under the impression that Gillett had shared in the expedition's expenses whereas he (Smith) would have saved money by not having had him along. "I have put Gillett forward in the paper and have given him credit for things I do not believe. I trust this will not be repeated by you. I only give you this information as a hint that there is a very sore point with me in regard to the trip that I wish to forget entirely." [7] It is unclear from this letter whether Smith and Gillett had a row in Africa or if their misunderstanding arose later over Gillett's getting undeserved credit for the expedition.

Keltie did change the language on the proof, conveying the sense of what Smith wanted, but putting it into slightly different words.[8] Smith and Gillett had mended their differences to some degree by early 1896, as the latter was present at the meeting of the Royal Geographical Society in London on January 6 when Smith presented the results of the expedition.[9] At the end of the presentation Gillett rose to speak and said, "I wish to express my admiration for the persevering way in which Dr. Donaldson Smith mapped out the country, in spite of the broiling and enervating climate, tiring marches, and the inaccurate reports given by natives, unaccustomed to precision." [9] Gillett then told a very humorous story about local impressions of the uses of the theodolite.

The evening of Gillett's departure almost everyone in the camp came down with malaria. Smith wrote: "I found only five men with a high temperature and rapid pulse, and naturally concluded that most of them were shamming; but I afterward learned by experience that the elevation of temperature and rapidity of pulse are not at all proportionable to the severity of the peculiar kind of malarial diseases found in many areas of Africa." [1(p137)] Smith and Dodson then came down with malaria themselves:

> . . . Dodson and I were down on our backs with pains from head to foot, and with headaches that would occasionally make us delerious. Our temperatures, however, only rose to 103 degrees F., with a pulse of 95 beats per minute . . . the outlook seemed serious; there was scarcely a sufficient number of men well enough to look after the camels, and I could just manage to get around the camp twice a day to attend to the sick boys.[1(p137)]

Statements of this kind, describing serious personal illness and suffering, reflect Smith's modesty and often appear in his writings. He never dwells on his own sufferings, but rather almost mentions them in passing.

Large doses of quinine eventually got almost everyone back on their feet. It is easy to understand today why Smith's expedition was so severely crippled by malaria at the time. He was camped along the Webi Shebeli River during the rainy season, when malaria transmission is at a peak. Although in 1894 Manson had put forward the theory that malaria is transmitted from man to man by mosquitoes, this had not yet been convincingly demonstrated, and would not be until 1898.[10]

SMITH RESUMES HIS MARCH WEST

On January 31, 1895, Smith and Dodson, with 75 men and 65 rifles, made a fresh start and headed southwest towards the Jub River. The 200-mile march to the Jub was through monotonous bush where at the time sizable herds of giraffe, waterbuck, oryx, and zebra were seen. It was while he was on this march that Smith saw 40 hyenas attack three lions. And he himself had another close call with a lion which lay outstretched outside his zareba, its nose two feet from his face!

On February 17, Smith and Dodson crossed the south-to-north lines of march of Prince Ruspoli and Captain Bottego and moved south along the Web to where the Daua River flows into the Ganana. The Daua River forms the border today between northeastern Kenya and Ethiopia. It and the Ganana form the Jub (Fig 4).

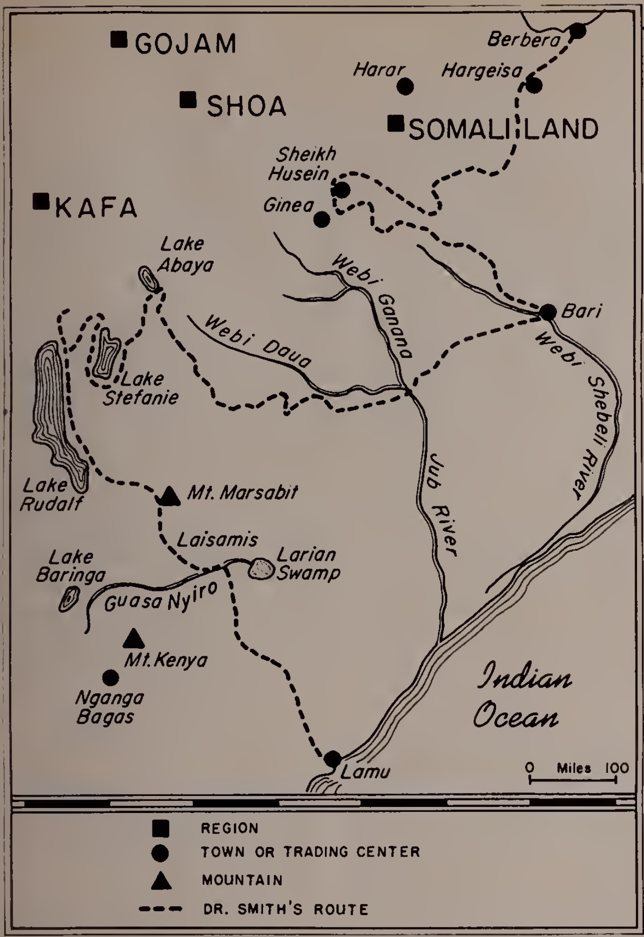


FIGURE 4. Arthur Donaldson Smith's route into the interior during his first expedition to Lake Rudolf, 1894–1895 (drawn by Saturnino B. Villapaz, SUNY Health Science Center at Brooklyn).

Most of Smith's undeveloped photographic plates were damaged during his crossing of the Ganana, which accounts for the fact that he made few photographs in what is now northern Kenya.[1(p158)] He and Dodson moved along the southern banks of the Daua and were soon in what is now the northeastern corner of Kenya, near Mandera. Smith wrote the following about this region: "... The Daua runs through a beautiful fertile plain, nearly a mile wide ... waterbuck, giraffes and lions abounded in this country. ..." [1(p163)] This was the first description of Kenya's northeast, Smith and Dodson being the first Europeans ever to see it.

The line of wells that run east to west from El Dere to Egder is in southern Ethiopia today, just north of the Kenya border. Smith and Dodson followed this line.

By March 31, 1895, they were marching west, 20 miles north of Moyale, in the country of the Boran, where in 1909 modern medical services would begin.[11] It was while he was in this area that Smith and his gunbearer, Karsha, were struck by lightning while hunting Thomson gazelle.[1(p189)] Both were knocked unconscious. Karsha had been on both the Teleki and the Chanler expeditions. So by the time Smith's caravan reached the area of Lake Stefanie, 200 miles northwest, Karsha was in familiar territory. He and Qualla Idris were the only men until that time to have reached Lake Rudolf from both the northeast and the southeast.

As Smith proceeded through the country of the Boran he had to engage in several pitched battles[1(pp193–200)] (Fig 5). As other explorers had done, Smith fired off several rockets at night, which lit up the sky with dazzling fireworks and made an impression on the locals. Smith provides graphic descriptions of his encounters with the Boran, who were and still are pastoral nomads with a strong military tradition. The trouble started when two of Smith's men were attacked by the Boran, with one of them, Elmi, being fatally wounded. "Poor Elmi lay dead behind a bush, horribly mutilated, his intestines scattered about, and a part of him carried away as a trophy by his murderers." [1(p193)] Smith was euphemistically referring to the Boran custom of removing the testicles of their victims. Between April 6 and 10, 1895, Smith and his caravan were constantly being harassed by Boran war parties consisting of thousands of men on foot and on horses. Smith used his well-drilled Somali men, who were as good as any soldiers of the day, to drive the Boran off, employing standard military tactics. In his diary entry of April 8, 1895, Smith wrote:

> ... The punishment the natives received was terrible; but still they came on, regardless of those that were falling about them

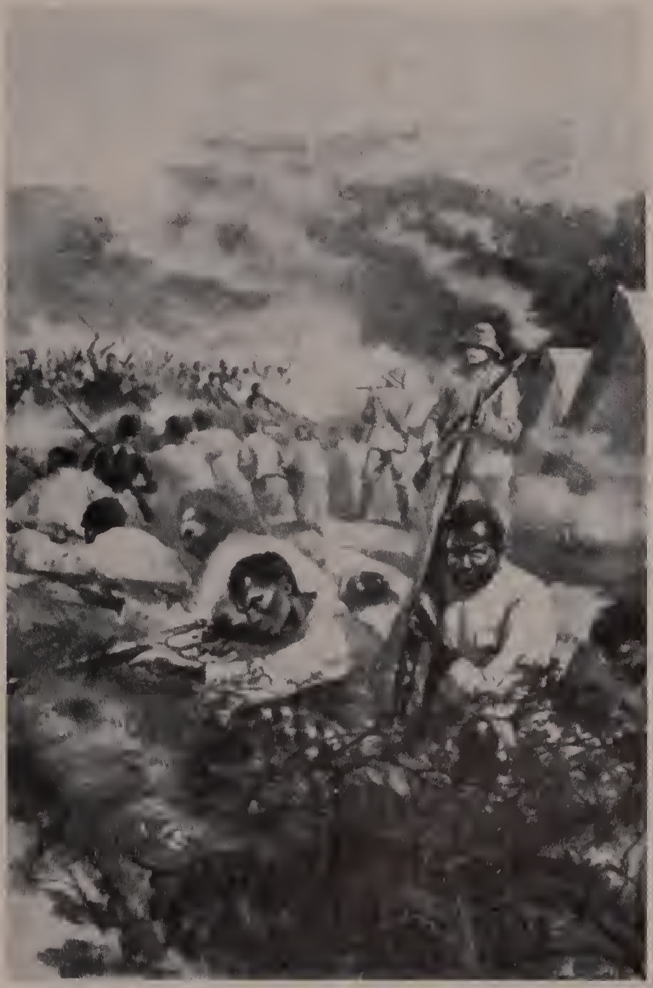


FIGURE 5. Arthur Donaldson Smith, MD, fighting off the Boran (reproduced from *Through Unknown African Countries*, London and New York, Edward Arnold, 1897). In this romanticized drawing Smith appears as a towering figure. In reality he was around 5′7″ tall and of slight build.

> and the din of musketry . . . frantic to get away the poor savages, who had now for the first time realized that a bullet will pierce a shield and kill a horse hundreds of yards away, were taking themselves off as fast as their legs could carry them.[1(p199)]

The Boran were interested in stealing Smith's camels and mules and in pilfering his caravan. He was surprised by their attacks because he did nothing to provoke them, and initially his contacts with them were peaceful. The effect of Smith's guns on the Boran and their horses must have been serious because the Boran sued for peace, returned the animals they had stolen, and let Smith proceed unhindered. Smith now turned northwest toward Lake Abaya. He wrote of this area:

> . . . There were many Burchell's zebras, Coke's hartebeests and giraffes . . . Never in my life have I seen more game than there was about the edge of these mountains. Hundreds of zebras, hartebeests, gazelles and ostriches, as well as many giraffes and rhinoceroses were to be seen on all sides. . . .[1(p213)]

It was in this bush country, so different from the dry scrub bush which lies to the south, that Prince Ruspoli was killed by an elephant on December 4, 1893. Ruspoli had reached the area by following a line of march far to the north of Smith's.[12] It was, as Smith described, a wildlife paradise.

SMITH REACHES LAKE RUDOLF

Smith now turned southwest toward Lake Stefanie, a distance of 100 miles. An epidemic of rinderpest had wiped out the buffalo population five years before, and Smith remarked that he came across thousands of skulls of these animals.

Smith and Dodson were now on the Tertala Plateau, 5,000 feet in altitude, in a country inhabited by the Konso people. Smith discovered that the Galana Amara River flowed from Lake Abaya into Lake Stefanie. He also traveled around the eastern, southern, and northern shores, observing large populations of elephant, waterbuck, and gazelle. Unlike Teleki and von Höhnel, who only saw the southern part of Lake Stefanie, Smith virtually walked around it. He found that there was a small lake ten miles long and two miles wide extending from the northwestern corner of Lake Stefanie. He named it Lake Donaldson. Since Smith's day both lakes have dried up and most of the wildlife he described has vanished.

Moving to the northwest of Lake Stefanie, into a region never yet explored by Europeans, Smith encountered numerous small ethnic groups. Among these, the Arbore gave him a lot of difficulty, ambushing his caravan on several occasions. Once they were attacked by 450 Arbore armed with bows and poisoned arrows.[1(pp264-265)] He also met the Dume people, whom he believed to be pygmies because of their short stature. In fact, on his return to England, his descriptions of the Dume provoked more excitement than any of his other discoveries. They may have been short, but they were not pygmies.

Smith had now been a year in the interior of Africa, and Lake Rudolf was only 100 miles to the southwest. Finally, after wading down a stream for five hours through thick forest, they came into open country, and on July 10, 1895, saw the lake. Smith wrote: I felt that I had attained in a measure the greatest ambition of my life—that of being able to add a little drop to the sea of knowledge. . . ." [1(p288)]

The marshland to the northeast of the lake was then alive with hartebeest, topi, gerenuk, giraffes, and zebra. On July 14, 1895, Smith and Dodson camped on the same spot where Teleki and von Höhnel had camped for a month in 1888. Since these two explorers had left the area, no Europeans had visited it. However, a Swahili caravan, the first ever to go that far north, had come from Mombasa and reached the area north of the lake a few years before Smith's arrival. Unknown to Smith was the fact that Arthur Neumann, an ivory trader, was heading north up to the lake, having come from Lamu on the coast. He reached the southern shore of the lake on December 6, 1895.[13] Von Höhnel had only gotten 15 miles above Lake Rudolf in 1888. Smith now determined to explore this unknown land and to settle the question of Lake Rudolf's source. Teleki and von Höhnel had found a river called the Nianamm flowing into the northern end of Lake Rudolf. What no one knew then was whether this was the same as the Omo River, whose upper course had already been explored by the Frenchman Borelli and others. To solve the issue Smith had to explore a strip of land a hundred miles long.

After starting out, he had to return to his camp because of severe attacks of malaria. However, on July 24, although still sick, he made a second start. His route took him through the fertile country of the Murle and Kere people, who made his transit difficult. Finally, about 80 miles north of Lake Rudolf, Smith, who had previously thought the Nianamm and the Omo were one, erroneously concluded they were not. The Omo, he said, probably flowed into the Jub or the Ganana,[1(p319)] and the Nianamm was a different river and the source of Lake Rudolf. Victor Bottego, the Italian explorer, would prove that the two rivers were one the following year, but it would cost him his life.[14]

Today, on modern maps, one can still find a mountain chain called Mount Smith, situated in southern Ethiopia, roughly 110 miles north of Lake Rudolf. It is the only permanent geographic reminder of Smith's gallant two-week trip to solve the mystery of the Omo River (Fig 6). Smith wrote: "One splendid group of mountains, thirty-five miles to the north of us and nine thousand feet high, I have named after myself." [1(p319)] In retrospect, Smith had gone around 80 miles north of Lake Rudolf, up the Omo, and probably crossed the Mago River, a major tributary of the Omo.

The immediate effect of this trip on Smith was attacks of malaria. He arrived back at his Lake Rudolf camp in sorry shape on August 5, 1895.

Smith had accomplished his mission. He had reached Lake Rudolf from the east and had, he thought, settled the question of the Omo River. He was anxious to get home and tell the world of his discoveries. So on August 8, 1895, he and Dodson and the caravan started down the eastern shore of Lake Rudolf, heading for Mount Kulal.

As they proceeded south the country grew drier, a dramatic change from the fertile, lush lands at the northern end of the lake. But there were plenty of rhinos, elephants, and waterbucks in the region. On August 12 they reached Alia Bay, where in recent years Richard Leakey and his

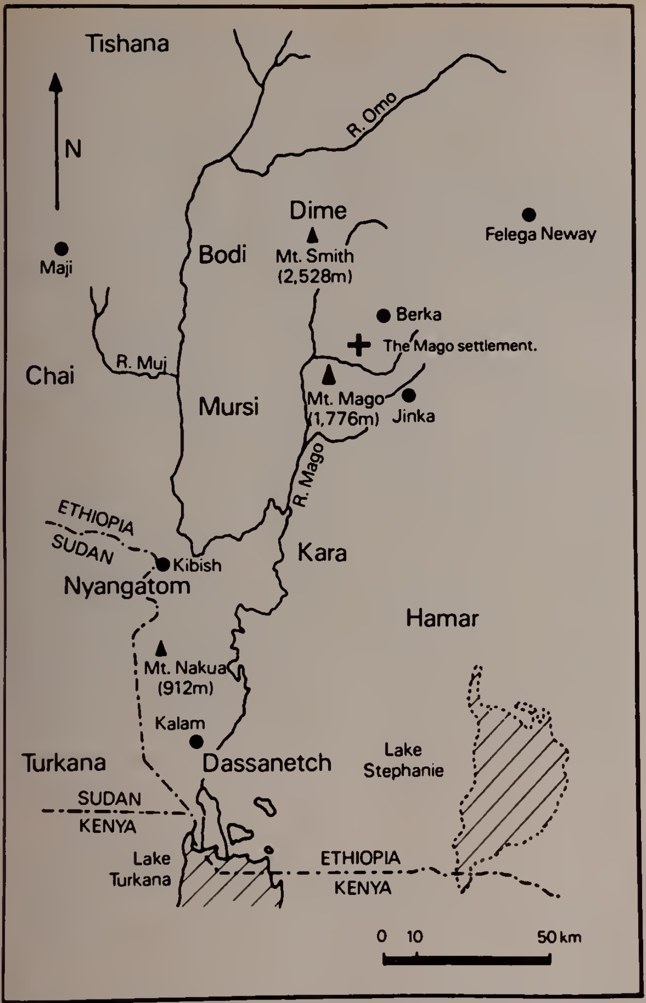


FIGURE 6. Current map of the lower Omo River showing Mount Smith and the location of the region's ethnic groups (courtesy of Professor David Turton, University of Manchester, Manchester, England).

colleagues have made stunning discoveries of man's ancestors. It was at Alia Bay that Smith first saw buffalo, they having been completely wiped out to the north by rinderpest. And a week later they got to El Molo Bay, near today's Oasis Fishing Camp and Loiengalani. It was from here that they saw a spectacular nighttime eruption of Teleki's volcano, with bright red lava flowing down its sides.[1(p333)] The region was full of rhinos and elephants, and the former were so numerous as to cause the caravan to halt several times.

Smith gave his Berthon boat to the El Molo and then on August 24 left the lake and headed east towards Mount Kulal. This huge, forested volcanic mountain had previously been visited by Teleki and von Höhnel. Smith and Dodson, however, went around the northeast side. Kulal is 7,500 feet high and remarkable because it is cut in half from its summit down for a height of 3,000 feet. This crack is 100 feet wide and essentially divides the mountain into two parts.

SMITH DISCOVERS MOUNT MARSABIT

Although Smith did not realize it, his expedition was now on the brink of making some truly unique geographic discoveries. He and Dodson were entering an enormous expanse of territory completely unknown to Europeans, stretching from Kulal to Mount Marsabit in the east and south to the Guaso Nyiro River. Ahead of them lay Marsabit, a high volcanic range some 30 miles long, whose summits were and still are covered by a dense forest. No European had ever seen Marsabit. Leon des Avanchers referred to it on his 1859 map under its Boran name Sakou, and von Höhnel accurately recorded what the Rendille and Samburu said about it and accurately placed it on Chanler's maps.[3(v2,pp4,76,184),4] However, no European had ever seen it.

Marsabit rises to a height of 5,000 feet. The forest begins at an altitude of 4,300 feet, and below that are dense bush and open glades. The southwestern slopes, consisting of conical hills and extinct volcanoes, move up gradually until three distinct summits are formed. This enormous verdant range is full of springs, pools, and spectacular ravines, and there is a crater lake at its highest summit. But around it, stretching for almost 100 miles in every direction, is a dry and waterless desert. The Dida Galgalu—"the land of thirst"—is a hot, shadeless, and waterless stretch of country which falls away from Marsabit to the north. And to the west are the Chalbi and Koroli Deserts and to the south, the Kaisoot Desert.

Smith and Dodson now headed across the desert to the west of the mountain. It took them five days to get to the base of Marsabit, traveling, as Smith says, over a path worn smooth by countless wild animals.

> . . . On the second day we commenced to ascend the mountain, but we did not reach the top until after three marches. I was attacked by fever on these marches, and twice had to be carried; but the cool bracing air at the top of Mt. Marsabit quickly cured me. . . .[1(p352)]

It was not so much the air that cured Smith as the heavy doses of quinine he later said he took. But in spite of the quinine, he was still rather sick. He said of Marsabit:

> According to European ideas, nothing could be more charming than this Marsabit. Surrounded by a large forest and lying at the top of the mountain, is a lake a mile square, clear and deep. The jagged walls of a crater form a semicircle about it, while from another side a broad road leads out from the forest to the open meadow beyond . . .[1(p352)]

This lake would be called "Crater Lake" by a number of travelers until it was renamed "Lake Paradise" in the 1920s by the American wildlife photographers Martin and Osa Johnson.[15] Although Smith does not mention his geographic survey work on Marsabit, he nonetheless took readings in addition to collecting geologic and natural history specimens.

Eighteen years later, in 1913, Geoffrey Archer, the first British administrator assigned to survey what is now northern Kenya, wrote the following tribute to Smith in *The Geographical Journal*:

> To Dr. Donaldson Smith is due the credit of having been the first scientific explorer to penetrate inland from the east coast of Rudolf and locate Marsabit; and the position he assigned to the Crater Lake in the year 1895 serves as a good example of the high degree of accuracy he maintained in the survey of his route . . . Considering that Dr. Donaldson Smith carried his traverse many hundreds of miles. . .this accuracy is very remarkable indeed. . . .[16]

In the dry season, Marsabit teemed with wildlife. The crater lake was full then, as it is now, depending on rainfall. It and Marsabit's pools, springs, and marshes contain the only water for miles around. For this reason and because of Marsabit's relative isolation, Martin and Osa Johnson chose it as their base for a few years in the 1920s. Smith treated himself and his men for malaria on Marsabit, thus rendering the first modern medical care on the mountain. It would be another 14 years before modern medical services would be started at the Marsabit post by the colonial military.[17]

March to the Coast

Smith left Marsabit on September 13, 1895, after spending only four days on the mountain. Before doing so he was almost killed by an elephant. He wrote the following understatement: "By this time I had begun to look forward to getting home safely." [1(p354)] Smith and Dodson reached Laisamis waterhole three days later. Smith described it as follows: "the enormous herds of zebra grevii, and the numerous giraffes and rhinoceroses that were to be found around Laisamis supplied these people [the Wanderobo] with food." [1(p354)] Laisamis was extensively used by Paul Rainey, the American sportsman and explorer, for making wildlife films during his several trips to East Africa in the early part of this century.[18-20] Martin Johnson also photographed here a decade later.[15,21] Guided by the Rendille, Smith and Dodson headed south through more unexplored country, which was full of giraffes, oryx, gerenuk, and rhino. Finally, on September 22, they reached the Guaso Nyiro River, having traversed 140 miles of some of the most difficult terrain in Kenya. Smith had a severe relapse of malaria here, which responded to heavy doses of quinine. But he had to camp for eight days on the northern banks of the Guaso Nyiro at Melka Koja before fully recovering. Chanler and von Höhnel had reached this point on their way to the Lorian Swamp.

One of Smith's Somali men, Yusif, was attacked by a crocodile and nearly dragged away while carrying a sheep across the river. "... I was obliged to amputate the arm close to the shoulder. While I was engaged in operating on Yusif's arm, a herd of elephants passed near the camp, but I did not go after them..." [1(p359)] Thus did Smith describe what is in effect a serious surgical procedure, even in a modern hospital.

On October 1, 1895, Smith and Dodson headed south across an unexplored stretch of country between the Guaso Nyiro and the Tana River, an area full of rhino. By October 7, they were at the Tana River. They had been traveling for 15 months. Only seven Europeans had seen the Tana at this point by that time. And a eighth came sailing along just as Smith and Dodson got to the river. "... Imagine our joy and astonishment ... when a canoe hove in sight ... and in that canoe sat a man holding a pink umbrella! Yes, true enough, a pink umbrella, and underneath, a man in a white suit!" [1(p363)]

This was an episode no fiction writer would dare to write. But it happened. The Reverend Robert Omrod of the Methodist Reformed Mission was on his way downstream after an exploratory trip for the purpose of setting up a new mission. Thanks to him, Smith and Dodson had a comfortable trip down the Tana, stopping off at mission stations and finally reaching Lamu at the end of the month. On October 29, 1895, Smith, Dodson, and their Somali men sailed to Aden. They had covered 4,000 miles in 16 months and filled in a number of blank spaces on the map of Africa. In the process only six of the 80 Somali lost their lives.[1(p367)] A one-word cablegram sent by Smith from Aden to Philadelphia summed up the results of his expedition. It read "successful." [22] A similar message was received at the Royal Geographical Society in London on November 4.

Smith and Dodson arrived back in England on November 30, 1895. On the evening of January 6, 1896, Smith presented his findings to the Royal Geographical Society. He had collected 700 specimens of birds, 24 of which were new to science; 300 reptiles and batrachians, of which 11 were new; 300 specimens of plants; 1,000 butterflies; 200 mammals; and close to 6,000 insects. The staff of the British Museum examined all of these specimens and Smith donated all of the new species to the museum.[1(pp368-369)] The remainder he donated to the Academy of Natural Sciences of Philadelphia. Of the new species, some were named after Smith; including the rare crested rat, *Lophiomys smithii*, a fish, *Synodontis smithii*, and a bird, *Turacus donaldsoni*. His geological collection was examined by J. W. Gregory, noted for his own expedition to Kenya's Rift Valley.[23]

Smith's natural history collections were described by various scientists in the *Proceedings of the Zoological Society of London*, the *Journal of Botany*, and the *Proceedings of the Academy of Natural Sciences of Philadelphia*.[1(p369)] Nine appendices in Smith's book also contain descriptions of part of these collections as well as of his ethnographic collection.[1(pp377-454)]

Smith's collection of birds was described in detail by R. Boulder Sharpe in an article that contains two magnificent color plates of *Turacus donaldsoni* and *Serinus maculicollis donaldsoni*. One hundred eighty-one different species are described, with details provided about their provenance.[24] Although Smith collected many species that were already known, he shed much light on their geographic distribution in Somaliland. His collection of reptiles and batrachians was described in two articles by G. A. Boulenger.[25,26] Both of these were accompanied by two black-and-white plates, one of which contains a drawing of *Agama smithii*, named after Smith.[26] Smith also collected 35 fish representing 18 species, one of which, *Synodontis smithii*, was named after him. These specimens were described by A. Gunter.[27] Smith donated part of his natural history collection to the University of Pennsylvania Museum (Fig 7).

Smith also gave his ethnographic collection to the University of Pennsylvania Museum, and in 1914 gave them a second such collection from his subsequent African expedition.[28] There are now 276 of Smith's objects in the museum's collection representing the artistic skills of the peoples of northern Kenya, Somalia, and Ethiopia. This is the most important early collection of ethnographic objects from northern Kenya in the United States.

The Royal Geographical Society received all of Smith's maps and he himself received considerable praise for their quality from John Coles of the society. However, E. G.

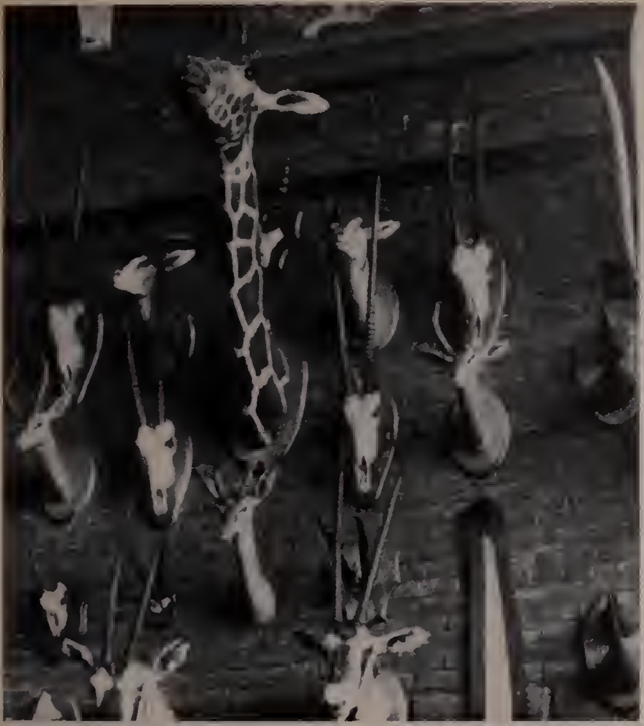


FIGURE 7. Part of Arthur Donaldson Smith's natural history collection at the University of Pennsylvania Museum (reproduced from *Through Unknown African Countries*, London and New York, Edward Arnold, 1897).

Ravenstein, a leading geographer, disagreed with Smith's claim that the Omo did not empty into Lake Rudolf. He suggested that Smith return to Africa to settle the question.[9]

Smith's verbal presentation about his trip before the Royal Geographical Society was well received. The session was chaired by Sir George D. Taubman Goldie, one of the society's six vice-presidents. The American ambassador who was present cogently observed in remarks made after Smith's speech that the trip was "... an honest, brave, modest endeavor to let all the world know something of distant regions of which nothing seems to have been known before ...."[9]

Ambassador Bayard also noted that Smith's trip had not been undertaken for profit or utility, remarks no doubt intended for those whose expeditions were politically motivated or motivated by profit.[9]

The focus of some of the speakers was on Smith's exploration of the area between the Webi Shebeli River and Lake Rudolf. Interestingly, nothing was said of his equally important accomplishment of exploring the area of present-day northern Kenya between Lake Rudolf and the Guaso Nyiro River.

John Coles, the map curator, praised Smith for the excellence of his geographical measurements and for the dedication he had shown in preparing for the trip. "I can bear witness, from the time he came to me, both in observing the stars at night and in surveying the country, that he gave his whole and undivided attention to the subject."[9]

Ernst George Ravenstein was the last to speak, before Smith said a few words of thanks. He was a German who had come to Britain in the 1850s to work at the War Office's Topographical Department. He took a leading part in the public debates about African geography during the second half of the 19th century and helped construct the maps of several African explorers including J. Ludwig Krapf, to whose book he also wrote an introduction.[29] He raised the issue of the Omo River and its course, disagreeing with Smith:

> ... I am inclined to think that the Omo, after all, takes its course to Lake Rudolf ... I may observe that neither Dr. Smith nor Count Teleki saw the Nianam at its best ... This question of the course of the Omo is perhaps the most interesting problem to be solved in connection with African hydrography, and it would redound to the honour of America if Dr. Donaldson Smith went out again and settled it.[9]

It rankled Smith that he had not resolved the question of Lake Rudolf's source and the matter of the Omo River.[30] This, coupled with his desire for exploration, led him to plan a return trip to the same region.

RETURN TO THE UNITED STATES

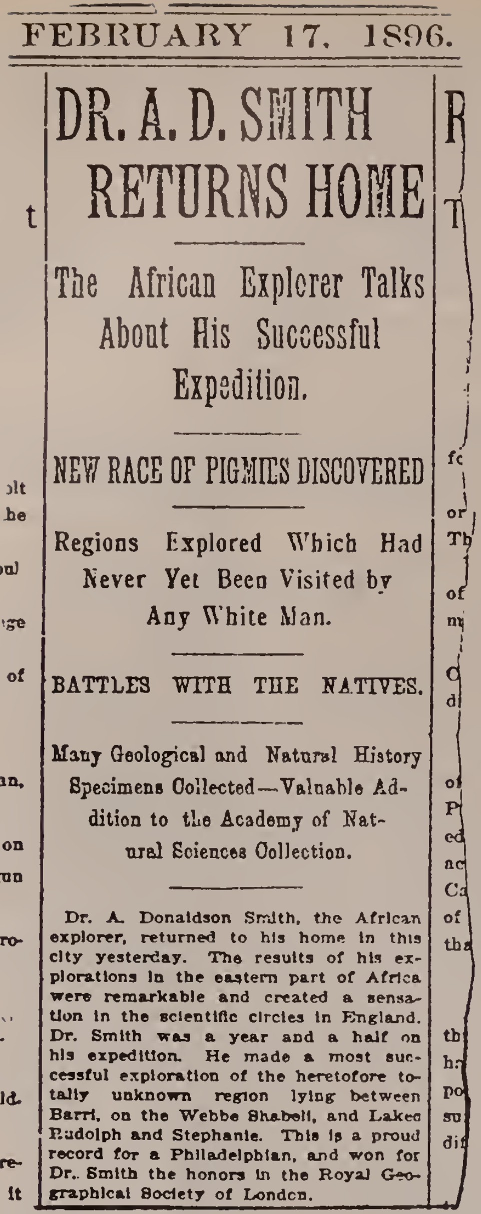
Smith left Great Britain from Southampton and arrived in New York aboard the *St Louis* on February 15, 1896.[31,32] He was met by members of his family and arrived in Philadelphia that afternoon.[32] He had been away almost two years. By any measure, his expedition had been an enormous success and he could well have rested on his laurels as an explorer for a lifetime. Unlike many other explorers of the day, his expedition had not been financially supported by any government, scientific society, or private commercial interests. He had spent £4,000 of his own money on it, which in today's currency would be equal to $220,000, close to 10% of his inheritance.[33]

Smith was honored and feted. "It has been an agreeable surprise to me that Americans in general have taken great interest in my expedition," he wrote on February 27, 1896, to J. Scott Keltie, secretary and editor of publications of the Royal Geographical Society in London.[7] In the same letter he said: "Many dinners and receptions have been given for me. I lecture before the Geographical Club in Philadelphia and New York soon."[7]

To a reporter for the *Philadelphia Press* he appeared "Bronzed and looking in excellent health"[32] (Fig 8). Henry Pleasants, Jr, MD, who was a nephew by marriage of Smith's sister, recalled his impressions as a young boy:

> It was quite something for us small boys to be even distantly related to a real live African explorer ... Personally, I was a bit disappointed when I saw him for the first time after his return. He had not grown two feet taller, and he was thinner than ever. His complexion was yellowish too. Father said it was because of malaria and quinine and bad food. Otherwise, he was just the same Uncle Donnie, as quiet and insignificant as ever....[34]

Young Henry Pleasant's perception of Smith as quiet and insignificant in the context of the larger Smith family is noteworthy. For this was the man who had courted maneating lions, outfoxed the cunning Ethiopians, and survived malaria and 4,000 miles of the African bush. His inheritance had provided him with material liberation and Africa with a horizon of freedom across which to trace his worth. Despite this dramatic demonstration of independence and accomplishment, back home in Philadelphia, his family's view of him seems to have remained unchanged.



FEBRUARY 17, 1896.

DR. A. D. SMITH RETURNS HOME

The African Explorer Talks About His Successful Expedition.

NEW RACE OF PIGMIES DISCOVERED

Regions Explored Which Had Never Yet Been Visited by Any White Man.

BATTLES WITH THE NATIVES.

Many Geological and Natural History Specimens Collected—Valuable Addition to the Academy of Natural Sciences Collection.

Dr. A. Donaldson Smith, the African explorer, returned to his home in this city yesterday. The results of his explorations in the eastern part of Africa were remarkable and created a sensation in the scientific circles in England. Dr. Smith was a year and a half on his expedition. He made a most successful exploration of the heretofore totally unknown region lying between Barri, on the Webbe Shabeli, and Lakes Rudolph and Stephanie. This is a proud record for a Philadelphian, and won for Dr. Smith the honors in the Royal Geographical Society of London.

FIGURE 8. Front-page story in the *Philadelphia Press* concerning Arthur Donaldson Smith's return to Philadelphia (courtesy of H. Spencer Potter, PhD).

GETTING INTO PRINT

Smith's major concern during the early months of 1896 was with the delay in the publication in the *Geographical Journal* of his maps and the oral presentation he had made before the society on January 6, 1896. This is reflected in his February 27 letter to J. Scott Keltie: "No proofs yet received. I do not give my permission to have maps published before I see and comment proofs."[7] Smith was clearly upset by the delays in producing his article and maps and was not in the best of health either. "My eyes are in a bad way," he wrote.[7] Keltie was surprised to learn of Smith's differences with Gillett during the trip and tried to mollify Smith about the delay in the proofs of his article. "I am surprised . . . I had no idea there was any difference between you and Gillett, otherwise I would not have asked him for the photos . . . if you wish I shall correct the illustrations . . . ." Keltie continued, ". . . of course a proof of the map will be sent. I can post a print very shortly. Let me have it back without delay. . . ."[35]

Smith received the proofs for his paper in late March, but not the proofs for the maps. On April 2, 1896, he wrote to Keltie, saying, ". . . I did not write immediately as the map proofs have not come . . . I hope to get the maps soon—it is getting late for them. There is plenty of work for me now getting my book ready but I feel in good condition, no more malaria or eye trouble. . . ."[36] In this same letter Smith told Keltie, ". . . please forget what I said about Gillett and the photographs and insert the three pictures where they belong,—in the beginning . . . ." Smith ended his letter on an upbeat note: ". . . Golf is quite the rage in Philadelphia as well as biking. . . ."[36]

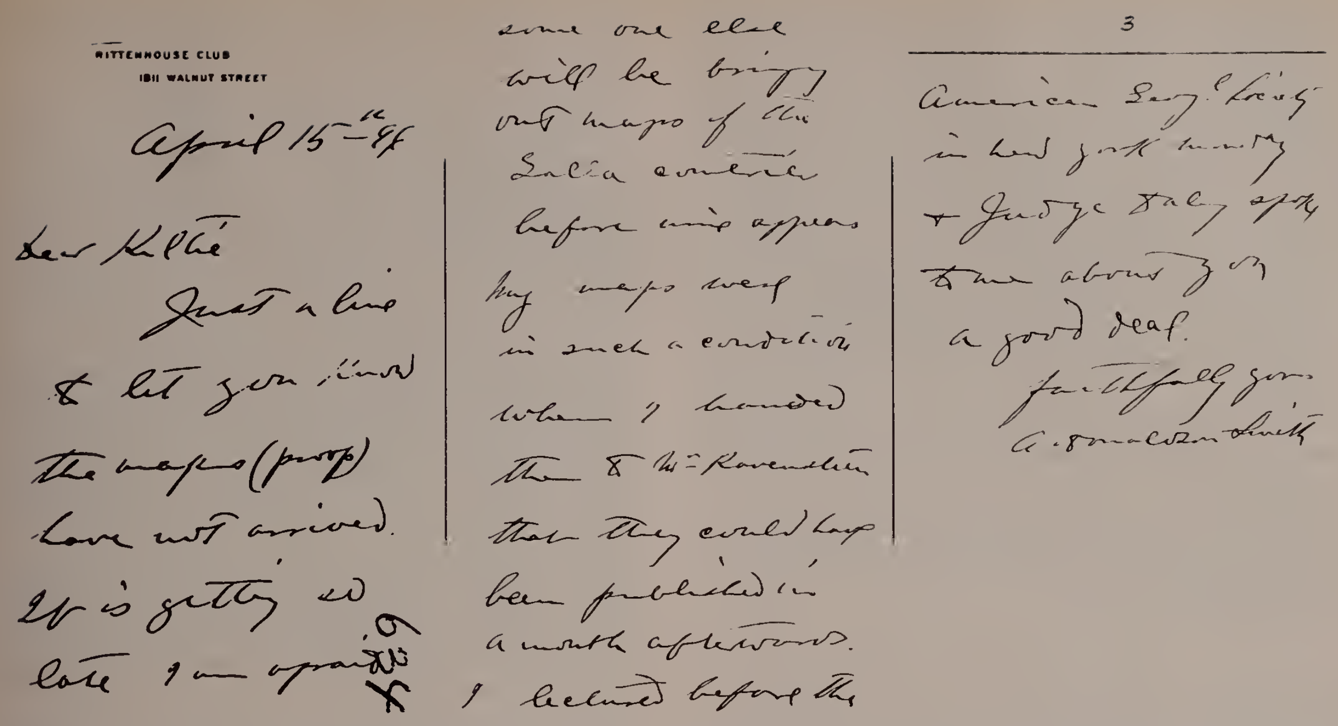
On April 15, Smith fired off another letter to Keltie (Fig 9):

> . . . the maps (proofs) have not arrived. It is getting so late I am afraid some one else will be bringing out maps of the Galla countries before mine appears. My maps were in such a condition when I handed them to Mr. Ravenstein that they could have been published in a month afterwards.[37]

Although the Royal Geographical Society seemed to Smith to be dragging its feet in publishing the results of his expedition, he himself was active in disseminating word of it in the United States. ". . . I lectured before the American Geographical Society in New York Monday and Judge Daly spoke to me about you a good deal . . . ."[37] Judge Charles P. Daly was the president of the American Geographical Society and would have known J. Scott Keltie. Smith may have been putting some pressure on Keltie to move his publication along as no doubt he discussed the publication delay with Judge Daly. However, proofs of the maps had still not arrived by June 17, when Smith sent his longest (eight pages) and strongest letter to Keltie. "Mr. Ravenstein's last letter to me stated that the proofs of my maps would be sent at once . . . no maps have since appeared . . . The R.G.S. Journal for June has reached me. Not a word has been inserted regarding my expedition . . . I feel most discouraged."[38] Smith had good reason to be discouraged with the slow pace of producing his article.

The world, however, was well aware of his accomplishments. The Philadelphia and New York City newspapers carried numerous stories about Smith and his expedition. His lecture before the American Geographical Society in Chickering Hall was attended by a large audience and covered by the papers and again his description of Dume dwarfs aroused much excitement. The Dume were not and are not dwarfs. It was that Smith had encountered some Dume who were short in stature. However, his report fed preconceptions about the distribution of pygmies in Africa, based on the reports of early explorers, including Krapf and D'Abbadie, which had been collated in 1891 by Dr Henry Schlichter of the British Museum.

Smith had a soft, monotone voice that did not carry far. Henry Pleasants recalled his father's impression of Smith's address before the Academy of Natural Sciences of Philadelphia: ". . . Father said that he acted as if he would rather have faced a wounded bull elephant than the audience. His knees shook so hard that anyone could see them; and his voice could not be heard farther back than the third row . . . ."[34]



RITTENHOUSE CLUB
1811 WALNUT STREET

April 15th 96

Dear Keltie

Just a line & let you know the maps (proof) have not arrived. It is getting so late I am afraid some one else will be bringing out maps of the Galla countries before mine appears. My maps were in such a condition when I handed them to Mr Ravenstein that they could have been published in a month afterwards. I lectured before the

3

American Geog. Society in New York recently & Judge Daly spoke there about you a good deal.

faithfully yours
A. Donaldson Smith

FIGURE 9. Letter written by Arthur Donaldson Smith, MD, to J. Scott Keltie, editor of the *Geographical Journal* (courtesy of the Royal Geographical Society).

Smith, who had shot 13 elephants and 20 rhinoceri, suffered stage fright to the degree that he avoided public speaking if he could. Henry Pleasants noted, "... he always gave the excuse that he was too busy writing his book."[34]

His primary concern was getting his findings into print in the *Geographical Journal*, which for him was where it mattered most.

The *Geographical Journal*, which made its appearance in 1893 under Keltie's editorship, published both written articles and articles derived from presentations made before the society by explorers, travelers, and others. J. Scott Keltie (1840–1927), whose association with the society began in 1884, was a distinguished and respected editor and writer. It was he who reorganized the society's publications and improved their quality.[39] In the 12 issues of the *Geographical Journal* published in calendar year 1896, a total of 17 articles were derived from presentations made before the society. On careful analysis this writer found that with the exception of a two-part series entitled, "The Pamirs and the Source of the Oxus," by George N. Curzon, the time lag from oral presentation to publication ranged from one to eight months, with an average of four months. Curzon, who later became Viceroy of India (1898–1905), was then under-secretary for foreign affairs in Lord Salisbury's government.[40] His two-part article appeared 17 months after his oral presentation. This must be viewed as a striking exception perhaps due to his not writing it up more quickly. Smith's article eventually appeared in two parts, in August and in September—seven and eight months, respectively, after his oral presentation. Thus the time lag for Smith's article was close to double the average (excepting Curzon's article). Was the Royal Geographical Society, an often cooperative partner with British Imperial aims, suppressing Smith's findings? Smith seemed to have thought so, at least during the period before publication. He wrote, "... my report should have been published earlier. I feel most discouraged ...."[38] Smith then spoke of having another map maker draw his maps and expressed suspicions of Ravenstein purposely holding up publication. Smith said that on receiving his note books made in the field, Ravenstein said, "... why, you deserve a medal ...."[38] Smith went on: "... now I venture to say, after he has gotten from me all the information I could give, he is against my getting any credit. Or else why would he have not published my maps at once?" He continued, saying, "... Mr. Ravenstein made another little remark to me to the effect that most of the tribes I discovered had been already heard of ...."[38]

Smith poured his heart out to Keltie, whom he obviously trusted. "... I am very 'blue' at the manner in which the R.G.S. has passed over my work ...." He then went on to describe for Keltie the accomplishments and discoveries of his expedition. He ended by saying, "... I shall go to some remote country next winter, what can you suggest? What will the R.G.S. give me to go to the Omo River? You will not expect me to give my results to the R.G.S. again, solely for a loan of instruments after the treatment my present maps have received ...."[38]

Smith's letter of June 17 crossed with the proofs of his maps, which arrived from London on June 20, 1896.[41] He hastened to write to Keltie four days after his letter of June 17.[41] "Proofs of my maps have arrived (yesterday) and I am greatly pleased. Very sorry I wrote you a few days ago when I had a fit of depression. Mr. Ravenstein has worked well to oblige me as well as to attain accuracy in the five maps he sent me. I have not seen the index map, but I cabled Mr. Ravenstein to publish all, on faith."[41]

Smith was clearly very happy with the maps and cheered by the fact that his work would soon be published. He quickly passed from the issue of the maps to future travel. ". . . Make me some propositions or suggestions about another expedition—preferably Somaliland or Gallaland. Could anything be done in a yachting cruise around the world? I have friends who would like me to go on a yacht—to give them points about exploring but I do not know where I could go. Any good islands? I am very much inclined towards my old country by Lake Rudolf . . . ."[41]

Keltie could not let Smith's highly accusatory letter of June 17 go unanswered. In his reply he said to him: "Dear Donaldson Smith: I cannot tell you how grieved I am that you should take so erroneous a view as to the attitude of the Society towards your work in Africa. The delay in the preparation of the maps is most annoying . . . ."[42] Speaking of Ravenstein, he said,

> . . . He is the best cartographer we have, and no one knows Africa so well as he does. We felt it was only justice to your admirable work to give the maps to the best man even at the risk of delay. Be assured this delay will do your reputation no injury . . . no one can . . . take away any of the credit which belongs to you. I am glad the maps are out of hand at last. Too late, unfortunately for the July *Journal*, but they shall go into the August number.[42]

Keltie clearly acknowledges that there was undue delay in bringing Smith's work out. And he explains that ". . . you are labouring under a serious misconception; and that the delay in the publication of your maps and paper is purely accidental. Rest assured that we place a high value on your work in Africa . . . ."[42] In this same letter, Keltie discusses the medals the society awards, saying ". . . They [the council of the Society] have their eye on you, and you may depend upon it that when you complete your work on the Omo region, your services will not go unrecompensed. We have only two medals to award . . . ."[42] Keltie ended his long letter to Smith by encouraging him to apply to the society for assistance for his next trip to Africa.[42]

SMITH GETS PUBLISHED

Smith's article finally appeared in two parts, in the August and September issues of the journal,[8,9] the maps being published with the first installment. The three Gillett photographs appeared in the beginning of the first installment. The latter was the second article in the issue, appearing after Part II of Curzon's article on the Pamirs and the Oxus. Smith's second installment was again the second article, appearing after one on "the Hausa Territories" by Charles H. Robinson, and followed by the third installment of Curzon's article. Taken on merit alone, Smith's work should have been treated as a lead article. However, Curzon was a powerful political figure, a member of parliament and the cabinet, a renowned traveler, and a vice-president of the society. One can understand why Smith's first installment followed Curzon's article. However, it is less explicable why the second one came after an article on what is now northern Nigeria. The delay in publication appears to have been due to the time that Ravenstein required to draw the five maps. These are detailed, covering much territory hitherto unexplored, and Ravenstein, being meticulous, took great care in preparing them.

There is no evidence that the Royal Geographical Society actively sought to suppress or diminish the importance of Smith's accomplishments. The facts speak to the contrary. The Society's leadership was extremely excited about Smith's trip and gave it considerable coverage in the *Geographical Journal* before his return. A preliminary report of the trip covering its early stages was published in 1895, based on information sent by Smith from the Webi Shebeli.[43] A detailed map (Fig 10) accompanied this communication. In December 1895, the president of the society in his annual address enthusiastically referred to Smith: "The news of the success of the enterprise of Dr. Donaldson Smith in Africa is most welcome. He has penetrated to the shores of Lake Rudolf from Somaliland, in the face of great difficulties, and has reached the coast at Lamu; so that we may soon expect to welcome him in this country."[44] Smith's arrival in Aden after his trip was published in an extensive notice in the monthly record of the *Geographical Journal*.[45] In part it read: ". . . Early in November Dr. Donaldson Smith reached Aden, having completed what has been apparently a most successful journey in the Somali and Galla countries . . . . "[45]

Smith voiced no complaints about the position of his article in the journal. He wrote to Keltie on August 20, 1896, saying: ". . . many thanks for your kind letter and for the *Geographical Journal*. Your letter put me in a very happy frame of mind . . . I am much pleased with the first part of my article appearing in a separate Journal with the maps . . . ."[46]

FUTURE PLANS AND RIVAL EXPEDITIONS

In his letter of August 20, 1896, Smith told Keltie that he did not yet know what to say about the Omo.[46] In it he said that he had heard from Dodson, the taxidermist, who was in Somaliland with Professor Elliott. Dodson had told him that camels were scarce and costly in Somaliland.[46]

Smith demonstrated increasing concerns about the costs of a new expedition, probably because his own assets

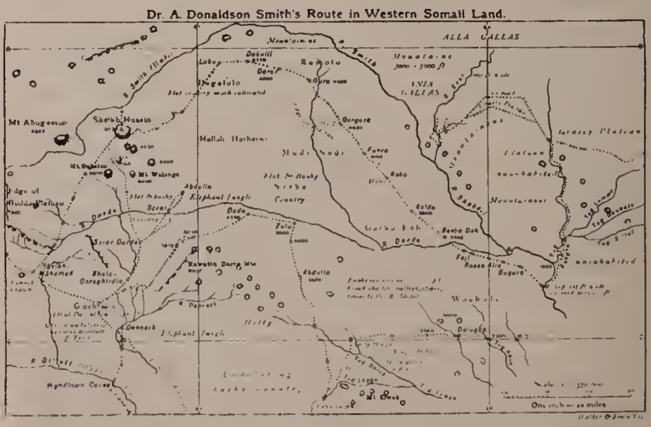


FIGURE 10. Preliminary map of Smith's journey through western Somaliland showing the Wyndlawn Caves (Sof Omar). Smith named the Web River after Fred Gillett and the upper reaches of the Webi Shebeli after himself (reproduced from the *Geographical Journal*, 1895; 5:124–127).

were not limitless. In his letter written from DeBruce, Pennsylvania, he said:

> I hope to be able to get once more in the Rudolf districts with aid from the R.G.S. I shall go to England next autumn—probably in October and will have a talk with you before doing anything definite. Perhaps I may run off to India for a couple of months with the two Messrs. Farnum whom you know. I can then stop at Aden and feel my way towards an African expedition. I shall put in a good deal of money myself if the R.G.S. will also chip in a like amount. The expedition to the Omo or west of Rudolf would be expensive . . .[48]

As 1896 drew to a close, Smith's plans for returning to Lake Rudolf to resolve the issue of the Omo River became more firm. He obviously had no plans for resuming the practice of medicine. He was also busy preparing his book for publication, writing to Keltie of problems he was encountering with the publisher, Arnold of London.

> . . . I have had much trouble with my book because Arnold is on one side of the Atlantic and the printer on the other and the printer delights in delaying . . . It is most gratifying to hear that the R.G.S. may probably assist me financially in my African expedition . . . P.S. I am quail shooting for a few days in North Carolina.[47]

Smith told Keltie that the society would have to pay two-thirds of the expenses of an expedition to the Omo and that he would assume responsibility for a third. "If you think the R.G.S. will pay two-thirds of the expenses if I undertook to solve the Omo question (or else explored between the Nianam and the Nile) please let me know at once and I will give up the Corea trip."[48] Smith put the total cost at £3,000. He seems to have known that the Omo issue might get resolved before he got back to the region and thus he put forth a back-up proposal of exploring the unknown area between the lake and the Nile. On January 3, 1897, Smith wrote to Keltie that he planned to go to Korea and Manchuria in February with his friend Edward Farnum.[48] In this same letter he said he had heard from Dodson that many of his "... old boys are away with Mr. Cavendish."[48] H.S.H. Cavendish was a wealthy young British aristocrat, the nephew of Lord Hartington (Duke of Devonshire), who after two years' shooting and sightseeing in South Africa decided to explore the western shores of Lake Rudolf. Cavendish and his companion, Andrew, retraced much of Smith's route, setting out from Berbera on September 5, 1896, and arriving at the northern end of Lake Rudolf on March 3, 1897.[49] Smith knew of the Cavendish expedition from Dodson, who along with Professor Elliot encountered it on its way into the interior.[41] What he did not yet know was the extent of area explored by the expedition nor the fact that the Italian explorer Vittorio Bottego had arrived on the northern shores of Lake Rudolf on September 1, 1896, and had definitively resolved the issue of the Omo River as being the principal source of the lake.[14] However, the results of the Bottego and Cavendish expeditions were not yet known.

Keltie wrote to Smith on February 2, 1897, saying that the Council of the Society were well disposed to Smith's plans but that they could not at this point vote the funds.[50] What he did not spell out, but which Smith certainly was aware of, was that the upper Nile region and the borders between Ethiopia and the British spheres of influence were rapidly becoming the interfaces for confrontation between France and Britain, each out to advance their colonial aims which could only be achieved at the cost of the other.[1(pp369-374)] In all this Ethiopia was playing a major role. Keltie was being euphemistic when he said, "... the regions between Somaliland and the Nile will be in a disturbed state . . . ."[50] None of this seems to have deterred Smith, who received Keltie's letter in New Orleans where he had stopped enroute to San Francisco.[51] He was aware of the political problems because he wrote, "the position of England and Abyssinia may be changed before next winter and there may be other political considerations that will affect the prospects of success in elucidating the Omo question etc. . . . ."[51] Smith again reiterated,

> . . . I have some hopes of the R.G.S. assisting me. If I find that an expedition will be feasible and productive of good results, I will then go to London and lay my plans in detail before the Council. I saw that Captain Bottego and a Mr. Neumann have both reached Lake Rudolf. You would do me a great favor if you would send me any accounts of their journeys if you think they bear on the question that is nearest my heart.[51]

Neumann's trip was not of significance as far as geographic discovery was concerned, although his book is a rich source of geographic description and accounts of people and fauna.[13] However, Bottego's discoveries had, unknown to Smith, settled the issue of the Omo River and Lake Rudolf's source, thus removing the need for a return trip for that purpose.[14] A relative profusion of expeditions was about to unfold in the Lake Rudolf region, primarily launched to further political interests.

The Cavendish Expedition

Cavendish's expedition was undertaken for both sport and geographic discovery. He traveled down the western shore of Lake Rudolf, which no one else had done before, while Lieutenant Andrew, his companion, went down the eastern shore. Their's was the fifth expedition to reach the lake, the others being Teleki's, Smith's, Neumann's, and Bottego's.

During 1897 and 1898 French-British rivalry over the Nile intensified, culminating in the Fashoda Incident of 1898. This rivalry involved not only the Nile but also adjacent areas such as that where Lake Rudolf is located. The intensity of the feeling over this issue gave rise to strident jingoism in both countries. This is evidenced by the comments made by Major Frederick D. Lugard on January 31, 1898, following Cavendish's speech before the Royal Geographical Society. Later, as Lord Lugard, he would become a legend in British Africa, best known for his concept of indirect rule. Speaking of Cavendish's trip he said:

> . . . and the more remarkable and the more pleasing to us because he is the first Englishman who has transversed that country—his predecessors, one of whom we welcomed here, were Dr. Donaldson Smith and Signor Bottego . . . they have done excellent work, but they are not of our own nationality. The country explored is part of our own British East Africa Protectorate, and therefore, I think, the more British explorers that go into the country the better . . . He was very reticent about those who had gone before

> him, but we gather that he met with less hostility than they had done.[49]

Lugard was clearly blinded by his own jingoism. Cavendish's geographic accomplishments were modest. True, he had been the first to travel down the western shore of Lake Rudolf. However, as Butzer cogently observes, his map of the Omo delta was pretty much adapted from Smith's, except for his original sketch of Sanderson's Gulf.[52] Cavendish's very modest natural history collection received faint praise from Dr R. Bowdler-Sharpe of the British Museum at the same meeting.[49] No one bothered to say that Cavendish and Andrew apparently spent more effort shooting elephants and collecting ivory and widely spoke of how much money they hoped to make from their haul when they arrived in Fort Smith in the British East Africa Protectorate.[18(p24)]

This was not all. Lugard insinuates that Teleki and Smith were somehow deficient because they encountered native hostility and that Cavendish was being polite in not speaking of this. He gathered that Cavendish had met with little hostility, implying that this somehow reflected superior skill. Although Cavendish mentions the difficulties he had with the Turkana, his conduct toward them was only brought to light later. Herbert H. Austin, a British explorer who later traveled along the western shore of Lake Rudolf, sharply criticized Cavendish for the "disgraceful way in which [Cavendish] had looted and plundered" the Turkana.[53] While Cavendish had somewhat minimized the seriousness of his armed conflicts with the Turkana in his presentation before the society, he had candidly told Reuter's in Zanzibar that "we had fighting every day and night . . . until we reached the south end of the lake."[54]

Rival explorers often expended a good deal of effort describing their own easy passage through areas where their predecessors encountered armed hostility. Missing from many of these accounts is a credible explanation of how the hostility began and why. In general, African groups had every reason to be concerned about large caravans traversing their country. In the Lake Rudolf region they knew nothing of the explorers' intentions and had in the past been abused by either trading caravans or Ethiopian raiding parties. Provocation was often subtle at first, prompting a stronger reply until, through escalation, both sides were fighting. The effect was often salutary for subsequent travelers, the locals having learned a lesson the hard way.

The responsive bashing Smith gave to the Boran made Cavendish's passage through this country easier. Yet Cavendish, who describes an easy passage through Boran country, behaved badly there, according to Lord Delamere.[53] The latter came down the same way a short while later and complained to the foreign office about Cavendish's conduct in the Boran country.[53]

Cavendish himself made no excessive claims about his trip. He was a young sportsman out for shooting and some exploration. It was people such as Lugard who exaggerated the importance of this trip to serve political ends. Lugard had reasons for doing this aside from jingoistic feelings. While Britain claimed the area around Lake Rudolf as being in its sphere of influence, the claim had never been secured through treaties with local chiefs. However, this loose end was soon to be tidied up by the Macdonald expedition and specifically by Herbert H. Austin, who under the guise of leading a scientific geographic effort would sign treaties and hand out Union Jacks in the Lake Rudolf region.[53]

BRITISH AIMS IN THE LAKE RUDOLF REGION

Britain's main concern in 1897 was securing the entire course of the Nile. In order to accomplish this the Mahdi's state in the Sudan had to be toppled and French advances from the west and through Ethiopia from the east had to be checked. It was British policy to avoid confrontation with Ethiopia while these larger Nile-related interests were being promoted. Menelik, the Ethiopian emperor, possessed a formidable military force. Britain made diplomatic efforts to see to it that no meaningful alliance developed between Ethiopia and the Mahdi's state. A union of these two military forces could have stalled British advances on the Nile and left gaps open for the French, who were ready and able to seize them.

Cavendish's trip provided the British with some diplomatic and legal grounds for their claims. However, all the bellowing and puffery about it were no substitutes for treaties and fluttering Union Jacks.

SMITH'S PLANS CONFLICT WITH POLITICAL AIMS

A purely scientific expedition of the kind that Smith now proposed was in a sense out of step with Britain's principal interests in the region which were political. This alone should have alerted Smith to the fact that the Royal Geographical Society, whose patron was the Queen and whose vice-patron was the Prince of Wales, would respond in a benign and vague manner to his requests for financial support. Although he was in China, Smith's mind was still on Lake Rudolf. On May 20, 1897, he wrote to Keltie from San Ho, 15 miles north of Peking,

> . . . I shall be most anxious to hear of the English mission to Abyssinia; the result of Bottego's journey and whether any explorers are doing or are trying to do the Omo district, the country to the west of Rudolf, and the southern Boran plains. You would do me a great favor if you write me on these points about August first and I will find the letter when I come out in September.[55]

Referring to his recently published book he said, ". . .I do not know much about the fate of my book excepting that it has been favorably criticized in the *Times* and in most of the English papers . . . Arnold, I think has done well with it, and brought it out in a sumptuous style."[55]

Henry Pleasants, Jr, says of Smith's book that ". . . the sale of the book had been limited to interested friends. Uncle Donnie had presented copies to about all of the big scientific organizations he could think of. Ordinary people did not seem to be interested particularly; if they were, they borrowed the book from a public library."[34] Yet Smith's book received extensive reviews in both the United States[56-58] and Britain.[59,60] It rapidly became a classic of 19th century African travel and exploration and has been widely cited by scholars over the decades.[52,61] A 1969 US reprint is still in print.[1]

The odds were heavily stacked against the possibility of Smith ever returning to the Lake Rudolf area and accomplishing anything new and unique. Away in China, he was cut off from the details of the complex web of events that were now engulfing this part of Africa. Perhaps it was just as well. That he did ultimately return and succeed in exploring the region between the lake and the Nile is a measure of his ingenuity, resourcefulness, and courage.

REFERENCES

1. Smith AD: *Through Unknown African Countries: The First Expedition from Somaliland to Lake Lamu*. London and New York, Edward Arnold, 1897, reprinted, New York, Greenwood Publishers, 1969.
2. Letter from A. Donaldson Smith, MD, to John Coles, Hargeisa, Somaliland, July 19, 1894. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
3. Von Höhnel L: *Discovery of Lakes Rudolf and Stefanie: A Narrative of Count Samuel Teleki's Exploring and Hunting Expedition in Eastern Equitorial Africa in 1887 and 1888*, Bell N (D'Anvers) (trans). London, Longmans, Green & Co, 1894, vols 1, 2.
4. Chanler WA: *Through Jungle and Desert; Travels in Eastern Africa*. New York, Macmillan and Co, 1896, reprinted, Detroit, Mich, Negro History Press, 1970.
5. *The Caves of Sof Omar*, Addis Ababa, Ethiopian Tourist Organization, 11 pages plus maps, no date (courtesy of D. Strother Pope, MD, Columbia, SC, 1982).
6. The Amharic to English translation of the letter was arranged by my cousin Sister Martha Casertano and Sister Marguerite Petraz, of the Daughters of the Heart of Mary, both of whom worked in Ethiopia, the former for 15 years. The letter was then taken to Addis Ababa by the Reverend Pierre Gouin, SJ, of the Centre Vimont, Montreal, who supervised its translation into English by Taye at Haile Selassie University.
7. Letter from A. Donaldson Smith, MD, to J. Scott Keltie, Philadelphia, Pa, February 27, 1896. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
8. Smith AD: Expedition through Somaliland to Lake Rudolf, Part I. *Geographical J* 1896; 8:120-137.
9. Smith AD: Expedition through Somaliland to Lake Rudolf, Part II. *Geographical J* 1896; 8:221-239.
10. Bruce-Chwatt LJ: *Essential Malariology*, ed 2. London, Heineman, 1985, pp 2-3.
11. Imperato PJ: Early modern medical services at Moyale, Kenya. *East Af Med J* 1983; 6:367-373.
12. Cufino L: Riccordi della seconda spedizione Ruspoli, Il diario del dott. Carlo Riva. *Bull Soc Geog Ital* 1912; 49:33-47.
13. Neumann AH: *Elephant-Hunting in East Equatorial Africa*. London, Rowland Ward, 1898, p 259, reprinted, New York, Abercrombie & Fitch, 1966.
14. Vannutelli L, Citerni C: *Seconda Spedizione Bottego, L'Omo: Viaggio d'Esplorazione nell'Africa Orientale*. Milano, Ulrico Hoepli, 1899.
15. Johnson O: *Four Years in Paradise*. Philadelphia, JB Lippincott, 1941.
16. Archer G: British East Africa. I. Recent exploration and journey in the North of British East Africa. *Geographical J* 1913; 42:421-435.
17. Imperato PJ: Early modern medical services at Marsabit, Kenya. *East Af Med J* 1981; 58:769-776.
18. Trzebinski E: *The Kenya Pioneers*. New York, WW Norton & Co, 1986, p 171.
19. Paul J. Rainey, in *The National Cyclopedia of American Biography*. New York, James T. White & Co, 1926, pp 411-412.
20. Binks HK: *African Rainbow*. London, Sedgwick and Jackson Ltd, 1959, pp 108-117.
21. Johnson M: *Safari: A Saga of the African Blue*. New York, GP Putnam's Sons, 1928.
22. Dr. Smith is Safe. A cablegram announces the success attending the explorer's efforts. *Philadelphia Press*, November 5, 1896 (courtesy of H. Spencer Potter, PhD).
23. Gregory JW: *The Great Rift Valley. Being the Narrative of a Journey to Mount Kenya and Lake Baringo*. London, John Murray, 1896.
24. Sharpe RB: On a collection of birds made by Dr. Donaldson Smith during his recent expedition to Western Somaliland. *Proc Zoolog Soc* 1895, pp 457-520.
25. Boulenger GA: An account of the reptiles and batrachians collected by Dr. A. Donaldson Smith in Western Somaliland and the Galla Country. *Proc Zoolog Soc* 1895, pp 530-540.
26. Boulenger GA: Second report on the reptiles and batrachians collected by Dr. A. Donaldson Smith during his expedition to Lake Rudolf. *Proc Zoolog Soc* 1896, pp 212-217.
27. Gunter A: Report on a collection of fishes made by Dr. A. Donaldson Smith during his expedition to Lake Rudolf. *Proc Zoolog Soc* 1896, pp 217-224.
28. Letter from Arthur Donaldson Smith, MD, to Dr E. Gordon, Kaolin, Pa, December 11, 1914. A. Donaldson Smith File, The University Museum, University of Pennsylvania, Philadelphia.
29. Krapf JL: *Travels, Researches, and Missionary Labours During An Eighteen Years' Residence in Africa*. London, Trubner & Co, 1860, reprinted, London, Frank Cass and Co, 1968.
30. Letter from A. Donaldson Smith, MD, to J. Scott Keltie, Palace Hotel, San Francisco, Calif, March 2, 1897. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
31. *Social Register*, New York, Social Register Association, May 1896, p 35.
32. Dr. A. D. Smith Returns Home. The African Explorer Talks About His Successful Expedition. New Race of Pygmies Discovered. *Philadelphia Press*, February 17, 1896, p 1.
33. Letter from Percy Z. Cox to Colonel Martelli, Baroda, India, December 29, 1897. Sir Percy Cox Papers, Royal Geographical Society Archives, London.
34. Pleasants H Jr: Some horses are lucky. Biographical sketch of Arthur Donaldson Smith, MD. *The General Magazine and Historical Chronicle*, University of Pennsylvania General Alumni Society, XLVIII, 1946; 4:254-261 (courtesy of H. Spencer Potter, PhD).
35. Letter from J. Scott Keltie to Dr A. Donaldson Smith, London, March 9, 1896. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
36. Letter from A. Donaldson Smith, MD, to J. Scott Keltie, Rittenhouse Club, Philadelphia, Pa, April 2, 1896. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
37. Letter from A. Donaldson Smith, MD, to J. Scott Keltie, Rittenhouse Club, Philadelphia, Pa, April 15, 1896. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
38. Letter from A. Donaldson Smith, MD, to J. Scott Keltie, Rittenhouse Club, Philadelphia, Pa, June 17, 1896. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
39. Weaver JRH (ed): Sir John Scott Keltie (1840-1927), in *The Dictionary of National Biography*. London, Oxford University Press, 1937, pp 463-464.
40. Weaver JRH (ed): George Nathaniel Curzon (1859-1925), in *The Dictionary of National Biography*. London, Oxford University Press, 1937, pp 221-234.
41. Letter from A. Donaldson Smith, MD, to J. Scott Keltie, Kaolin, Pa, June 21, 1896. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
42. Letter from J. Scott Keltie to Dr Donaldson Smith, London, June 26, 1896. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
43. Donaldson Smith's expedition to Somaliland. *Geographical J* 1895; 5:124-127.
44. Markham C: Opening Address by the President, Session 1895-96, *Geographical J* 1895; 6:497-499.
45. Dr. Donaldson Smith's Somaliland expedition. *Geographical J* 1895; 6:568.
46. Letter from A. Donaldson Smith, MD, to J. Scott Keltie, DeBruce, Pa, August 20, 1896. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
47. Letter from A. Donaldson Smith, MD, to J. Scott Keltie, Hotel Lawrence, Scotland Neck, NC, November 9, 1896. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
48. Letter from A. Donaldson Smith, MD, to J. Scott Keltie, Rittenhouse Club, Philadelphia, Pa, January 3, 1897. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
49. Cavendish HSH: Through Somaliland and around and south of Lake Rudolph. *Geographical J* 1898; 11:372-396.
50. Letter from J. Scott Keltie to Dr A. Donaldson Smith, London, February 2, 1897. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
51. Letter from A. Donaldson Smith, MD, to J. Scott Keltie, Palace Hotel, San Francisco, Calif, March 2, 1897. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
52. Butzer KW: *Recent History of an Ethiopian Delta: The Omo River and the Level of Lake Rudolf*. Chicago, University of Chicago Department of Geography Research Paper No. 136, 1971.
53. Austin HH: Personal diary; February 2, 1898, to April 9, 1899, as cited by A. T. Matson in Introduction, *With Macdonald in Uganda*, by H. H. Austin, ed 2, Folkestone and London, Dawsons, 1973, p xv.
54. *Zanzibar Gazette*, December 1, 1897.
55. Letter from A. Donaldson Smith, MD, to J. Scott Keltie, San Ho, China, May 20, 1897. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
56. Review of *Through Unknown African Countries. Bookman: A Review of Books and Life*. 1897; 5:334-336.
57. Review of *Through Unknown African Countries. Dial* 1897; 22:249.
58. Review of *Through Unknown African Countries. Nation* 1897; 64:459-460.
59. Review of *Through Unknown African Countries. Athenaeum: A Journal of Literature, Science, the Fine Arts, Music and the Drama*. 1897; 109:371-372.
60. Review of *Through Unknown African Countries. Spectator* 1897; 79:460-461.
61. Von Höhnel L: The Lake Rudolf Region. Its Discovery and Subsequent Exploration 1888-1909. Part I. *J of the Royal African Society* XXXVII, 1938; 21-45.

Chemical perspectives in the design of a pediatric meningitis vaccine

CHRISTOPHER L. PENNEY, PhD

Haemophilus influenzae, type b (Hib), is responsible for up to 20,000 cases annually of meningitis in children.[1] A similar number of cases of sepsis, epiglottitis, pneumonia, septic arthritis, and osteomyelitis are also caused by this organism, along with a number of neurologic problems. Prophylaxis of these infections has been hampered by increasing bacterial resistance to antibiotics, and there is evidence of increased risk of infection in nursery schools and day care centers.[2]

In April 1985, the Food and Drug Administration (FDA) approved the universal vaccination of children to protect against Hib infection. The vaccine is a capsular polysaccharide consisting of ribose-ribitol units connected by phosphodiester linkages. (It is manufactured by Praxis Biologics under the name "b CAPSA 1.") However, this vaccine is poorly immunogenic and displays a T-independent response. It does not protect children under one year of age, the age group with the highest incidence of infection.[3]

Therefore, Hib semisynthetic vaccines are under development by at least three companies in the United States: Praxis Biologics (Rochester, NY), Merck and Company, Inc (Rahway, NJ), and Connaught Laboratories, Inc (Swiftwater, Pa). The approach is to covalently link the Hib polysaccharide antigen to a macromolecular (protein) carrier molecule, and thereby induce a T-dependent response. However, these candidate vaccines have not necessarily been formulated with a consideration of optimal chemical design.

SEMISYNTHETIC VACCINES

Two basic approaches have been developed in the design of polysaccharide antigen-protein carrier (semisynthetic) vaccines. The first approach is to activate the polysaccharide (with cyanogen bromide) and thereby create reactive (electrophilic) sites capable of reacting with a nucleophile on the carrier molecule, or a linker connected to this carrier. The second approach is to create a terminus within the polysaccharide by fragmenting the chain that contains a reactive group that can react with an amino group on the carrier, to form an imine linkage (Schiff base). This linkage is stabilized by reduction to an amine. Merck and Connaught have adopted the first approach, whereas Praxis has taken the second approach.

In the method reported by Connaught,[4] an adipic acid dihydrazide linker is covalently attached by a carbodiimide-mediated reaction to a diptheria toxoid carrier. The nucleophilic hydrazide from the linker is then reacted with the cyanogen bromide-activated polysaccharide. In the method described by Merck,[5] the cyanogen bromide-activated polysaccharide is reacted with the nucleophilic amino function of a caproic acid linker, and this complex is then covalently attached by a carbodiimide-mediated reaction to a specific serotype of outer membrane protein of *Neisseria meningitidis*. In the method described by Anderson (Praxis),[6] a reducing terminus is created by partial acid-catalyzed hydrolysis of a high molecular weight polysaccharide chain. The transient aldehyde that results from this cleavage is trapped by Schiff-base formation with an amino group from the protein carrier.

In the Merck patent,[5] it was noted that IgM to IgG seroconversion in mice could be observed only if the carrier was a serotype outer membrane *N meningitidis* protein. This is contrary to the T-dependent response that is usually induced by covalent linkage to an immunogenic protein carrier. While no data were reported for children, such data were recently published regarding the antibody response to the Connaught semisynthetic vaccine.[7] The vaccine is immunogenic and induces significant seroconversion, even among one-year-olds. Although the response is not clearly anamnestic[8] in adults, in two groups of infants receiving vaccine at three, five, and seven months, or at seven and nine months, long-term protection (seroconversion?) was demonstrated in 50% and 57%, respectively.[7]

Although this relatively low response rate might be attributed to the immature immune systems of these children, it could also be due to a less than optimal molecular design of the vaccine. Furthermore, a number of chemical considerations would indicate that reproducible preparation of a well-defined vaccine could be difficult to achieve by the cyanogen bromide-carbodiimide method. These chemical parameters, and the hypothesis of optimum antigen presentation, are described below.

From the Department of Chemistry and Biology, York University, Downsview, Ontario.

Address correspondence to Dr Penney, Agritech Research, Inc, 890 Harrington Ct, Burlington, Ontario, Canada L7N 3N4.

CONSIDERATIONS FOR A DEFINED SEMISYNTHETIC VACCINE

Activation of polysaccharide with cyanogen bromide creates "hooks" for attachment to a carrier molecule. However, these hooks are poorly reproduced and defined, and give rise to various linkages between the polysaccharide and the carrier. The hooks and resultant links can include nonreacted or partially reacted cyclic and acyclic imidocarbonates, acyclic nitriles, isoureas, and hydrolysis products (carbamates and carbonates). Some of these links are labile,[9] and gradual leakage of proteins from cyanogen bromide-activated polysaccharides has been reported.[10] Furthermore, some of these organic functions may give rise to unwanted antigens.

Symmetric linkers such as adipic acid dihydrazide may introduce unwanted cross-links, resulting in the creation of contaminating higher molecular weight structures. Unreacted linker molecules on the surface of the carrier can give rise to unwanted antigens.

The reaction of proteins with carbodiimides generates unwanted antigenic sites.[11] In addition, carbodiimides are known to cause cross-linking among proteins, again resulting in the creation of contaminating higher molecular weight structures.[12]

Thus, the cyanogen bromide-carbodiimide method of preparing semisynthetic vaccines is a random approach which results in chemical modification of the polysaccharide antigen and the protein carrier. This method can be expected to produce various unwanted antigenic structures and to yield a vaccine product of variable potency. These problems are further compounded when the protein carrier is a heterogeneous toxoid. It is not surprising, therefore, that preparation of a polysaccharide-protein carrier semisynthetic vaccine (such as a meningococcal-polysaccharide bound to a tetanus toxoid protein carrier) by this approach was reported to be difficult to reproduce.[13] In a recent study with human antisera,[14] it was shown that cyanogen bromide activation of *Haemophilus influenzae* type b polysaccharide, followed by attachment of the adipic acid dihydrazide linker, resulted in a change in the antigenicity of the polysaccharide.

These problems are not associated with the fragmentation-Schiff base method, which is characterized by the following: it creates one hook (chain terminus) with which to attach the polysaccharide to the protein; it does not employ linker molecules; and it does not produce random, unwanted antigenic sites. However, this method will result in the use of relatively smaller polysaccharide antigens. This might be considered a disadvantage, since it is generally observed that the immunogenicity of a pure polysaccharide is proportional to the molecular weight.[15]

Both the Merck and Connaught patents[4,5] describe the use of high molecular weight polysaccharides (1.1×10^6 daltons). This is thought to be necessary, especially for the immature immune system of the infant. However, with semisynthetic vaccines the problem may be at the molecular level, by virtue of the antigen's spatial relationship to the carrier surface. This conclusion leads to the hypothesis that a high molecular weight antigen is necessary only if the antigen is not presented in the optimal immunogenic form.

THE HYPOTHESIS

It is well known that antigens are substances of high molecular weight, whereas haptens are either nonantigenic high molecular weight substances or smaller molecules which become antigenic on covalent linkage to a macromolecular carrier. Dinitrophenyl linked to albumin is a classic example. Therefore, in the design of a semisynthetic vaccine, if the polysaccharide is viewed as a hapten, and is covalently linked in such a fashion to a protein carrier, it could become an effective antigen without being larger than the carrier molecule.

In fact, a number of articles confirm that the carbohydrate need not be an oligosaccharide or polysaccharide in order to function as a hapten. A hexasaccharide isolated from *Streptococcus pneumoniae* when coupled to various protein carriers was able to induce an anamnestic response;[16] the antibodies were protective. IgG monoclonal antibodies have been raised against a glucose-containing tetrasaccharide by coupling to various protein carriers.[17] Covalent attachment of lactose to albumin can give an anti-lactose antibody response.[18] Interestingly, the antibodies were cross-reactive with lipopolysaccharide of *Neisseria gonorrhoeae*. Disaccharides and trisaccharides composed of glucose and mannose, again coupled to various protein carriers, elicited antibodies cross-reactive with polysaccharides carrying the corresponding epitope pattern.[19] Even a monosaccharide (3,6-di-O-methyl-β-glucopyranoside linked to albumin) mimicked the behavior of a natural glycolipid antigen, as regards the ability to bind antisera from patients with leprosy.[20]

The common denominator with these small carbohydrate haptens coupled to a protein carrier is that the haptens are linked through their terminal carbon atom (the anomeric center), and not via random linkages along the chain. Such linkage at the chain terminus may provide an effective concentration of the same molecular antigen, as opposed to random linkage along the chain, which would give rise to more than one antigenic species. There is no evidence that a small carbohydrate hapten of *Haemophilus influenzae* type b linked through a terminal carbon would not elicit a protective antibody response. In fact, some proof to the contrary stems from Anderson's work.[6] In these studies, virtually the same antibody response is observed from small, medium, and large chain Hib polysaccharides terminally linked by the fragmentation-Schiff base method. More recently, the same method of vaccine preparation was employed with an Hib oligosaccharide (consisting of three to ten ribose-ribitol units) in a study with eight two-year-old children.[21] A seroconversion rate of 100% was observed. With the same vaccine, and in a study with infants immunized at two, four, and six months of age, 86% seroconversion was observed.[22]

The magnitude of the antibody response in this latter study was similar to that observed with the high molecular weight Connaught semisynthetic vaccine.[7] In fact, an even greater antibody response has recently been reported with infants, two to six months of age, immunized with a Merck vaccine candidate presently under clinical evaluation.[23] Merck has apparently abandoned the cyanogen bromide-carbodiimide approach for a better-defined conjugation technique. Investigators at Merck now report that the use of cyanogen bromide is not uniformly effec-

TABLE I. Summary of Polysaccharide-Protein Conjugate, Semisynthetic Vaccines Under Consideration for Manufacture for the Prevention of *H influenzae* type b infection

Manufacturer	Polysaccharide; Protein Coupling Reaction	Carrier Protein	Spacer
Praxis			
1st vaccine	Reductive animation, transient aldehyde in equilibrium with acetal at the anomenic center of ribose	CRM; a mutant (nontoxic) diphtheria toxin	None
2nd (newest) vaccine	Reductive amination, permanent aldehyde		None
Merck			
1st vaccine	Cyanogen bromide; carbodiimide (ethyl diaminopropylcarbodiimide)	Outer membrane protein (*N meningitidis*)	6-aminocaproic acid (6 carbons)
2nd (newest) vaccine	Carbonyl diimidazole; thiolactone		Peptide (at least 11 carbons, and thioether link from homocysteine)
Connaught			
1st vaccine	Cyanogen bromide; carbodiimide (ethyl diaminopropylcarbodiimide)	Diphtheria toxoid	Adipic acid dihydrazide (6 carbons)

tive[24] and leads to formation of unstable products,[25] so they have replaced a caproic acid linker[5] with a longer, synthetic peptide thioether linker. The advantage of this linker is that it allows a chemical means by which to confirm that a covalent bond has formed between the hapten and the carrier protein. They have used a high molecular weight polysaccharide hapten, with linkage along the chain to the carrier, as in the earlier vaccine.[5] Investigators at Merck retained the outer membrane protein of *Neisseria meningitidis* as a carrier, and they have noted that it is a T-cell stimulator.[25] Therefore, while this vaccine may not produce the best seroconversion or antibody response, according to the hypothesis, it is too early to tell if the observed response is or is not a result of the immunostimulatory protein carrier.

Nonetheless, acid-catalyzed hydrolysis should not be seen as the only method of creating a hook at the chain terminus. Another chemical method is under development whereby a more stable hook (permanent aldehyde) is created under neutral conditions, namely, through oxidation of polysaccharides with sodium metaperiodate.[26] In a current study, infants immunized at two, four, and six months of age with this Hib oligosaccharide (consisting of 20 ribose-ribitol units) coupled with diphtheria toxoid in a semisynthetic vaccine produced the best antibody response yet observed.[27,28] As a result, this will be a model for the vaccine that Praxis will develop. Therefore, in accordance with the hypothesis presented in this article, if optimal antigen presentation as well as high chemical specificity are to be achieved, then the fragmentation-Schiff base method should be used in the preparation of carbohydrate semisynthetic vaccines.

The Protein Carrier

In the classical approach to bacterial vaccine production, either whole cells or exotoxins are inactivated. These are not pure preparations, and the immunogenicity often increases as the purity of the preparation decreases.

The process of creating impure toxoid antigens with aldehydes (potential cross-linking agents) increases product heterogeneity. This occurs in the treatment of tetanus toxoid with formaldehyde.[29] While such impure antigens have long served as vaccines, the role of these materials as carriers must be questioned. Such complex preparations can become more impure as they undergo chemical transformation. Therefore, diphtheria and tetanus toxoids, on reacting with reactive carbodiimides, can give rise to byproduct formation, and the possibility of cross-link formation becomes more probable.

An interesting alternative has been described that uses diphtheria toxin from mutant strains of *Corynebacterium diphtheriae* (CRM 45[30] and CRM 197[6]). This material is immunologically active but biologically inactive, and so does not have to be reacted with aldehydes during vaccine manufacture. The result is a homogeneous protein carrier. Although vaccine regulatory agencies might be reluctant to employ a toxin as a toxoid, it should be noted that purified diphtheria toxoid is capable of reversion.[31] Whatever the choice, appropriate quality control must be instituted to guard against residual toxicity. Furthermore, this method does not preclude the use of a reasonably purified toxoid in the fragmentation-Schiff base procedure, or the use of another homogeneous carrier protein.

Conclusions

Haemophilus influenzae type b is the most important cause of meningitis in young children, as well as being the causative agent for other infections. This has resulted in licensing by the FDA of the first new vaccine recommended for universal use in the United States since 1969. However, the vaccine, a capsular polysaccharide, has been shown to prevent meningitis in only 60% of cases. It is poorly immunogenic, and displays a nonmemory response.

To meet this challenge, second generation vaccines are under development. They have not always been formulated with a view towards optimum chemical design. Therefore, attention is focused on the parameters that could prove important in the manufacture of these new vaccines. A hypothesis is presented that describes the optimum antigen presentation in terms of immunochemical behavior observed in similar nonvaccine antigens. These considerations would allow the design of reproducible and effective vaccines for use in pediatric immunization programs.

Acknowledgments. The author thanks Dr Silvio Landi, Connaught Laboratories Ltd (Canada), for useful discussions and his

thoughtful reading of this manuscript, and Dr Harold Jennings, National Research Council of Canada, who previously recognized the disadvantages of the random (activation) preparation of semisynthetic vaccines.[32,33]

REFERENCES

1. Marwick C: FDA soon to license H influenzae vaccine [news]. *JAMA* 1985; 253:1232–1233.
2. Katz SL: b-CAPSA I vaccine; An overview clinical monograph. Evansville, Ind, Mead Johnson and Company, 1985.
3. Fox J: Vaccine programs; poised for progress. *ASM News* 1985; 51:272–276.
4. Connaught Laboratories Inc: a) European patent application 1984, 98-518-A. b) World Patent, 1984, 8400-300-A. c) US Patent, 1985, 4996-538-A.
5. Merck and Company, Inc: a) European Patent Application, 1984, 117-783-A. b) US Patent, 1984, 4459-286-A.
6. Anderson P: Antibody response to Haemophilus influenzae, type b and diphtheria toxin induced by conjugates of oligosaccharides of the type b capsule with the non-toxic protein CRM197. *Infect Immun* 1983; 39:233–238.
7. Eskola J, Kayhty H, Peltola H, et al: Antibody levels achieved in infants by course of Haemophilus influenzae type b polysaccharide/diphtheria toxoid conjugate vaccine. *Lancet* 1985; 1:1184–1186.
8. Granoff DM, Boies EG, Munson RS Jr: Immunogenicity of Haemophilus influenzae type b polysaccharide—diphtheria toxoid conjugate vaccine in adults. *J Pediatr* 1984; 105:22–27.
9. Ngo TT: Facile activation of sepharose hydroxyl groups by 2-fluoro-1-methylpyridinium toluene-4-sulfonate. *Biotechnology* 1986; 4:134–137.
10. Sasaki T, Tsukada Y, Hirai H: A new procedure for coupling antibody to paper discs for radioimmunoassay: Application to the determination of alpha-fetoprotein. *J Immunol Methods* 1983; 64:25–29.
11. Briand JP, Muller S, Van Regenmortel MH: Synthetic peptides as antigens. Pitfalls of conjugation methods. *J Immunol Methods* 1985; 78:59–69.
12. Timkovich R: Polymerization side reactions during protein modifications with carbodiimide. *Biochem Biophys Res Commun* 1977; 74:1463–1468.
13. Beuvery EC, Kaaden AVD, Kanhaiv et al: Physiochemical and immunological characterization of meningococcal group A polysaccharide—tetanus toxoid conjugates prepared by two methods. *Vaccines* 1983; 1:31–36.
14. Sutton A, Vann WF, Karpas AB, et al: An avidin-biotin based ELISA for quantitation of antibody to bacterial polysaccharides. *J Immunol Methods* 1985; 82:215–224.
15. Bishop CT, Jennings HJ: Immunology of polysaccharides. *The Polysaccharides* 1982; 1:291–330.
16. Snippe H, van Houte AJ, van Dam JE, et al: Immunogenic properties in mice of hexasaccharide from the capsular polysaccharide of Streptococcus pneumoniae type 3. *Infect Immun* 1983; 40:856–861.
17. Lundblad A, Schroer K, Zopf D: Radioimmunoassay of a glucose-containing tetrasaccharide using a monoclonal antibody. *J Immunol Methods* 1984; 68:217–226.
18. Bundle DR: Antibody to an artificial disaccharide antigen cross-reactive with *Neisseria gonorrhoeae* lipopolysaccharide. *Can J Biochem* 1979; 57:367–371.
19. Richter AW, Eby R: Studies on artificial oligosaccharide-protein antigens: Induction of precipitating antibodies to defined epitopes on natural and synthetic dextrans and mannans. *Mol Immunol* 1985; 22:29–36.
20. Brennan P: New-found glycolipid antigens of mycobacteria. *Microbiology* 1984; 366–375.
21. Anderson P, Pichichero M, Insel R, et al: Capsular antigens noncovalently or covalently associated with protein as vaccines to Haemophilus influenzae type b: Comparison in two-year-old children. *J Infect Dis* 1985; 152:634–636.
22. Anderson P, Pichichero M, Insel R, et al: Immunization of 2-month-old infants with protein-coupled oligosaccharides derived from the capsule of Haemophilus influenzae type b. *J Pediatr* 1985; 107:346–351.
23. Granoff DM, Munson RS: Prospects for prevention of Haemophilus influenzae type b disease by immunization. *J Infect Diseases* 1986; 153:448–461.
24. Marburg S, Jorn D, Tolman RL, et al: Bimolecular chemistry of macromolecules: Synthesis of bacterial polysaccharide conjugates with *N. meningitidis* membrane protein. *J Amer Chem Soc* 1986; 108:5282–5287.
25. Merck and Company, Inc: European Patent Application, 1986; 161–188-A.
26. Canadian Patents and Developments Ltd: US Patent, 1982, 4356–170.
27. Anderson PW, Pichichero ME, Insel RA, et al: Vaccines consisting of periodate-cleaved oligosaccharides from the capsule of *Haemophilus influenzae* type b coupled to a protein carrier: Structural and temporal requirements for priming in the human infant. *J Immunol* 1986; 137:1181–1186.
28. Anderson P, Pichichero M, Lawrence E, et al: Vigorous antibody responses in early infancy to Dpo20. A conjugate vaccine of diphtheria toxoid and periodate-cleaved oligosaccharides from the *Haemophilus influenzae* b capsular polymer PRP. *Pediatr Res* 1986; 20:304A.
29. Murphy SG: Tetanus toxin and antigenic derivates. II. Effect of protein and formaldehyde concentration on toxoid formation. *J Bact* 1967; 94:586–589.
30. Rappuoli R, Ratti G, Perugini M, et al: Production of large quantities of diphtheria toxoid CRM 45. *Biotechnology* 1985; 3:161–163.
31. Linggood FV, Stevens F, Fulthorpe AJ, et al: The toxoiding of purified diphtheria toxin. *Br J Exp Path* 1963; 44:177–188.
32. Jennings HJ: Polysaccharides and conjugated polysaccharides as human vaccines, in *New Developments of Industrial Polysaccharides*. New York, Gordon and Breach Science Publishers, 1984, pp 325–344.
33. Jennings HJ, Lugowski C: Immunochemistry of groups A, B, and C meningococcal polysaccharide-tetanus toxoid conjugates. *J Immunol* 1981; 127:1011–1018.

See also pp 195–196.

Regents Physician Loan Forgiveness Program

The New York State Education Department has announced the 1987 Regents Physician Loan Forgiveness Program. This program is designed to attract direct patient care physicians to selected areas of New York State. This year awards will be made to 50 physicians who are practicing in or who agree to practice in an area of New York State critically short of physicians.

Legal residents of New York State who have graduated from an approved medical school and who have completed their residency training in medicine within the last five years are eligible to apply for the award.

The application deadline is May 8, 1987. Award recipients will be selected in June for the 50 awards and may receive up to $10,000 a year for four years. The award payment is based on undergraduate and medical school student loan and loan interest expenses. Further information may be obtained from the Bureau of Higher and Professional Educational Testing, Albany, NY 12230. Tel: 518-474-6394.

Lithium poisoning

Paolo Decina, MD; Anne M. Schlegel, RN; Ronald R. Fieve, MD

Lithium is the drug of choice for the treatment and prevention of manic episodes of manic-depressive illness (ie, bipolar disorder*).[1] The drug is also indicated as a maintenance therapy in recurrent episodes of depression without mania (ie, unipolar disorder*).[1] Lithium has a narrow therapeutic index, and poisoning is the most serious, albeit infrequent, complication of therapy, with an estimated mortality rate of 15% and permanent neurologic and/or renal morbidity in more than 10% of survivors.[2] Approximately 150 to 200 cases of lithium poisoning have been reported in the literature.[3]

Lithium poisoning is usually unintentional, and acute overdose with suicidal intent may account for less than 15% of reported cases.[4] Excessive lithium dosage prescription is probably the most common single cause of poisoning, which develops shortly after beginning treatment or following increases in daily dosage during maintenance therapy. Lithium poisoning may also occur at previously innocuous, stable dosage levels when clinical situations that predispose a patient to lithium retention—such as renal diseases and conditions characterized by sodium and water deficiency—develop.[4] We present two cases of lithium poisoning that occurred while the patients were in the hospital for treatment of medical illnesses.

Case Reports

Case 1. A 67-year-old woman suffering from recurrent unipolar disorder that had been stabilized for seven years on 1,050 mg of lithium carbonate daily, with serum lithium levels of about 0.6 mEq/L, was admitted to the hospital because of pneumonia. Lithium therapy had been discontinued shortly before she entered the hospital.

On admission, the patient was febrile, and antibiotic therapy with ampicillin was started. On the fourth day of her hospital stay, the patient's condition had improved, but she was still febrile; lithium therapy was reinstituted. After five days of therapy with 1,050 mg of lithium carbonate without assessment of serum levels, the patient was reported to be "depressed"—sluggish and listless with decrease in appetite—and she suffered two bouts of diarrhea. At that time the patient was afebrile and her lung fields on a chest radiograph had improved. The ampicillin was discontinued as a possible cause of gastrointestinal upset. The serum lithium level was assessed on the following day (sixth day of lithium therapy) and found to be 4.8 mEq/L. Psychiatric consultation at the time revealed an unresponsive and lethargic patient who had had a generalized seizure. Pneumonia subsequently developed, with respiratory failure and renal failure. The patient went into a coma and died. No autopsy was performed.

Case 2. A 56-year-old man suffering from bipolar disorder, who had been on maintenance therapy for 11 years with 2,400 mg of lithium carbonate daily and who had serum lithium levels of about 1 mEq/L was admitted to the hospital for a urinary tract infection. On admission, the serum lithium level was 0.48 mEq/L, and lithium therapy was continued. The patient was placed on ampicillin therapy and, after four days, became restless and agitated. A serum lithium level assessed on the fourth day of his hospital stay was 1.8 mEq/L. The hematocrit and plasma protein concentration were increased over admission values, but were within the normal range. The patient's mental condition was thought to be related to mania, and lithium therapy was continued.

On the sixth day of his hospital stay, a psychiatric consultation was requested. At that time, the patient was febrile, disoriented, and agitated, and the serum lithium level was 3.8 mEq/L. Therapy with forced diuresis was promptly instituted, and the patient responded well to treatment in the intensive care unit. His condition stabilized over a period of ten days. As his condition improved, he was transferred to the medical ward. The patient suddenly died in the hospital six days later of a massive pulmonary embolism. The embolism was confirmed at autopsy and was secondary to phlebothrombosis of the veins of both lower extremities. Other autopsy findings, particularly pertaining to the kidney, were unremarkable.

Discussion

These two cases are illustrative of common situations that predispose a patient to lithium poisoning and of typical manifestations of neurotoxicity with prominent mental symptoms.

On a stable lithium dosage, poisoning is almost invariably preceded by some reduction in renal lithium clearance, as 98% of bodily lithium is eliminated through the kidney. Lithium is reabsorbed competitively with sodium in the proximal tubule. A reduction in the glomerular filtration rate or relative sodium deficiency can cause lithium retention. In virtually all published cases in which poisoning developed while the patient was on a stable lithium dosage, either renal disease or conditions affecting water and electrolyte balance (such as infectious diseases, dehydration, anorexia, low sodium diet, or diuretics) have been implicated as major predisposing factors.[4] The possibility that ampicillin therapy may have further contributed to the pathogenesis of poisoning by nephrotoxic action in the two patients described here cannot be definitely ruled out. There are reports of lithium retention associated with tetracyclines and other antibiotics,[5] although more recent experimental work has failed to confirm the effect of antibiotics, including ampicillin, on lithium renal clearance.[6]

From the New York State Psychiatric Institute, New York, NY.

Address correspondence to Dr Decina, Department of Biological Psychiatry, New York State Psychiatric Institute, 722 W 168th St, New York, NY 10032.

* Bipolar disorder and unipolar disorder are major psychiatric illnesses characterized by recurrent episodes of mania and depression (bipolar disorder), or depression without mania (unipolar disorder). During an episode, the disturbance of mood is marked, often necessitating admission to a psychiatric hospital, and there is always a substantial impairment in psychosocial functioning.

The early stage of lithium poisoning is characterized by neurotoxicity which is often oligosymptomatic.[2] The mental status is characterized by apathy and sluggishness (case 1), as well as by motor agitation (case 2). The patients are distractible and may complain of muscle weakness, unsteady gait, or impaired motor coordination. Muscle tone is increased, and there may be extrapyramidal rigidity and choreoathetoid movements. The fine hand tremors often associated with lithium therapy become coarse and irregular. Cerebellar signs such as ataxia and dysarthria may be present. Diarrhea and nausea with vomiting are occasionally seen. As noted above, the change in a patient's mental status can be incorrectly ascribed to manifestations of psychiatric affective disorder. In case 1, the diarrhea had been attributed to antibiotic therapy

Recognition of the early signs of neurotoxicity followed by appropriate intervention is crucial. Both the severity and outcome of poisoning appear to be associated with prolonged exposure to neurotoxic levels. Moreover, occasional cases of lithium poisoning have been reported with serum lithium levels within the therapeutic range—up to 1.5 mEq/L—precluding blind diagnostic reliance on laboratory findings.[7]

Treatment of lithium poisoning begins with the immediate discontinuation of the drug and centers around measures directed at quickly removing lithium from the body, either by dialysis or forced diuresis with furosemide and isotonic solutions.[2] Prevention of lithium poisoning rests on knowledge of the conditions leading to lithium retention and the recognition of initial neurotoxic symptoms, as well as regular monitoring of serum lithium levels and glomerular filtration rates at appropriate intervals. Since it cannot be assumed that all medical practitioners are thoroughly familiar with lithium pharmacokinetics and the mental symptoms produced by excessive levels of lithium, psychiatric consultation is in order before lithium treatment is continued or reinstituted in medical patients or other treatment procedures are initiated.

References

1. Task Force on Lithium Therapy: The current status of lithium therapy: Report of the APA Task Force. *Am J Psychiatry* 1975; 132:997-1001.

2. Schou M: The recognition and management of lithium intoxication, in Johnson FN (ed): *Handbook of Lithium Therapy*. Lancaster, England, MTP Press, 1980, pp 394-402.

3. Vestergaard P: Long-term risks, complications, and drug interactions: Lithium. Abstract in *Mood Disorders: Pharmacologic Prevention of Recurrences*. Consensus Development Conference, Washington, DC, April 24-26, 1984.

4. Hansen HE, Amdisen A. Lithium intoxication (Report of 23 cases and review of 100 cases from the literature). *Q J Med* 1978; 47:123-144.

5. Mann J, Gershon S: Absolute and relative contraindications to lithium treatment, in Johnson FN (ed): *Handbook of Lithium Therapy*. Lancaster, England, MTP Press, 1980, pp 265-278.

6. Lassen E: Effects of acute and short-time antibiotic treatment on renal lithium elimination and serum lithium levels in the rat. *Acta Pharmacol Toxicol* 1985; 56:273-277.

7. Lewis DA: Unrecognized chronic lithium neurotoxic reactions. *JAMA* 1983; 250:2029-2030.

Isolated cardiac metastatic recurrence of epithelioid sarcoma after two and a half disease-free years

LAUREL J. STEINHERZ, MD; GERALD ROSEN, MD; PETER G. STEINHERZ, MD;
JUNE ROBINS, RDMS; ANDREW HUVOS, MD; PHILIP R. EXELBY, MD

Cardiac involvement with metastatic malignancy has become more common with longer patient survival[1-3] and with the availablility of sophisticated, noninvasive diagnostic techniques.[4,5] Cardiac involvement is usually associated with widespread metastatic disease.[2] We report an unusual case of extensive, isolated myocardial involvement by recurrent metastatic tendosynovial sarcoma in an 18-year-old boy, two and a half years after the conclusion of chemotherapy.

Case Report

In January 1979, the patient was found to have an epithelioid sarcoma involving the left shoulder, with axillary and cervical node involvement. Forequarter amputation and radical neck dissection were performed. An adjuvant, multidrug chemotherapeutic regimen, administered after surgery, including vincristine, methotrexate, cyclophosphamide, bleomycin, dactinomycin, and doxorubicin (T9) was completed in March 1980.[6] Serial echocardiograms during therapy remained normal, and surgery in December 1980 revealed no evidence of residual disease. The patient remained asymptomatic through two more years of follow-up.

In August 1982, the patient complained of an aching sensation in the left precordium and at the amputation site. He sought treatment at a local hospital after an acute, short bout of tachycardia followed by precordial pain and a heavy sensation in his chest. T-wave inversion was seen in the precordial leads. He was thought to have viral pericarditis and was given aspirin, with improvement of symptoms.

The patient then returned to Memorial Sloan-Kettering Cancer Center for consultation. He was not in distress. A physical examination revealed no abnormalities except for the amputation and some diminution of intensity of the heart tones. There was no evidence of tamponade. An electrocardiogram showed inverted T waves, and a chest film revealed a mildly enlarged globular cardiac silhouette. A moderate amount of pericardial effusion and borderline myocardial function was seen on the M-mode echocardiogram. A diagnostic pericardiocentesis yielded serosanguineous fluid with clumps of cells with dark nucleoli and scant cytoplasm. No definitive evidence of a malignant process could be made on cytomorphologic evaluation. The cultures were negative.

Two weeks later, signs of tamponade developed. During an emergency pericardiocentesis, 350 cc of bloody fluid was removed showing sheets of cells. A pericardial window and biopsy were performed the next day. On that day, the chest radiograph showed pleural fluid for the first time. At

---

From Memorial Sloan-Kettering Cancer Center, New York, NY.

Address correspondence to Dr Laurel Steinherz, Memorial Sloan-Kettering Cancer Center, 1275 York Ave, New York, NY 10021.

thoracotomy, the pericardium was found to be grossly thickened due to infiltration with recurrent tendosynovial sarcoma. The epicardium was invaded by tumor nodules to such an extent that all normal landmarks were distorted. There was minimal involvement of the pleura, with pinpoint specks of tumor and a small pleural effusion.

The patient's recovery was complicated by continued drainage of massive amounts of serosanguineous fluid. Signs of cardiac failure developed, but the patient responded to digoxin. Chemotherapy with weekly doses of intravenous methotrexate, 300–600 mg/M^2, was given. Drainage from the chest tube diminished and eventually ceased, and the patient was discharged. At the time of discharge, the lung fields were clear on physical examination and on chest film. There was resolution of pericardial fluid and thickening on an echocardiogram, and there were no signs of cardiac failure.

The patient continued to receive chemotherapy as an outpatient for five months, with no clinical or histologic signs of disease progression. A recurrent episode of precordial pain and ventricular tachycardia necessitated another hospital admission. During the next three days clinical congestive heart failure developed. An echocardiogram revealed doubling of apical septal thickness when compared to the previous month's evaluation, with partial obliteration of the right ventricular cavity (Fig 1). The patient's dysrhythmia was controlled with procainamide and diphenylhydantoin, and the congestive heart failure was treated with diuretics and vasodilators. The tumor did not respond to emergency radiation therapy to the heart, and the patient ultimately succumbed to the cardiac compromise.

A postmortem examination revealed massive enlargement of the cardiac walls, which were invaded and replaced by tumor (Fig 2). The tumor also involved the left lung and pleura, the left thoracotomy incision scar, the diaphragm, liver, and abdominal lymph nodes. There was also tumor wrapped around the left carotid artery.

On microscopic examination, the tumor tissue was characterized by medium sized ovoid or polygonal cells with eosinophilic cytoplasm and ill-defined cellular borders (Fig 3). The cellular pleomorphism was not pronounced, and multinucleated giant tumor cells were absent. The spindle cell component of the tumor was minimal but, instead, a prominent clear cell change of the tumor was noted. These two components, the epithelioid and the clear cell aspects, merged, but the epithelioid appearance of the tumor predominated. Special techniques were employed to highlight the favored histologic diagnosis of epithelioid sarcoma, but these were of limited help. The histochemical studies included Grimelius stain for neurosecretary granules, periodic acid-Schiff with and without diastase digestion, and melanin stains.

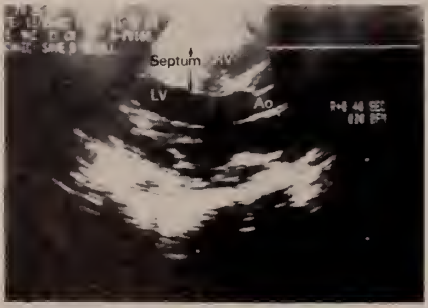


FIGURE 1. Two-dimensional echocardiogram at the time of readmission for ventricular tachycardia. Massive apical septal thickening (arrow) with obliteration of the right ventricular cavity is demonstrated. Ao, aorta; LV, left ventricle; RV, right ventricle; septum, interventricular septum. Arrow indicates the extent of septal thickening.

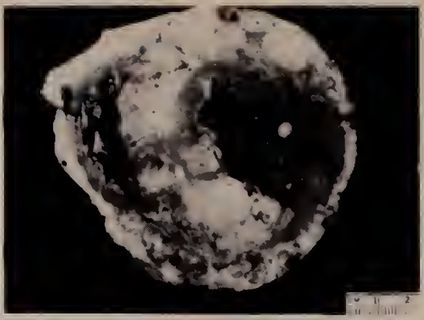


FIGURE 2. Postmortem gross cross-section through the ventricles. Extensive tumor invasion (white areas) produces thickening of the cardiac walls, most prominently of the interventricular septum.

Microscopic examination of the primary sarcoma in the left scapular region, the lymph node metastases in the left axilla and left posterior triangle (all in 1979), as well as the pericardial metastasis in September 1982 revealed identical morphologic features.

DISCUSSION

The heart is the site of metastatic spread in 2–20% of malignancies.[1-3,7] Cardiac involvement is usually seen in the setting of widespread metastases. Invasion of the heart occurs most often via direct extension from intrathoracic tumors, but may also occur via lymphatic drainage, embolization through blood vessels, or direct endocardial implantation.[2] The metastatic process involves the pericardium and epicardium most frequently. Invasion of the myocardium is less common, and invasion of the endocardial surfaces and intracavity spaces is even less frequent. Cardiac metastases can produce symptoms such as pericardial tamponade and constriction, dysrhythmia, altered myocardial function, and obstruction of cavities and valves.[1-3] Helpful noninvasive diagnostic techniques include echocardiography,[1,4] computed tomography,[4] and gallium scan.[1,5] The latter technique is still under evaluation. Cytologic analysis of the pericardial fluid may be misleading, although it can be helpful in some cases.

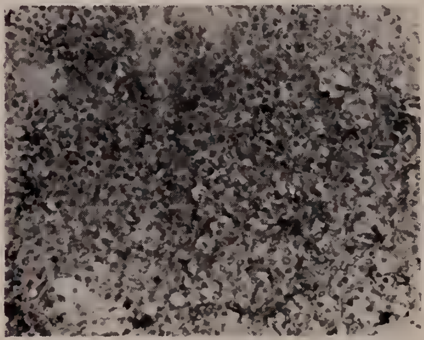


FIGURE 3. Sheet-like arrangement of epithelioid cells with clear cytoplasm. There is no obvious pattern formation in the epithelioid sarcoma of soft tissues of the left scapular region (hematoxylin-eosin stain, original magnification × 260).

The patient described here is unusual, not only because the heart was the first site of metastasis, but also because the metastasis occurred two and a half years after discontinuation of therapy and almost three years after the disappearance of any sign of malignancy. The only other two cases of cardiac metastases of epithelioid sarcoma described in the literature followed multiple recurrences and widespread metastatic involvement of other organs.[8] At the time of presentation, this patient had no detectable evidence of other metastases. Minimal pleural involvement was apparent only after some weeks of cardiac symptoms and two pericardiocenteses. The mode of spread was probably through lymphatic drainage. The initial signs and symptoms of isolated cardiac recurrence could easily have been passed off as viral pericarditis. However, a high index of suspicion—because of the previous malignant disease and an awareness of the possibility of cardiac involvement with prolonged survival—prompted an aggressive workup that led to the correct diagnosis. It is noteworthy that despite the relatively resistant nature of the tumor and the extensive spread throughout the myocardium, there was temporary improvement with systemic chemotherapy.

Acknowledgment. The authors thank Ms Lee Roepke for her secretarial assistance.

REFERENCES

1. Stark RM, Perloff JK, Glick HJ, et al: Clinical recognition and management of cardiac metastatic

disease: Observations in a unique case of alveolar soft-part sarcoma. *Am J Med* 1977; 63:653-659.

2. McLoughlin MJ: Obstruction of the right ventricular outflow tract due to metastasis. *Brit J Radiol* 1970; 43:573-576.
3. Mann T, Brodie BR, Grossman W, et al: Effusive-constrictive hemodynamic pattern due to neoplastic involvement of the pericardium. *Am J Cardiol* 1978; 41:781-786.
4. Das L, Farooki ZQ, Hakimi MN, et al: Asymptomatic intracardiac metastasis from osteosarcoma: A case report with literature review. *Med Pediatr Oncol* 1983; 11:164-166.
5. Yeh SD, Benua RS: Gallium-67-citrate accumulation in the heart with tumor involvement. *Clin Nuc Med* 1978; 3:103-105.
6. Rosen G, Caparros B, Nirenberg A, et al: Ewing's sarcoma: Ten-year experience with adjuvant chemotherapy. *Cancer* 1981; 47:2204-2213.
7. Chan HS, Sonley MJ, Moës CA, et al: Primary and secondary tumors of childhood involving the heart, pericardium, and great vessels. A report of 75 cases and review of the literature. *Cancer* 1985; 56:825-836.
8. Prat J, Woodruff J, Marcove R: Epithelioid sarcoma: An analysis of 22 cases indicating the prognostic significance of vascular invasion and regional lymph node metastasis. *Cancer* 1978; 41:1472-1487.

See also pp 197-198.

The use of computed tomographic scanning to diagnose sterile gluteal abscesses

RIFFAT R. CHAUDARY, MD, KUMUDHA RAMANATHAN, MD

Computed tomographic (CT) scanning has been used for the evaluation of soft tissue masses.[1,2] We present a case with a striking CT pattern of fat/fluid levels in bilateral gluteal masses.

CASE REPORT

An eight-year-old boy was seen in the clinic for increasing swelling and pain in the left buttock. There was no definite history of trauma, fever, chills, or sweats. On physical examination, the left buttock had a firm mobile mass with skin fixation; the mass was slightly tender. The right buttock was slightly enlarged without any focal mass.

The patient was referred for radiologic evaluation. Plain films (Fig 1) demonstrated soft tissue, subcutaneous masses with rim-like calcifications.

CT scan (Figs 2, 3) showed lobulated, rounded cystic masses in the gluteal regions bilaterally, surrounded by subcutaneous fat. The larger lobulations had calcific rims and a fluid/fluid level. The anterior layer of fluid measured −49 Hounsfield units (Hu), whereas the posterior layer had an attenuation of +5 Hu. These measurements were consistent with fat and fluid densities, respectively, creating a fat/fluid level.

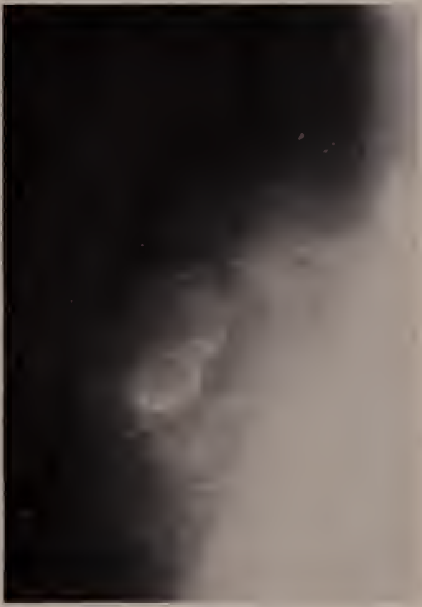


FIGURE 1. Oblique radiograph of the right buttock using soft tissue technique.

This important observation led to further investigation of the patient's clinical history. It was learned that the patient had diabetes insipidus (probably secondary to meningitis contracted as an infant) and had been treated with vasopressin tannate injections in the buttocks until two years prior to this examination. Vasopressin tannate in oil is a water-insoluble compound of vasopressin in suspension with peanut oil. A diagnosis of sterile gluteal abscesses was made based on this information.

DISCUSSION

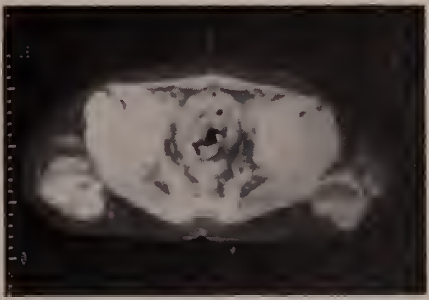


FIGURE 2. Consecutive CT scans through the midpelvis showing bilateral loculated, rounded cystic masses in the gluteal regions.

The value of CT scanning in localizing soft tissue masses has been well documented.[1,2] The unique capability of computed tomography to distinguish attenuation values helped in making the correct diagnosis in this patient. Without the knowledge of fatty density in the gluteal masses, the differential diagnosis would have included hematoma, abscesses, parasitic cysts, and necrotic tumors. The low attenuation value of the anterior fluid component in the gluteal masses was most likely due to the peanut oil present in the vasopressin tannate injections. Peanut oil (arachis) has been shown to have an attenuation value of −100 Hounsfield

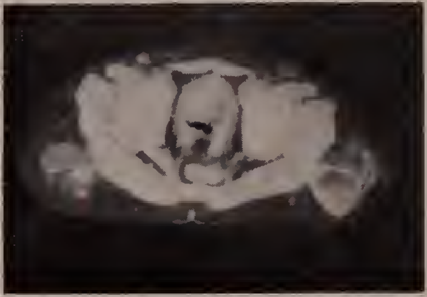


FIGURE 3. CT scan through the midpelvis (same image as Fig 2) depicting attenuation values in H units.

From the Department of Radiology, The Long Island College Hospital, 340 Henry St, Brooklyn, NY.

Address correspondence to Dr Chaudary, Chief, Division of CT Scanning, Department of Radiology, The Long Island College Hospital, 340 Henry St, Brooklyn, NY 11201.

units during its use as an intravesical contrast medium for computed tomography.[3]

In 1985, Munk and Muller[4] reported the case of a patient with findings similar to those described here. In that case, small cystic and calcified areas were seen in both thighs where the patient had received vasopressin injections. A single area of fat density was described, which raised the possibility of oil-induced granulomas. At surgery, oily material was evacuated; pathologic analysis demonstrated granulation tissue with fat vacuoles.[4] The plain film findings of eggshell calcifications in that patient are similar to those described here. However, the patient presented in this report had much larger areas of involvement, with definite fat/fluid levels. He did not undergo surgery and is being treated with desmopressin acetate, a synthetic analogue of vasopressin which is administered intranasally.

REFERENCES

1. Murphy WA, Gilula LA, Destouret JM, et al: Musculoskeletal system, in Lee JKT, Sagal SS, Stanley RJ (eds): *Computed Body Tomography*. New York, Raven Press, 1983, pp 453–515.
2. Egund N, Ekelund L, Sako M, et al: CT of soft-tissue tumors. *AJR* 1981; 137:725–729.
3. Hildell JG, Nyman VR, Norlindh ST, et al: New intravesical contrast medium for CT: Preliminary studies with arachis (peanut) oil. *AJR* 1981; 137:777–780.
4. Munk PL, Muller NL: CT diagnosis of oil-induced granulomas secondary to diabetes insipidus therapy. *J Comput Assist Tomogr* 1985; 9:1147–1148.

FROM THE LIBRARY

CARDIOVASCULAR ABNORMALITIES DURING SURGERY SECONDARY TO METASTATIC LESIONS OF THE HEART

Metastatic disease of the heart and pericardium is not a rare occurrence. The incidence varies in different series from 1.8 to 13.9 per cent of all autopsies performed on patients with malignant disease, but the condition has been diagnosed prior to death in only a comparatively few cases. After reviewing the literature, Prichard reported that by 1950 only 21 cases of secondary malignant disease of the heart had been discovered before death. In 1953, DeLoach and Haynes noted 1 case in which the diagnosis was definitely established antemortem and 2 additional cases of suspected cardiac metastatic disease which were subsequently confirmed by autopsy. Although Bisel, Wroblewski, and LaDue did not mention a specific number of cases of metastatic cardiac disease diagnosed prior to autopsy, they reported that 8.5 per cent of patients with autopsy findings of metastatic cardiac disease exhibited symptoms and physical signs of heart disease prior to death, and that there were electrocardiographic records suggestive of cardiac involvement in 28.8 per cent of the patients.

The pathologic conditions, the symptoms, and the physical and laboratory findings in patients with metastatic cardiac lesions have been reviewed by several investigators. . . . Their studies showed that although metastatic lesions to the heart and pericardium may arise from any organ, the most frequent sites of origin are the breast and the lung with metastatic melanoma next in order of frequency. The tumor cells reach the heart by a lymphatic or blood vessel spread or by direct extension from an adjacent organ. Metastatic lesions are found with equal frequency in the epicardium, the myocardium, and the endocardium and may occupy an entire chamber of the heart, extending at times into other chambers. Pericardial involvement by tumor is manifested by a diffuse thickening or obliteration of the structure or by the presence of an effusion, either clear or hemorrhagic in nature. Diffuse metastatic disease is always present in patients with cardiac involvement.

From the standpoint of antemortem diagnosis, there are no specific signs or symptoms which are characteristic of metastatic disease of the heart or pericardium. Many patients exhibit no evidence of cardiac difficulty prior to death. In other cases cardiac metastases have resulted in a variety of symptom complexes including sudden death; unexplained congestive failure; subacute bacterial endocarditis; or abnormalities of rhythm such as auricular fibrillation, various degrees of heart block, extrasystoles, or paroxysmal tachycardia. Among the electrocardiographic changes listed by Bisel, Wroblewski, and LaDue as suggestive of cardiac metastatic disease were T-wave abnormalities, low voltage of the QRS complexes, S-T segment deviations, Q waves, prolonged auriculoventricular conduction, and arrhythmias. Roentgenologic evidence of the enlargement of the heart or the fixation of the right border of the heart may assist further in the diagnosis. . . .

OLGA SCHWEIZER, MD
WILLIAM S. HOWLAND, MD
(*NY State J Med* 1961; 61:955–959)

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication. All letters are personally acknowledged by the Editor.*

Gastric antacid bezoar in a patient with chronic renal failure

TO THE EDITOR: The common types of gastrointestinal bezoars include trichobezoar, phytobezoar, and lactobezoar. Medication bezoars are rare, and antacid bezoars causing intestinal obstruction are a known, but uncommon, entity. Reported cases in medical and pediatric literature have involved infants and patients with renal failure. In patients with renal failure, aluminum hydroxide suspension has been the implied culprit. We detected an aluminum-containing gastric bezoar in a young, ambulatory, adult patient with chronic renal failure. The diagnosis of a bezoar in this patient was not apparent due to the lack of symptoms.

Case Report. A 25-year-old man, who had been on hemodialysis for about five years, underwent an upper gastrointestinal radiographic series in preparation for a kidney transplant. At that time, he had been taking aluminum carbonate gel capsules for about two years. The patient was taking eight capsules of the gel three times a day after meals. Each capsule was equivalent to 500 mg of aluminum hydroxide. The patient had complained of increasing flatulence and belching for a period of several months without any definite symptoms of gastrointestinal obstruction. His serum blood urea nitrogen and creatinine levels two days prior to examination were 93 mg/dL and 23.8 mg/dL, respectively. The scout radiograph of the abdomen showed a large radiodensity outlined by air in the region of the fundus of the stomach (Fig 1). A double contrast upper gastrointestinal examination demonstrated movable filling defects in the stomach consistent with a bezoar (Figs 2, 3).

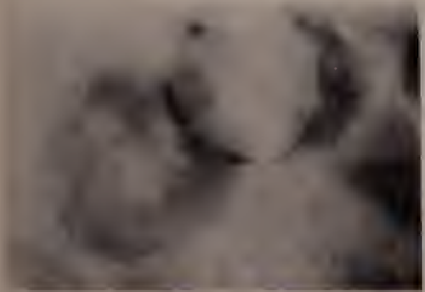


FIGURE 1. Supine scout view of abdomen showing left upper quadrant radiodensity outlined by air in the region of the gastric fundus.

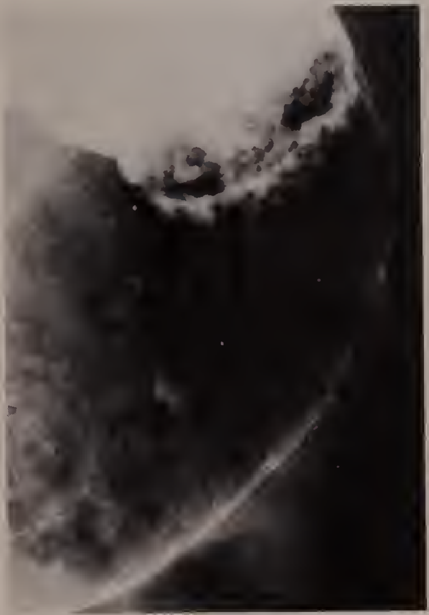


FIGURE 2. Spot film from a double contrast upper gastrointestinal series showing a filling defect in the gastric fundus.

There was no evidence of gastric outlet obstruction. Endoscopy of the esophagus and stomach revealed a bezoar in the gastric fundus. A gastric aspirate obtained at the time of endoscopy showed an aluminum content of 255 ng/ml. The bezoar was broken up and partially removed. A second upper gastrointestinal series obtained four months later showed no evidence of a bezoar. The patient was put on an aluminum carbonate gel suspension, and the capsules were discontinued.

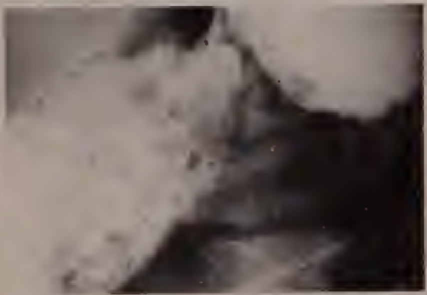


FIGURE 3. Spot film from a double contrast upper gastrointestinal series showing the filling defect, which had moved into the gastric body and antral regions.

Discussion. Basic aluminum compounds are given to patients with renal failure as antacids and to bind phosphate. Antacid bezoar is a rare complication of antacid therapy—the exact rate of occurrence is unknown. In one study involving 60 patients with acute renal failure following trauma or surgery, a 5% complication rate was found.[1] The complications were mainly large or small bowel obstruction. The same authors did not find any incidence of complications arising from antacid therapy for patients on home dialysis or preparing for renal transplantation. Other complications can result from antacid bezoars. Korenman et al[2] reported a case involving an ambulatory patient who had bowel perforation associated with an antacid bezoar.

In adult patients, antacid bezoars have mainly been found in the distal small bowel or in the colon. Rosenberg[3] and Portuguez-Malavasi and Aranda[4] reported two cases of antacid bezoars in newborns. In both cases, the bezoars were found in the stomach, and they resolved with conservative treatment including discontinuation of the antacid and saline or water irrigation. Decreased bowel motility, dehydration, and debilitation are believed to increase the risk of antacid bezoar formation. The patient reported here is illustrative of an antacid bezoar in the stomach—an uncommon site of occurrence in adults. The patient was not physically debilitated and had only nonspecific symptoms. Gastric bezoars may be relatively asymptomatic, and their discovery, therefore, can be delayed until obstruction occurs in the bowel more distally.

Slatopolsky and Weerto[5] studied the efficacy of calcium carbonate as an alternative to aluminum compounds for phosphate binding. They found that six patients had a reduction of their serum aluminum levels from about 53 μg/L to 26 μg/L after discontinuation of aluminum phosphate binders. The patient described here had a gastric aluminum level of 255 ng/mL. The relationship between gastric fluid and serum aluminum levels is unknown. The treatment regimen for this patient was altered after the bezoar was discovered. The possibility of bezoars in patients with renal failure should be considered irrespective of the patient's physical condition and the severity of symptoms. Under the proper clinical settings, the diagnosis may be suggested on plain films. An upper gastrointestinal radiographic series can be performed to confirm the diagnosis.

PAUL MOH, MD
BURTON GOLD, MD
Department of Radiology
Nassau County Medical Center
East Meadow, NY 11554

---

1. Townsend CM Jr, Remmers AR Jr, Sarles HE, et al: Intestinal obstruction from medication bezoar in patients with renal failure. *N Engl J Med* 1973; 288:1058-1059.
2. Korenman MD, Stubbs MB, Fish JC: Intestinal obstruction from medication bezoar. *JAMA* 1978; 240:54-55.
3. Rosenberg HK: Antacid bezoar in a premature infant. *Clin Pediatr* 1982; 21:503-504.
4. Portuguez-Malavasi A, Aranda JV: Antacid bezoar in newborn. *Pediatrics* 1979; 63:679-680.
5. Slatopolsky E, Weerto C, Lopez-Hilker S, et al: Calcium carbonate as a phosphate binder in patients with chronic renal failure undergoing dialysis. *N Engl J Med* 1986; 315:157-161.

Central phlebothrombosis with two venous irritants

TO THE EDITOR: Semipermanent, surgically placed central venous and right atrial catheters have gained widespread use for the infusion of chemotherapeutic agents and parenteral nutrition.[1] The large blood flow around the catheters' internal orifices dilutes irritant medications, thus making the problems of associated chemical phlebitis and thrombosis rare events. The following case illustrates extensive but asymptomatic superior vena caval and right atrial thrombosis associated with a Hickman catheter, through which two known phlebitogenic drugs were delivered.

Case Report. A 26-year-old woman was found to have acute promyelocytic leukemia in April 1984, 18 months after isolated radiotherapy for Hodgkin's disease. During an 11-month period, she received daunomycin, cytarabine, and 6-thioguanine. In May 1984, histologically proven hepatosplenic candidiasis developed. The patient was treated by splenectomy, amphotericin B (40 milligrams by vein daily) and 5-fluorocytosine. Amphotericin B was administered through a Hickman catheter for several months after discharge. The candidiasis was suppressed. However, multiple leukemic relapses led to further cytarabine, 6-thioguanine, amsacrine (a daunomycin derivative), and intrathecal cytarabine therapy.

Intercurrent complications included two cerebral infarctions which were ascribed to extreme leukocytosis with "sludging," disseminated intravascular coagulation (DIC), and transverse myelopathy which was ascribed to intrathecal cytarabine. There were no clinically apparent pulmonary emboli, episodes of low blood flow, problems with the Hickman catheter, or features of right heart dysfunction. The patient died 12 months after diagnosis, with refractory leukemia, cachexia, *Staphylococcus aureus* septicemia, and leukoencephalopathy.

An autopsy revealed a 9-cm yellow-white endothelial vegetation adjoining the Hickman catheter, and extending from the superior vena cava across the right atrium to the tricuspid valve and its chordae tendineae. This growth consisted of bland fibrin and collagen without inflammatory cells, fungi, or bacteria. Multiple histologic sections of the thrombotic vegetation showed some features of granulation tissue and organized thrombus, along with fibroelastosis and smooth muscle loss in the subjacent superior vena cava, and scarring in the nearby atrium. Considerable crystalline matter, including calcium, was seen in the vegetation, but no inflammation or fungus was detectable, even with special stains. Marked inflammation and fungal elements persisted in residual hepatic and renal candidal abscesses. The autopsy also showed anasarca, fibrinous pericarditis, marantic mitral valvulitis, bacillary respiratory and enteric infection, and a nonspecific myelopathy.

Discussion. The initial presumption at autopsy, that this thrombus represented candidal endocarditis, was disproven. DIC produces thrombi, but the high flow in the largest veins should spare them, even when foreign bodies are present. We concluded that the thrombus began as a fibrin "sleeve thrombus," an entity commonly found around Hickman catheters.[2-4] Thrombosis was augmented and enlarged by chemical phlebitis associated with the instillation of two known venous endothelial irritants—amphotericin B and cytarabine.[5] We postulate that a small nidus of inflammation, which was all that this patient's cytopenic blood could muster, developed near the catheter, and that the thrombus propagated into the heart. This focal venous inflammation may have receded between the time of phlebitis and death or may have persisted but escaped detection. Thrombus propagation, terminal volume expansion, and hypoalbuminemia likely contributed to anasarca, even without obstruction to right heart inflow. The lack of clinical findings associated with this large thrombus underscores the fact that veins produce signs and symptoms only when substantially inflamed, distended, or obstructed.

Other infusions, such as lipid-rich parenteral nutrition solutions, have been associated with the formation of thrombi around Hickman catheters, but the agents involved in this case have not previously been implicated. This case of central phlebothrombosis and endocardial thrombosis resulted from the administration of two irritant medications, amphotericin B and cytarabine, through a Hickman catheter positioned in the right atrium.

HENRY SCHNEIDERMAN, MD
RICHARD A. GARIBALDI, MD
PAUL F. DAVERN, MBA, RPH
University of Connecticut Health Center
Farmington, CT 06032

---

1. Wagman LD, Kirkemo A, Johnston MR: Venous access: A prospective, randomized study of the Hickman catheter. *Surgery* 1984; 95:303-308.
2. Brismar B, Harstedt C, Jacobson S: Diagnosis of thrombosis by catheter phlebography after prolonged central venous catheterization. *Ann Surg* 1981; 194:779-783.
3. Jacobs MB, Yeager M: Thrombotic and infectious complications of Hickman-Broviac catheters. *Arch Intern Med* 1984; 144:1597-1599.
4. Pessa ME, Howard RJ: Complications of Hickman-Broviac catheters. *Surg Gynecol Obstet* 1985; 161:257-260.
5. American Hospital Formulary Service: *Drug Information '85*. Bethesda, Md, American Society of Hospital Pharmacists, 1985.

Extensive calcified pleural plaques

TO THE EDITOR: The association between pleural plaques and asbestos exposure has been amply confirmed by clinical and epidemiologic studies.[1] The prevalence of pleural changes in the chest radiograph increases in relation to estimated exposure to asbestos.[1-3] Pleural plaques occur as discrete, raised, gray-white lesions and can involve any portion of the pleura but usually spare the apices. It has been claimed that symmetrical lower zone pleural or diaphragmatic plaques are markers of asbestos exposure.[2] Microscopically, pleural plaques consist of collagenous connective tissue with calcium deposition.

The pathogenesis of pleural plaques is not well established. One hypothesis is that pleural plaques result from trauma to the parietal pleura during breathing, as inhaled sharp asbestos spicules penetrate the pleura.[1] Calcified plaques are usually seen on the posteroanterior film as irregular outlines of uneven density. Oblique films are necessary to visualize plaques that are located anteriorly or posteriorly. Hyaline plaques

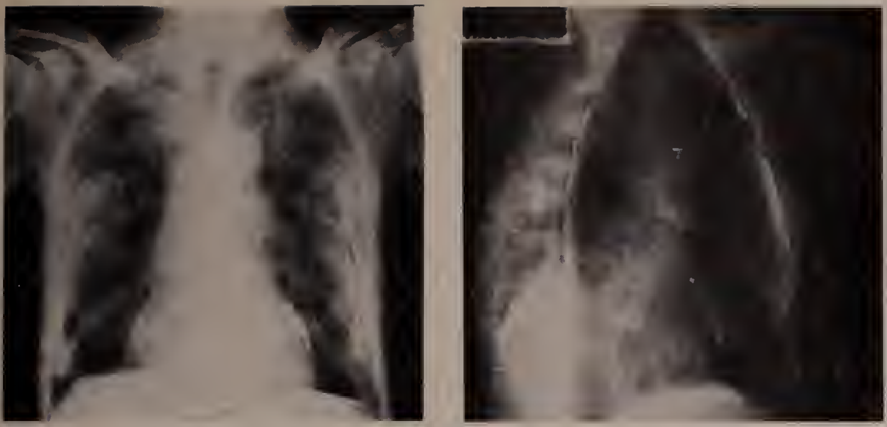


FIGURE 1. Antero-posterior roentgenograph (left) and oblique roentgenograph (right) of the right hemithorax showing extensive calcified pleural plaques on the chest wall, diaphragmatic, and mediastinal pleural surfaces.

may not be seen on a routine chest film.[1]

Case Report. An 85-year-old man with atherosclerotic heart disease, cerebrovascular disease, and organic mental syndrome was admitted for mild shortness of breath, general weakness, fever, and increased frequency of urination. He was treated for a urinary tract infection and did well. The patient was a chronic smoker who had worked for over two decades as a mechanic in the Brooklyn Navy Yard. Arterial blood gas studies on room air showed pH, 7.48; P_{CO_2}, 28 mm Hg; P_{O_2}, 66 mm Hg; with an alveolar-arterial O_2 gradient of 49 mm Hg. A chest film showed a normal heart and large lung fields. There were extensive but even pleural calcifications along the mediastinal, diaphragmatic, and chest wall surfaces (Fig 1). The patient was unable to perform pulmonary function tests. The diagnosis was extensive calcified asbestos pleural plaques.

Discussion. Pleural plaques in the absence of obliterative pleural lesions or pulmonary fibrosis are infrequently associated with pulmonary symptoms. Clinically they may cause mild dyspnea, and pulmonary function testing may show a restrictive ventilatory defect. Rarely, they result in severe ventilatory impairment and ventilatory failure.[4] The vital capacity, total lung capacity, and maximal voluntary ventilation may all be reduced, but if there is little parenchymal disease, the diffusing capacity will be relatively unaffected. Asbestos workers with pleural changes are more likely to develop progressive parenchymal fibrosis[5] and may have an increased risk of developing mesothelioma.[6,7]

The case presented is unusual in that the patient had extensive asbestos-caused pleural disease that only recently produced respiratory symptoms. However, this does not appear to have affected the patient's longevity.

Acknowledgment. The authors thank Mrs Norma Johnson for her assistance in preparing this manuscript.

SUBRAHMANYA BALLAMBATTU, MD
STEVEN POWELL, MD
DAWN PETERS, BA
Department of Medicine
Woodhull Medical and Mental Health Center
760 Broadway
Brooklyn, NY 11206

1. Becklake MR: Asbestos-related diseases of the lung and other organs: Their epidemiology and implications for clinical practice. *Am Rev Respir Dis* 1976; 114:187-227.
2. Churg A: Asbestos fibers and pleural plaques in a general autopsy population. *Am J Pathol* 1982; 109:88-96.
3. Selikoff IJ, Lilis R, Nicholson WJ: Asbestos disease in United States shipyards. *Ann NY Acad Sci* 1979; 330:295-311.
4. Miller A, Teirtein AS, Selikoff IJ: Ventilatory failure due to asbestos pleurisy. *Am J Med* 1983; 75:911-919.
5. Sheers G: Asbestos-associated disease in employees of Devonport Dockyard. *Ann NY Acad Sci* 1979; 330:281-287.
6. Warnock ML, Isenberg W: Asbestos burden and pathology of lung cancer. *Chest* 1986; 89:20-26.
7. Edge JR: Incidence of bronchial carcinoma in shipyard workers with pleural plaques. *Ann NY Acad Sci* 1979; 330:289-294.

Acyclovir in the treatment of primary varicella pneumonia in non-immunocompromised adults

TO THE EDITOR: Chicken pox is usually a benign childhood disease, and varicella pneumonia is rare among children. Chicken pox pneumonia in adults is also a rare occurrence, but when it does occur the morbidity and mortality rates are high, ranging from 15% to 25%.[1,2] Isolated reports describing the use of antiviral chemotherapy (adenine arabinoside[3,4] or acyclovir[4-6]) for this problem have appeared in the literature. We describe four adult non-immunocompromised patients who were successfully treated for varicella pneumonia using acyclovir (9-[2-hydroxyethoxymethyl] guanine) and respiratory support.

Case Reports. All four patients had respiratory insufficiency of varying severity, including one (case 3) with adult respiratory distress syndrome (ARDS). The ages of the patients ranged from 24 to 61 years. The diagnosis of chicken pox was established on clinical grounds. All four patients had a history of household contact with a child who had chicken pox, and demonstrated characteristic maculovesicular rashes. Progressive respiratory insufficiency developed two to five days after the cutaneous eruption.

Roentgenographic examination of the chests of these patients on admission demonstrated interstitial infiltrates with confluence of varying degrees. The severity of clinical illness and the extent of the roentgenographic abnormalities correlated with the ages of the patients and the degree of the cutaneous rash. The older patients were the most ill. Cultures of blood and sputum, by induction or bronchial aspirate, were negative for bacteria. Presumptive diagnoses of varicella pneumonia were therefore made. The pertinent physiologic data and the respiratory support the four patients received are summarized in Table I.

All patients were given acyclovir, 500 mg intravenously every eight hours, after the presumptive diagnosis of varicella pneumonia was made. The duration of treatment ranged from six to ten days. All four patients demonstrated dramatic and complete clinical, physiologic, and roentgenographic resolution (Figs 1,2,3) on institution of acyclovir treatment, and they were discharged. During the course of treatment, all patients developed mild reversible elevations (50-88 U, mean 67.1 U) of serum glutamic oxalacetic transaminase (SGOT).

Discussion. Although chicken pox is one of the most common communicable diseases, less than 2% of cases occur in adults. However, adults with chicken pox have far more serious infections,[1] and pneumonia is much more likely to be present among adults with chicken pox.[2] Given the severity of varicella infection and mortality rates in adults, early diagnosis and aggressive management of patients with varicella pneumonia are the keys to optimal outcome, as was observed in the patients described here.

It is advisable to consider the possibility of pulmonary involvement in an adult with chicken pox. As seen in these four cases, respiratory insufficiency is rapid (as shown in case 2). In addition, some patients will need ventilatory support (case 3).

Acyclovir is a selective antiviral agent, which has been used intrave-

TABLE I. Physiologic Data and Description of Respiratory Support

				Supportive Treatment		
Patient	Age (yr)	Admission PaO_2 (torr)	(A–a) O_2* Gradient	O_2	Mechanical Ventilation	Positive End-expiratory Pressure
1 BR	57	59 on room air	51	+†	−**	−
2 SL	24	87 on 3 liter, nasal cannula		+	−	−
3 YM	61	50 on room air	71	+	+	+
4 DW	24	93 on room air	26	+	−	−

* (A-a)O_2 Alveolar-arterial oxygen gradient
† Patient received
** Patient did not receive

nously to treat varicella-zoster infections and disseminated herpes simplex infections at doses of 10 mg/kg and 5 mg/kg, respectively, every eight hours in patients with normal renal functions.[7] However, in patients with chicken pox pneumonia who have had successful outcomes, the dose of acyclovir used has ranged from 250 mg every eight hours[5] to a weight-adjusted dose of 10 mg/kg intravenously every eight hours.[4,6] Although experience is limited, we used acyclovir at a dose of 500 mg intravenously every eight hours. It is our belief that the early, aggressive use of acyclovir prevented the progressive worsening of respiratory insufficiency and allowed recovery from the pulmonary infection in these patients. We believe that treatment with antiviral chemotherapy should be started early in the course of this potentially life-threatening disease.

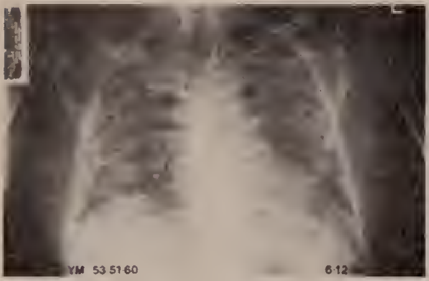


FIGURE 1. Admission chest film showing the severity of varicella pneumonia (case 3).

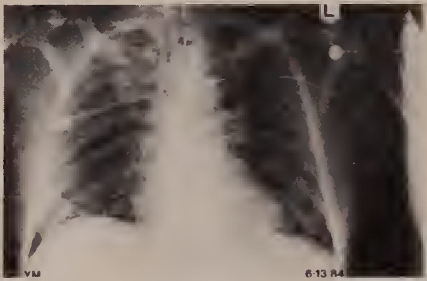


FIGURE 2. Chest film taken 24 hours after admission (note intubation) (case 3).

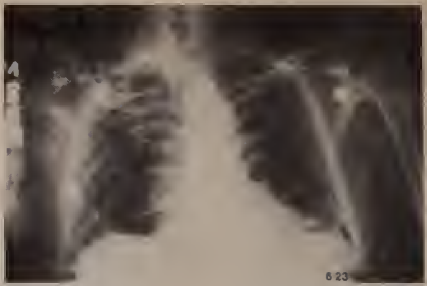


FIGURE 3. Chest film taken ten days after treatment with acyclovir (case 3).

RAJINDER CHITKARA, MD
RICHARD E. GORDON, MD
FAROQUE A. KHAN, MB
Division of Pulmonary Medicine
Queens Hospital Center Affiliation of
Long Island Jewish Medical Center
82–68 164th St
Jamaica, NY 11432

1. Varicella-zoster immune globulin for the prevention of chickenpox. *MMWR* 1984; 33:84–100.
2. Triebwasser JH, Harris RE, Bryant RE, et al: Varicella pneumonia in adults. Report of seven cases and a review of literature. *Medicine* 1967; 46:409–423.
3. Macfarlane JT, Smith FD, Finch RD: Vidarabine in fulminating chickenpox pneumonia. *Thorax* 1982; 37:226–227.
4. Landsberger EJ, Hager WD, Grossman JH: Successful management of varicella pneumonia complicating pregnancy. *J Reprod Med* 1986; 31:311–314.
5. Bryer A, Potgieter PD, Moodie J: Acyclovir and varicella pneumonia [letter]. *S Afr Med J* 1984; 66:515.
6. Van Der Meer JW, Thompson J, Tan WD, et al: Treatment of chickenpox pneumonia with acyclovir [letter]. *Lancet* 1980; 2:473–474.
7. Laskin OL: Acyclovir. Pharmacology and clinical experience. *Arch Intern Med* 1984: 144:1241-1246.

Taking stock: Physicians' memoirs

Manhattan Country Doctor: A Memoir. By Milton Jonathan Slocum, MD. 271 pp. New York, Charles Scribner's Sons, 1986. $16.95 (hardcover)

Surgeon! A Year in the Life of an Inner City Surgeon. By Richard T. Caleel, MD, with John Littell. 280 pp. New York, Rawson Associates, 1986. $15.95 (hardcover)

Once a Doctor, Always a Doctor. The Memoirs of a German-Jewish Immigrant Physician. By Heinz Hartmann, MD. 189 pp. Buffalo, NY, Prometheus Books, 1986. $18.95 (hardcover)

Fighting the Third Death. By Arthur Linksz, MD. 340 pp. New York, Julia F. Linksz, 1986. $19.50 (hardcover)

Although the college years traditionally divide students into those who can write and those who want to become doctors, the history of medicine would be written differently. We all are familiar with the great literary artists of this century and last who were physicians by trade, and who used the insights gained therefrom to fuel the novels that made them famous. It would seem the perfect marriage, then: a life spent in the service of humanity, with secret nights spent transmuting the reality into the highest art. Rare, indeed! The issue comes before us again as we consider no fewer than four books written by physicians in the past decade.

They are all, alas, autobiographies. Alas, I say, because there is something about the raw transmittal of personal information that keeps us bogged down out of reach of the higher world of literature to which we long to escape after a hard day of attestation forms, disappointing tomographies, and old ladies who have nowhere to go when discharge date arrives. If we pick up a book by a fellow physician, we want, if not art, then at least a rousing good time or an educational experience. Do we get it from these four volumes? Well, let me see . . .

Yes, from Dr Slocum, author of *Manhattan Country Doctor: A Memoir*, to whom I have totally lost my heart. He is an octagenarian, living in a retirement in California he doesn't deserve. This man is our dream: the doctor we wanted to be when we started out; the doctor who continued to be what we gave up being after we got too smart; the guy we want to commemorate on the coin of the realm along with Lincoln (whom he closely resembles in attitude if not in visage). He is the man whom television could not succeed in reproducing when it created Marcus Welby; the man the BBC would have created a miniseries for, had he been but British, Scottish, or practicing in the Punjab during the raj. I am so nuts for Dr Slocum, I will not give him up even when the world discovers him, and turns him into a piece of cinematic box office. He's our kind of guy.

His book is a recounting of his adventures as a GP in Hell's Kitchen for 40-some years. He had a wife like Ozzie Nelson's Harriet and a nurse like Dr Christian's Judy, and the three of them took on the world of the sick and turned it into one spectacular example of human connectedness in the face of poverty, crime, struggle, hardship, and endurance. With rollicking good humor, down-to-earth common sense, and a total lack of heroic egoism, our Dr Slocum reveals himself as a delightful, self-actualizing (There! I've finally found a use for that awful word!) candle who lights the darkness and has a darned good time flickering away at it.

Buy this book before your bookstore runs out of it. Lock yourself in the bathroom with it (unless your chamber-of-last-resort has a phone). Hug it to your bosom and relive your life with it. If the characters are recognizable as people you yourself treated . . . if your reactions are identically those of the narrator . . . if you feel that you are with a classmate, whatever his or her age . . . you're a good doctor. Yes, indeedy, this book is a good barometer of how good a doctor you are. Measure yourself up against ol' Doc Slocum and quiver if you don't make it. Then relax and enjoy, enjoy, enjoy. I couldn't put him down. I even had to restrain myself from running over to the building that housed his office and family for many years just to put up a plaque:

> Here Resided and Practiced a Man Who Was the Doctor We Are Coming to Know Only in Our Memories. Tarry Here, Stranger, and Consider What Was Lost! Revolt! Rebel! Restore our Heritage, Our Joy, Our Salvation!

Moving from the sublime, I thought, to the ridiculous, I trekked over to *Surgeon!*, one of those "with somebody" books, which disqualifies the writer from being the writer, and generally gives evidence that he could have done better himself. The author, an osteopathic surgeon from Chicago, has made little effort to rein in those qualities that made him a surgeon, and instead spreads them at our feet like an eager puppy who has brought home the family rabbit in his teeth. He gives us a year in the life of an inner city surgeon who lives to cut, break his residents in half, curse his way through the slime, then elect to remain on longer, hooked by the savagery of both his deeds and misdeeds. Although his good heart is not to be questioned, his attitudes *are*, representing as they do a great deal of sexism, a residual "fodder" concept of the clinic patient, and a clear-cut inability to tolerate the niceties of professional relationships.

I am struggling to remain fair. Dr-Caleel-with-Mr-Littell's book is a fast-paced, action-oriented recitative, where the blood from one gory operation sloshes over precariously into the next abdomen. Tragedy is real (no one could make that up) and true human emotions are wrung from the breast of even the hardened doctor/writer like myself. At times, despite myself I had to laugh aloud. How else to deal, for example, with the clinic patient with female troubles, who solemnly announces that her problem is "fireballs of the eucharist"?! It took me longer than the authors to figure out that she was suffering from fibroids of the uterus, but I doubt that I shall ever again run across this diagnosis without a quiver of a smile.

Considering that I have spent a good deal of my professional life trying to promulgate a different model of medical education than that practiced and openly espoused by Dr Caleel, it is perhaps unfair of me to review his book. Still, never having been to Viet Nam, I am willing to concede that perhaps he's on to something I just haven't run into. Let us leave it to each reader to judge. Macho man may speak to other macho men in tongues I cannot decipher.

A horse of a different color is Dr Heinz Hartmann. Unfortunately, his memoir, *Once a Doctor, Always a Doctor*, belongs to the "And then I did this . . ." and "I am still in touch with so-and-so . . ." school of medical autobiography. Having read many autobiographies of great and famous men, it occurred to him, he explains, that the ordinary public might want to read the biography of an ordinary man. I can't imagine why he should draw this conclusion. He airbrushes away the fact that extraordinary men are far more interesting than the man next door, whose biography we live with and in part write ourselves on a daily basis. He believes the world is interested, and so in fact, it is, but I suspect only *his* world, the world of the many friends, relatives, acquaintances, and, yes, detail men, whose names pepper its pages.

Dr Hartmann's book is most disappointing even for those of us who have no bias against the ordinary man (particularly if he is a doctor like ourselves) because he had the good fortune to live in extraordinary times, and then neglects to discuss them at all. A Jew fortunate enough to have escaped the Holocaust (unlike most of his family and friends), he has virtually nothing to say about it. His book is closed, and with it whatever might have made his autobiography worth the time of a busy reader. A man unfortunate enough to have had a vaccine-damaged child, he devotes only several pages to discussing his feelings and attitudes concerning it. Instead, we see his children grow, his grandchildren sport their first teeth, and his cronies thanked one by one for their services throughout his productive lifetime. Our only insight is what is inadvertently revealed when this immigrant physician wanders up to a small town in upstate New York, and makes the adjustment we are all only too grateful to have been spared.

Still, I commend Dr Hartmann warmly. He has troubled to write a chronicle for his family that coming generations will rightfully cherish. He has attempted to fight off the Third Death (described forthwith). I wish him a long and happy life.

As of this writing, I have not finished Dr Linksz' book, *Fighting the Third Death*, but not because I have abandoned it in frustration. I have merely decided to read it a bit at a time, unrushed by the need to complete this composite review. It is a book that demands a relaxed, contemplative attitude on the part of the reader, a willingness to step into the writer's canoe and float along with him on a sea of free association, philosophic meanderings, and pods of time rushing past in nonchronological order. If the past sentence has bewildered you, veer away from *Fighting the Third Death*. It is composed of the stuff most often heard by orthodox analysts who do not believe in clarification.

The few chapters I have read of this memoir of 18 youthful years in Hungary have already proved their worth. For example, I recently received a call from a young cousin whose attic-clearing efforts in a house about to be sold had produced family memorabilia consisting of old photographs, letters, my aunt's paintings. He wanted to share them with me. As I imagined the eight sibs, each so different, yet so alike, posed with their spouses at one moment in their life spans, the thought crossed my mind that with my death, and those of a handful of remaining relatives, these eight would at last be dead. Only two or three are now alive, in their 80s, waiting for the time when they will exist only in the memories of we few who survive. And when we die, their Third Death will have arrived. Dr Linksz has sensitized me to the awareness that while the body may die, and the soul, there is indeed another death which occurs when the last living person who knew the deceased dies. He is no more. An extraordinary thought.

The last of this autobiographical quartet appears to have been published privately by this ophthalmologist, whose dust jacket declares him a prominent clinician, researcher, and professor. It was written in 1977 and translated from the Hungarian, winning positive reviews from distinguished writers and critics from the old country. It clearly shows the nonclinical side of a man who once aspired to be a poet, writer, and leader of men, but ended up writing texts on vision.

He has written his memoir, by his own admission, to stave off his own Third Death, the fundamental reason that we all write—autobiographies, diaries, poetry, it matters not. Dr Linksz' book is an authentic search for meaning, for comprehension of the ineffable, for resolution of the fates that determine our inner and external lives. It is best read by philosophers, poets, and dreamers who are not obsessional about beginnings, middles, ends, story lines, logical progressions, or predigestion for the reader. I apologize to readers for selfishly withholding it to savor at my leisure. You know enough by now to decide whether or not you wish to read the inner workings of this man's head. If you are students of the Holocaust you may have a special interest; the author lost his father, the man against whom all men are measured, to the camps of Hitler. His photographs wrench the heart from the body.

As I consider the four books that have occupied the past several weekends, I am struck by the variety of styles in writing, the differences in focus, and the great variability in careers chosen by men who ultimately go on to put their thoughts to paper. It would seem that the whole genre of medical autobiography was worth studying in itself. To that end, I paced my own shelves, pulling down examples of medical autobiography immediately accessible to me, and I now contemplate them lined up on my desk. None are new, all bear mention in support of the idea that medicine may have much to learn from its members who have chosen to draw public meaning from their personal lives.

Here is Michelle Harrison's book, *A Woman in Residence*, reviewed for the *Journal* several years ago. What better book, with the exception perhaps of Fitzhugh Mullan's *White Coat, Clenched Fist*, gives a better description (however inadvertent) of the bitterness of the social revolutions in feminism and medical care of the past several decades?! What worlds they are apart from Drs Linksz and Hartmann!

And here is *Doctor in the Land of the Lion*, a precocious memoir of a medical student's adventures in East Africa, written by none other than the present editor of this journal, Dr Pascal James Imperato. It is high adventure.

At the other end of the life spectrum comes *Confrontations with Myself*, written by the great psychoanalyst Helene Deutsch. "I have a feeling [she writes], now that the biological end of my existence is near, that I have reached a height from which to review my life's achievements and disappointments." To relive them, she says, is a challenge. ". . . I hope I will succeed, while my identity is still intact, in re-creating the story of my life—what I was once, and what I am now." And, she cautions the reader, "I also hope that the poet who with his creative fantasy lies hidden within each one of us will not make a 'literary' tale out of my personal reality." The Third Death of this well-known woman will be a long time in coming.

The next item on my book shelf is my old favorite, *When Doctors Are Patients*, long since out of print and as timely today as the day these fascinating essays were collected into a book by two Montefiore Hospital clinicians. To read how these physicians all manipulated the health care system as only a physician can for the sole purpose of denying their own illnesses is a lesson in autobiography unsurpassed to this day.

If I were to be fair, I would include in this personal collection my own book, *So You Want to Be a Doctor*, which drew, inevitably and heavily, on my own experiences. I had kept copious notes during my medical school days chronicling the experience, and threw in a few pages verbatim. Predictably, these were the only passages ever questioned by the editors; otherwise, the book was published word for word as I had written it!

One of my great favorites is *The Horse and Buggy Doctor*, autobiography of one Arthur E. Hertzler, MD, and published in 1938. He is the quintessential country doc, the spiritual father of Dr Slocum, whose teacher he might have been but for circumstances of geography and fate. He was asked to write his autobiography and does it in a straight, no-nonsense style which recounts his long buggy rides, his kitchen surgeries, his useless meds, his heartbreaks, and his savage quest for knowledge, knowledge, and more knowledge which forever puts the lie to the myth of the LMD who no longer knows his anus from his antecubital fossa.

Dr Hertzler is timeless. Consider this remark from half a century ago:

> What is disease and what worry tries the skill of the most astute. A perplexing problem is the adolescent girl. Christian lady and plain animal sometimes wage a terrible subconscious battle. Sick or well she is a problem. The commonest complaint is monthly pains—a symptom more often than a disease. We doctors sometimes forget, as well as learn, about the young dysmenorrheaic. The trouble used to be perfectly amenable to treatment by us country doctors when we got hold of her first. The treatment was well described in such recent books as Hartshorne's *Practice of Medicine*. My edition is about eighty five years old. We now know polyglandular disturbances are responsible. Dr. Hartshorne does not say anything about that. We just added it. It doesn't mean that we know any more than he did. And, while advancing in theory, we have forgotten his treatment. He gave potassium iodide. These patients have small goiters, and when these disappear the dysmenorrhea does likewise . . . . The old doctors all knew potassium iodide would relieve dysmenorrhea in young girls. That is true yet. It will cure all cases except those complicated by other things, particularly malformations of the pelvic organs which naturally are not amenable to treatment. The use of potassium iodide for dysmenorrhea is one of the most valuable contributions ever written in a medical book. Yet it has been forgotten except by a few old papas.

Hertzler's autobiography is medicine, for medicine was his life.

Next, in paperback, is the granddaddy of them all, *Religio Medici*, by the 17th century physician and philosopher, Sir Thomas Browne, whose reconciliation of medicine and the ineffable, while not strictly speaking an autobiography, provides insights into the guiding light of this rich life. Sir William Osler was buried with his copy of this classic. And I know of at least one other person who was.

Finally, a hand-written manuscript, never committed to publication, although richly deserving, the memoirs of my own grandfather, Dr Joseph Isaac Bluestone. Written in 1930 at the end of a most interesting life (which was directed only in small part to the practice of medicine), it makes turn-of-the-century patients of New York's Lower East Side come very much alive. They could all be neighbors and friends of the patients of Dr Slocum, or my own, for that matter, separated only by a small consideration . . . a century in time. Perhaps all these folks were spared their own Third Death, kept alive in the pages of literature.

I have always found the biographies of the ordinary docs of this world more interesting than those of the Schweitzers, perhaps because there is more with which I can identify. I had always assumed that a steady stream of selfless biographical material would continue to flow through time, but recent events in medicine have led me to consider that the heroic medical autobiography may become an endangered literary form. Like the paucity of good literature from a crumbling civilization, the shrinkage of good medical vitae reporting is inevitable, as medicine becomes one more commodity from the company store. Nothing of interest to the philosophers of mankind can come from physicians who function as agents of the bottom line. It is even conceivable that someday our literature will resemble that of the advertising copywriters, government technocrats, and air traffic controllers.

Perish the thought.

NAOMI R. BLUESTONE, MD, MPH
Consulting Editor

ITALIAN BROADSIDES CONCERNING PUBLIC HEALTH. DOCUMENTS FROM BOLOGNA AND BRESCIA IN THE MORTIMER AND ANNA NEINKEN COLLECTION, NEW YORK ACADEMY OF MEDICINE

Edited by Saul Jarcho, MD. 421 pp. Mount Kisco, NY, Futura Publishing Co, Inc, 1986. $49.50 (hardcover)

Sometime during the Second World War, Walter Rappaport, an American dealer in stamps and antiques, bought from an unnamed professor in Italy a large batch of old documents which had been stored in an unused building that had to be cleared. A large part of this comprised rolls of old manifestos, which were later acquired by Mortimer Neinken. In 1957–1958 Mr Neinken donated this acquisition to the New York Academy of Medicine, which collection was formally designated as the Mortimer and Anna Neinken Collection. It consists of 1,970 Italian broadsides (broadsheets) dealing with public health, issued between the years 1576 and 1849. Although brief reports on the Neinken Collection were published in 1958, there has been no detailed description of its composition and no translations or summaries of selected portions were available. This has now been done by Dr Jarcho, who when confronted with the prospect of preparing a volume of 4,000 pages, wisely decided to limit himself to the presentation of the original text of a set of 198 broadsides issued by the large city of Bologna and one smaller set of 44 broadsides from Brescia. In the present book each sheet is designated by its title (often containing the word *bando* or edict). The text of the typical "bando" usually begins with a long clause or sentence stating that news has been obtained that a disease has broken out in some foreign locality and that the vigilant authorities have issued a series of restrictions on the movements of persons, goods, etc, to stop the spread of the pestilence. Corporal or other punishments were threatened should the orders be disobeyed. There often follows a list of localities barred, and at the end the place and date of the broadside is given, followed by the name of the issuing authority.

The original documents generally measured 30 × 37 cm but a few larger and some smaller sheets were printed; most were printed on strong Italian paper that has withstood the passage of time with only minor damage.

Although Dr Jarcho admits that he did not attempt an epidemiologic study of these documents, he nevertheless more than adequately describes their importance. Thus the Bolognese series tells that by 1613 the city had banned a large group of countries or districts, including England, much of Germany, Switzerland, Austria, Bohemia, and Savoy because a disease had occurred in these localities. The name of the disease, whether confirmed or suspected, is rarely given, although Constantinople has been mentioned as a source of plague. No mention of plague was given with regard to other countries such as Scotland, Ireland, or Scandinavia, not even when Turkey, Russia, Egypt, Tunis, Tripoli, or the main Mediterranean islands were named. Nevertheless, the imposed regulations imply that plague must have been the principal danger. Many edicts refer to animal diseases and prohibit or restrict the importation of animals, the entry of persons handling cattle, the sale of meat or hides, the way dead animals should be buried, etc.

As Dr Jarcho points out, the documents bear evidence of a powerful and well organized society, hierarchically responsible to the ecclesiastical or other head of state. Much incidental knowledge on all the strata of the medieval Italian communities could be gleaned from the study of the whole Neinken Collection or at least from the present book.

Those who admire Dr Jarcho's scholarship, his dedication to the history of medicine, his skill in transmitting his erudition without pomposity, and the clarity and distinction of his writing, will have to hitch still higher the degree of their respect after having read or perused the new fruit of his labors.

LEONARD JAN BRUCE-CHWATT, MD
Wellcome Tropical Institute
London, England

COLONIC NEOPLASMS

Edited by Hans Elias. 184 pp. St Louis, Mo, Ishiyaku EuroAmerica, Inc, 1985. $30.00 (hardcover)

This book attempts to engender a better understanding of colonic cancer. The author is highly qualified to write it, judging by his bibliography and time-consuming morphometric methods. The usefulness of the book, however, is somewhat questionable. Although the illustrations are outstanding, clear, and self-explanatory, the book's intended audience is unclear. For a resident in pathology or gastroenterology who seeks fundamental, basic information, the illustrations are helpful, but the text is confusing. Widely accepted terminology is not followed and new controversial designations, many without any clinical bearing, are introduced. For established pathologists this book may be provocative and interesting. The historical overview and new concepts presented are challenging, although not convincing. If established pathologists are the intended audience for this book then less emphasis on well-known facts would have been in order.

The author presents a historical background of cancer in general and the origins of cancer followed by a description of the normal architecture of the colonic mucosa. The general characteristics of colonic polyps and their origins in relation to normal structures are also given. After describing a well-differentiated adenocarcinoma as the prototype of a colonic carcinoma, the author develops theories to explain the multiplicity of lesions, metastasis, and defense mechanisms against carcinoma. Recruitment as an important process in carcinogenesis is introduced.

The author's ideas are expressed with clarity. Some of the concepts, though, are not convincing. Too much is concluded from a given photograph, such as the concepts of leukophagia and recruitment—transformation of non-neoplastic, ie, hepatocytes, into colonic carcinoma cells. The illustration showing recruitment in the liver appears to be more like regenerating hepatocytes.

The book is well organized in the sense that it presents a logical development of the author's ideas. However, too much emphasis is placed on accepted facts such as the multicentric origin of tumors. The historical overview could have been shortened.

This book differs from similar publications in that it is a mixture of pictorial representation and philosophical observation. Although the text is provocative and original, I do not think that the author's stated objective contributes to a better understanding of colonic carcinoma.

ELLEN I. KAHN, MD
North Shore University Hospital
Manhasset, NY 11030

COST CONTAINMENT AND DRGs. A GUIDE TO PROSPECTIVE PAYMENT

By Allen D. Spiegel, PhD, and Florence Kavaler, MD. 565 pp, illustrated. Owings Mills, Md, Rynd Communications, 1986. $49.00 (hardcover)

The Social Security Amendments of 1965 created the Medicare and Medicaid programs, and ushered in an era of expanding health care resources. Employers, purchasing private insurance for their employees, soon followed suit. Most readers of the *Journal* have spent the greater part of their professional lives in the resulting milieu of widespread third-party coverage and spiraling costs.

Eighteen years later, the Social Security Amendments of 1983 officially signaled the end of this era. They mandated a new system for Medicare reimbursement of in-patient hospital care, aimed at reversing two decades of rising expenditures. Unlike the patchwork of state and federal cost containment legislation passed in the 1970s, the 1983 Medicare law did not tinker with the existing system, modifying a rule here and capping a payment there. Rather, it changed the basic framework of reimbursement that had existed for most of this century, replacing retrospective, per diem payments with a prospective payment system (PPS) based on diagnosis related groups (DRGs). There are already signs that the prospective payment approach may soon spread beyond acute hospital care for the elderly, to encompass most of the care rendered to many other Americans.

Cost Containment and DRGs: A Guide to Prospective Payment is a comprehensive analysis of this fundamental change in our health care system. The authors have extensive experience in the delivery of medical care and in the analysis of health care reimbursement. Between them, they have looked at these issues first-hand from a number of perspectives; as a physician, a hospital director, a consultant, a regulatory official in local government, and a federal administrator.

The book begins with a primer on the prospective payment system and DRGs; 80 pages on the current workings of the system, including concise explanations of its more arcane components. This basic material is also available elsewhere, though it is rarely presented in such a clear manner, spelling out the regulatory details and illustrating them with concrete examples.

What is *not* available elsewhere is the material contained in the book's other 480 pages. *Cost Containment and DRGs* examines the impact of PPS on the structure and operation of hospitals, on medical records systems, on physicians, nurses and other health professionals, and on patients. It explores the extension of PPS to psychiatric, long term, and ambulatory care services, and to the non-Medicare population. It also discusses the impact of DRGs on the quality of clinical care. The final 50 pages of the book are devoted to future possibilities and predictions.

This volume owes one of its greatest strengths to the varied backgrounds of its authors. Patients, physicians, hospital administrators, and regulatory officials have widely varying opinions about the initial impact of DRGs, and predict very different possibilities for the future. Spiegel and Kavaler present these contrasting perspectives with a great deal of objectivity. The book is well organized and written with clarity and wit. It contains a wealth of references to the medical, management, public health, and financial literature, as well as numerous up-to-date tables, charts, and sample forms. The index is extensive, and the technical terms and abbreviations that can obscure discussions of prospective payment are fully listed and defined in a glossary.

Clinicians, administrators, and planners who deal with the prospective payment system will find that this is the most definitive reference available to date.

STEPHEN ROSENBERG, MD, MPH
Columbia University
School of Public Health
New York, NY 10032

NIGHT CALLS. THE PERSONAL JOURNEY OF AN OB/GYN

By Henry Eisenberg, MD, Arlene Eisenberg, and Howard Eisenberg. 281 pp. New York, Arbor House Publishing Co, 1986. $16.95 (hardcover)

This is an intriguing title for a fairly disappointing book. Henry Eisenberg, the physician who co-authored it with his writer-brother and sister-in-law, explains in the opening pages that the book was conceived as a way to draw together brothers who had never been close. Apparently in that respect it succeeded. Perhaps for them that is enough. Certainly it is far from clear who else the authors are writing for. Whether from the perspective of a physician or of a woman needing care from one, much is left to be desired.

Most of the first part of the book relates the story of Dr Eisenberg's career. His decision to go to medical school, his experiences once there, his rotating internship at a large inner-city hospital, his two years of residency at Yale, his years practicing in the Air Force, and his struggle to establish a private practice, as well as his courtship, marriage, and early parenthood make a mildly interesting, though hardly unusual or compelling, account of how it was—and in some ways still is. The tale, however, is superficially told, and whether an accurate portrayal or an artistic embellishment to contrast with the "New Man" who supposedly develops later, Dr Eisenberg seemed an impulsive, rather thoughtless young man.

After the turning point—a crisis in his marriage when his long-suffering wife confronts him with her dissatisfaction with their relationship—the remainder of the book is largely devoted to a description of how and why he now practices as he does. The practice style itself is benign. Dr Eisenberg seems sincere and largely successful in his attempt to balance the good use of technology, medical skills, and experience with good communication and cooperation in an era when patients' requests (and sometimes demands) and the threat of lawsuits make ob/gyn practice ever more stressful. But again, while he claims to "tell it like it is," the story suffers from the superficial treatment of its most crucial elements. Much is said about Dr Eisenberg's malpractice trial, for example, while almost nothing is revealed about how he and his wife actually confronted and reconstructed their marriage. Increased time and involvement at home were apparently part of this, yet almost nothing is said about his three children or the conflicts of ob/gyn practice and parenthood. Is the "personal journey of an ob/gyn" even in 1986 no more than window-dressing on a life still confined almost exclusively to work?

Dr Eisenberg begins and ends his book noting the vague feelings of dread he has struggled with of late—fears of the heavy responsibilities he carries and of the lawyers waiting in the wings. No doubt many if not most physicians can relate to these feelings and welcome a thoughtful exploration of them. Unfortunately none is offered. The lay reader may be reassured, but the physician can only be disappointed with the glib conclusion: "What could I possibly do when I retire that would be better than what I'm doing now?"

JOAN E. LISTER, MD
112 Henry St
Cambridge, Mass 02139

CERVICAL ADENOCARCINOMA: A COLPOSCOPIC ATLAS

By Minoru Ueki, MD. 73 pp, illustrated. St Louis, Mo, Ishiyaku EuroAmerica, Inc, 1985. $30.00 (hardcover)

This colposcopic atlas is a well-organized, pictorially splendid book about cervical adenocarcinomas, about which relatively little is known because their significance has been eclipsed by squamous neoplasms. Not only are the majority of invasive cancers of the cervix squamous, but when preinvasive neoplasia is diagnosed it is almost exclusively squamous.

In decades past, the frequency of adenocarcinomas was reported to be about 5% of cervical cancers. This relative frequency has been rising. The current frequency is upwards of 10%. It is unclear whether this increase is real, or simply relative, due to better screening for and elimination of squamous precursors.

Either way, cervical adenocarcinoma is playing a greater role on the clinical scene. Therefore, this is a timely publication. However, this atlas will have limited appeal. It will be of interest mainly to advanced colposcopists and oncologists with an interest in colposcopy and these tumors.

This atlas is unique, with 67 beautifully reproduced colpophotographs of adenocarcinomas culled from the author's experience with 90 patients over a 15-year period. The book begins with several short introductory chapters on colposcopy. The third chapter lists the general international classification of colposcopic findings. The author then describes his colposcopic findings in cervical adenocarcinomas.

The fourth chapter is devoted to the microvasculature. Six types of normal microvasculature (network-like, red dotted, red spotted, branch-like, loop-like) and seven atypical variations (glomeruloid, corkscrew, mosaic, tendril, waste-thread, willow branch, and root-like) are described. Then follows pictorial chapters on adenocarcinomas: early, typical, mucus-secreting, and adenosquamous. The final, brief chapter is on endocervicoscopy.

The early diagnosis of cancers and their precursors is the main clinical use of colposcopy. The author discusses the difficulties of accomplishing this with adenocarcinoma of the cervix. Adenoprecursor lesions lie below the surface and cannot be seen by colposcopy.

The principal use of colposcopy with adenocarcinoma is the detection of macroscopically occult disease. More of the introductory sections could have been devoted to this topic, with some elucidation of how the patients with occult diseases came to colposcopy—whether through abnormal cytology or whatever. The general, introductory remarks about colposcopy could have been omitted since this is not a general atlas of colposcopy. Nineteen (29%) of the 65 regular colpophotographs (excluding the two endocervical photos) appeared to be of occult invasive cancers. More use could have been made of this material and subject.

The classification of adenocarcinomas into typical, mucus-secreting, and adenosquamous is straightfoward, and the notes on mucus-secreting tumors are particularly helpful.

The author's colposcopic terminology is a bit confusing. Five characteristics are described: papillary, transformation zone-like, atypical vessels, granulation-like, and net-like changes. This reader could not discern what was meant by "net-like changes" in the text or photographs. The use of standard terms describing changes in color, contour, and vessels, plus the addition of descriptions of transformation zone-like changes, would be less confusing. The author uses seven terms to describe atypical vessels, which is a lot, but they are well illustrated in sketches and photographs. With this pioneering effort, it is possibly better to be more rather than less thorough.

No patients with diseases due to diethylstilbesterol (DES) use are described, because of a lack of them at the Osaka Medical Center.

Colposcopy can on occasion be helpful in the diagnosing and staging of endometrial carcinoma. Although this is not the main theme of this atlas, a few photographs of cervical involvement with endometrial carcinoma might have been of interest. The section on endocervicoscopy is too brief and there is no bibliography. The few references are listed only by year, leaving the interested reader to his own devices to search out the articles.

In sum, this is a groundbreaking, specialized atlas, well put together by Professor Ueki, who has thought about cervical adenocarcinoma for many years. Few others can make this claim. The book is a valuable addition to the literature and interesting reading and viewing for those concerned about the early diagnosis of cervical adenocarcinoma.

FREDERICK H. SILLMAN, MD
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

OB/GYN EMERGENCIES. THE FIRST 60 MINUTES

Edited by Roy G. Farrell, MD. 341 pp, illustrated. Rockville, Md, Aspen Publishers, Inc. 1986. $37.00 (hardcover)

This is a book written by and for emergency room physicians. None of the authors appear to be obstetricians or gynecologists. In the preface, the authors acknowledge that this book does not limit itself to emergency medicine but also covers aspects of ambulatory care in obstetrics and gynecology. Thus they think that the text will be of "almost equal value to general internists, family practitioners and non-hospital based emergency physicians who treat women with emergencies and perceived emergencies of their reproductive system in an outpatient setting."

By their backgrounds as examiners for the American Board of Emergency Medicine, the authors are qualified to identify the relevant areas considered by that body. As such, the book has meaning for this discipline but a good deal of the material in it can be found in existing textbooks of obstetrics and gynecology.

The book consists of five sections and 19 subsections. One of the subsections is on neonatal resuscitation. The illustrations, which consist of line drawings, are helpful. However, many of the photographs and tables are borrowed from other articles and books. One possible future change would be to produce this as a softcover spiral-bound book with room for notes. This might lower the cost and expand its usefulness.

There are discussions of certain topics which might have benefited had the authors provided more information about the examples they use. In the chapter on emergencies of delivery, for example, the performance of an episiotomy is described but not its repair. The management of breech delivery is reviewed but the possibility of nuchal arms is overlooked. In the same section contraindications to tocolysis are listed but the side effects of the drug are not mentioned. Endotracheal intubation is described in the subsection on neonatal resuscitation but it is not illustrated. In the subsection on third trimester bleeding vasa previa is mentioned but the diagnostic procedures are omitted. In the list of complications of recent therapeutic abortions the possibility of uterine perforation is not considered. With ectopic pregnancy gynecologic consultation appears to be requested late in the process rather than early, unless the patient is in shock. This might well result in an adverse outcome. The need for early gynecologic consultation can be critical. The management of abnormal vaginal bleeding in the postmenopausal patient is reviewed without drawing attention to the importance of endocervical curettage as an aid to determining the extent of the disease.

The tabular presentation on the use of immunizing agents during pregnancy and the subsection on the differential diagnosis of abdominal pain are quite helpful. The subsections on pelvic inflammatory disease and gynecologic emergencies in the prepubertal female should be of considerable aid to the emergency room physician.

The book ends with a very handy subsection on ob/gyn emergency department procedures but does not include monitoring techniques such as the use of central venous lines or Swan-Ganz catheters. This text can serve as a brief guide for physicians in facilities where on-site obstetric and gynecologic consultations may be difficult to obtain promptly. Subsequent editions will have the benefit of having had the text tested under fire and accordingly refined.

RANDALL D. BLOOMFIELD, MD
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

ERRATUM

Because of an editing error, a review of *Tumor Markers: Clinical and Laboratory Studies* published in the November 1986 issue of the *Journal* erroneously referred to the book's author, Dr Janis Klavins, as female.

Effects of copper deficiency suggest sex-related differences in vulnerability to heart disease

Researchers have demonstrated a sex-related difference in the development of coronary heart disease associated with a low-copper diet. Nutritional studies suggest that females are somehow protected against the effects of copper deficiency. This finding prompts several questions: Are females less susceptible to heart disease? If so, what aspect of their physiologic make-up serves as a protecting factor? And how reliable and accurate are human nutritional studies, which commonly involve only male subjects?

Previous nutritional studies have established that feeding animals a low-copper diet can lead to symptoms of coronary heart disease, and that this effect could be greatly enhanced by a diet high in fructose—the form of sugar found in fruits. Now, scientists at the Agriculture Department's Human Nutrition Research Center (Beltsville, Md) have found that after 11 weeks on a low-copper, high-fructose diet, all of the male subject rats had died of heart disease—with symptoms of heart enlargement, anemia, and increased cholesterol levels. On the other hand, female rats maintained on the same diet had high cholesterol levels but showed no evidence of heart disease, according to an article in *Science News* (1987; 131:70).

In the future, the researchers wish to examine the possible role of sex hormones in protecting the females or in making the males more susceptible to heart disease. In addition, "it's unlikely that the dramatic sex-differentiated effects seen in the low-copper, high-fructose experiments are unique to copper," notes one researcher. "Many trace metals have similar effects on the body . . . if there is a sex-linked vulnerability to copper deficiency's effects, there may be a similar one for zinc, manganese, vanadium or nickel." The possibility of such sex-linked differences in response to dietary components may have been overlooked in the past, because many nutritional studies include only males, with the intended purpose of eliminating the effects of fluctuating sex hormone levels among females.

Oral contraceptives and breast cancer risk

For women less than 45 years of age, the use of oral contraceptives before age 25 or before the birth of their first child does not increase their risk of breast cancer. These findings, reported by Miller et al (*Obstet Gynecol* 1986; 68:863), are based on a case control study that compared 521 patients with breast cancer to 521 control subjects. The subjects, all under 45 years of age, were matched for age, time of interview, and geographic location, in order to control for differing patterns of availability and usage of oral contraceptives.

In addition, Miller et al report that long-term use of oral contraceptives does not affect the risk of breast cancer, and that no specific brand of oral contraceptive is associated with an increased risk. The authors observed no elevation in risk even among subjects with underlying risk factors such as a family history of breast cancer or a history of cystic breast disease.

The use of ultrasonography in fine-needle aspiration biopsy of breast masses

Ultrasonography has proved useful and cost-effective as a method of guiding fine-needle aspiration biopsies of breast masses. In addition, high-resolution real-time ultrasonography requires no anesthesia, carries virtually no risk to the patient, is effective in the visualization of small and deep masses, and enables the physician to monitor the biopsy procedure.

Palpation has been the method of choice for guiding biopsies of breast masses. However, palpation does not allow for visualization of the mass and can be ineffective with small or deeply situated masses. In a recent issue of *Radiology* (1987; 162:409–414), Fornage, Faroux, and Simatos recounted their experience with ultrasonography (US)-guided fine-needle aspiration biopsy, and compared the accuracy of this procedure to that of palpation.

The authors performed 111 US-guided fine-needle aspiration biopsies in 99 patients using high-resolution ultrasound probes connected to real-time scanners. One aspiration usually produced enough material for diagnostic purposes. The procedure was performed on an outpatient basis.

The aspiration was successful in 110 (96.5%) cases. Cytologic diagnoses made based on biopsy specimens were confirmed following surgery, bacteriologic studies, or mammographic follow-up. Initial cytologic diagnoses were negative in all 49 cases of cysts, in eight of 11 (73%) cases of fibroadenomas (the biopsy specimen was insufficient in three of 11 cases), in four of five cases of benign dystrophic nodules (one biopsy sample was inadequate), and in all three cases of abscesses. Cytologic analysis yielded positive findings in 35 of 38 (92%) cases of carcinoma, and produced suspicious results in three cases. Twelve cases of malignancy were nonpalpable. There was one false-positive result among the 73 cases of benign masses: a positive cytologic report in the case of a papilloma was later found to be in error.

The authors concluded that, "Whenever a tumor has been detected with both mammography and real-time US, the latter technique should be preferred for biopsy guidance, since it is accurate, nonionizing, expeditious, and associated with no additional cost."

The role of apolipoprotein B-100 in cardiovascular disease

Add a new entry to the list of natural, yet potentially harmful, compounds that lurk in our bodies: apolipoprotein B-100, or simply apo-B. Stealing the limelight from low-density lipoprotein (LDL), which upstaged cholesterol as the cause of atherosclerosis, apo-B is now the focus of much attention.

Apo-B comprises the protein portion of low-density lipoprotein and "is probably the most crucial protein involved in atherosclerosis," according to a recent article in

---

NEWS BRIEFS are compiled and written by Vicki Glaser.

Science News (1987; 131:90-91). In the body, apo-B attaches to LDL receptors on cell surfaces, enabling the cells to absorb LDL—which acts as a carrier of cholesterol molecules—from the bloodstream.

High levels of LDL-cholesterol in the bloodstream can cause atherosclerosis. And deviations in the activity or quantity of apo-B could alter LDL-cholesterol uptake. Preliminary studies demonstrated a correlation between increased levels of apo-B and coronary artery disease. Subsequent analysis of the structure of apo-B has led to the identification of specific genetic forms of the molecule that have been linked to an increased risk of heart attacks, regardless of the overall level of apo-B or LDL.

Apo-B measurements cannot yet be used as reliable predictors of heart disease risk for the general population, the article states, but they may play an important role in identifying heart disease risk among people with a family history of cardiovascular disease and with normal levels of cholesterol and LDL. A better understanding of apo-B's role in atherosclerosis has led to new therapeutic options: antibodies to apo-B that would remove LDL from the bloodstream; and drugs that would block the binding of apo-B to cellular receptors. Further research on the structure and function of apo-B is required to assess its clinical value.

AIDS update

The *AIDS Record* is a new national newsletter that features information for professionals on the acquired immunodeficiency syndrome (AIDS). The first issue of this twice-a-month publication, which appeared in December 1986, addressed topics such as the status of drugs to combat AIDS, the results of military testing for HIV antibody, and the opening of the first residential center for children with AIDS. The *AIDS Record* also provides epidemiologic data and educational and training resources.

Two New York universities will receive three-year grants from the National Institute of Mental Health (NIMH) to develop training programs for health care workers who treat patients with AIDS and their families. The Nursing Division of the New York University (NYU) School of Education, Health, Nursing and Arts Professions and the Research Foundation of the State University of New York at Stony Brook are among five recipients of NIMH grants that total more than $3 million, according to the *AIDS Record* (1986; 1:7).

The NYU Nursing Division will conduct workshops to familiarize health care workers with the risks involved in treating patients with AIDS and with the psychological and emotional needs of these patients. In addition, NYU plans to provide training guides on videotape.

The European Economic Community (EEC) is developing a campaign to help control the spread of AIDS in Africa. Receiving financial support from the 12 EEC members, the program would focus on increasing public awareness of AIDS and testing blood bank supplies for antibodies to the AIDS virus.

The Pasteur Institute (France) will begin testing an AIDS vaccine in humans sometime this year, according to a West German medical journal. A source at the Pasteur Institute was quoted as saying that an AIDS vaccine should be available to the general public by 1991. The vaccine would consist of deactivated portions of the virus' protein coat and would only be effective in persons whose blood tests negative for the AIDS virus.

In 1986, twice as many countries (91) reported cases of AIDS as in 1985, the World Health Organization (WHO) reports. This probably does not accurately represent the spread of the disease, but rather the increase can be partially attributed to more countries recognizing and testing for AIDS. According to WHO, there are now more than 10,000 known cases of AIDS worldwide, with 5–10 million people having been exposed to the AIDS virus.

The Centers for Disease Control (Atlanta) has reported that AIDS rose from the 13th to the 11th leading cause of premature death, or "lost years of potential life," in the United States. Heart disease, cancer, and accidental injuries still rank as the leading causes of premature death in this country.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Clarence F. Ackernecht, MD, Schenectady. Died January 1, 1987; age 92. Dr Ackernecht was a 1923 graduate of Albany Medical College of Union University, Albany. He was a member of the Medical Society of the County of Schenectady and the Medical Society of the State of New York.

Herbert Adler, MD, West End, New Jersey. Died January 12, 1987; age 94. He was a 1914 graduate of Columbia University College of Physicians and Surgeons. Dr Adler was a member of the New York County Medical Society and the Medical Society of the State of New York.

Leonard Emanuel Ancona, MD, New York. Died November 21, 1986; age 44. Dr Ancona was a 1968 graduate of New York Medical College. He was a Diplomate of the American Board of Internal Medicine and a member of the New York County Medical Society and the Medical Society of the State of New York.

Kamila Czeslawa Balk, MD, New York. Died November 14, 1986; age 79. Dr Balk was a 1931 graduate of Uniwersytet Jana Kazimierza Wydzial Lakarski, Lwow-Lemberg. Dr Balk was a member of the New York County Medical Society and the Medical Society of the State of New York.

Clara Adele Brown, MD, Oswego. Died September 29, 1986; age 91. She was a 1921 graduate of the University of Toronto Faculty of Medicine, Toronto. Dr Brown was a member of the American Public Health Association, the American School Health Association, the Medical Society of the County of Oswego, and the Medical Society of the State of New York.

Lawrence L. Carlino, MD, Lewiston. Died December 11, 1986; age 84. Dr Carlino was a 1927 graduate of the University of Buffalo School of Medicine. He was a member of the Academy of Medicine, the American Academy of Family Practice, the Medical Society of the County of Niagara, and the Medical Society of the State of New York.

George Colling Cash, MD, Olean. Died January 27, 1987; age 75. He was a 1937 graduate of the University of Nebraska College of Medicine, Omaha. Dr Cash was a member of the Medical Society of the County of Cattaraugus and the Medical Society of the State of New York.

Ramon Castroviejo, MD, New York. Died January 1, 1987; age 83. He was a 1927 graduate of Facultad de Medicina de la Universidad de Madrid. Dr Castroviejo was a Diplomate of the American Board of Ophthalmology and the American Board of Plastic Surgery. His memberships included the Academy of Medicine, the American Society of Plastic and Reconstructive Surgery, the American Academy of Ophthalmology and Otolaryngology, the New York County Medical Society, and the Medical Society of the State of New York.

Morris Chester, MD, Lakehurst, New Jersey. Died November 20, 1986; age 88. Dr Chester was a 1940 graduate of Deutsche Univerzita Medizinische Fakulta, Praha. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Albert A. Cohen, MD, New York. Died December 25, 1986; age 71. He was a 1943 graduate of Faculty of Medicine, National University of Athens. Dr Cohen was a Diplomate of the American Board of Otolaryngology and a member of the Academy of Medicine, the American Academy of Facial Plastic and Reconstructive Surgery, the American Academy of Otolaryngology, the New York County Medical Society, and the Medical Society of the State of New York.

Morris Dinnerstein, MD, New York. Died November 28, 1986; age 91. He was a 1918 graduate of Columbia University College of Physicians and Surgeons, New York. Dr Dinnerstein was a Diplomate of the American Board of Internal Medicine and a member of the New York Pathological Society, the New York County Medical Society, and the Medical Society of the State of New York.

Sol Dombeck, MD, White Lake. Died January 12, 1987; age 77. He was a 1935 graduate of Tulane University School of Medicine, New Orleans, Louisiana. Dr Dombeck was a member of the American Academy of Family Practice, the American Psychiatric Association, the Sullivan County Medical Society, and the Medical Society of the State of New York.

Donald Edward Donovan, MD, East Aurora. Died December 20, 1986; age 80. Dr Donovan was a 1931 graduate of State University of New York Health Science Center at Buffalo. He was a member of the American Academy of Family Practice, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Thomas Murray Drislane, MD, Albany. Died December 13, 1986; age 66. He was a 1948 graduate of Albany Medical College of Union University, Albany. Dr Drislane was a Diplomate of the American Board of Psychiatry and Neurology and a member of the American Academy of Neurology, the Medical Society of the County of Albany, and the Medical Society of the State of New York.

Herman Ehrlich, MD, Mount Vernon. Died August 23, 1986; age 83. Dr Ehrlich was a 1937 graduate of the University of Vermont College of Medicine, Burlington. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Louis Fox, MD, Boca Raton, Florida. Died January 19, 1987; age 90. Dr Fox was a 1919 graduate of New York University School of Medicine, New York. Dr Fox was a Fellow of the American College of Surgeons and a member of the New York County Medical Society and the Medical Society of the State of New York.

John M. Galbraith, MD, Sun City, Arizona. Died November 10, 1986; age 82.

Dr Galbraith was a 1931 graduate of the University of Michigan Medical School, Ann Arbor. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Leo G. Goldberg, MD, LaJolla, California. Died December 6, 1986; age 82. He was a 1927 graduate of the State University of New York Health Science Center at Brooklyn. Dr Goldberg was a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Urology. His memberships included the American Urological Association, the American Fertility Society, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Joseph William Harris, MD, Citrus Heights, California. Died January 6, 1987; age 89. Dr Harris was a 1931 graduate of the University of Maryland School of Medicine, Baltimore. He was a member of the Medical Society of the County of Albany and the Medical Society of the State of New York.

Thomas Shetrond Hershey, MD, Tucson, Arizona. Died December 29, 1986; age 72. Dr Hershey was a 1940 graduate of Temple University School of Medicine, Philadelphia. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Eleonore Hinse, MD, Schenectady. Died December 24, 1986; age 70. Dr Hinse was a 1942 graduate of Faculteit der Geneeskunde Rijksuniversiteit te Utrecht. Dr Hinse was a Fellow of the American Academy of Family Practice and a member of the American Medical Women's Association, the Medical Society of the County of Schenectady, and the Medical Society of the State of New York.

John George Fred Hiss, MD, Syracuse. Died December 5, 1986; age 92. Dr Hiss was a 1917 graduate of the State University of New York Health Science Center at Syracuse. He was a Diplomate of the American Board of Internal Medicine and a member of the American Public Health Association, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Robert A. Holden, MD, Plattsburgh. Died November 1, 1986; age 51. He was a 1960 graduate of Harvard Medical School, Boston. Dr Holden was a member of the Medical Society of the County of Clinton and the Medical Society of the State of New York.

Robert Katz, MD, Spring Valley. Died December 5, 1986; age 57. Dr Katz was a 1959 graduate of Université de Lausanne Faculté de Médecine. He was a Fellow of the American College of Angiology. His memberships included the Academy of Medicine, the New York State Society of Internal Medicine, the Clinical Society of the New York Diabetes Association, the American Geriatrics Society, the Medical Society of the County of Rockland, and the Medical Society of the State of New York.

Victor B. Lampka, MD, Lancaster. Died December 22, 1986; age 76. He was a 1935 graduate of State University of New York Health Science Center at Buffalo. Dr Lampka was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Max Landsberger, MD, Bethesda, Maryland. Died January 1, 1987; age 92. He was a 1923 graduate of Medizinische Fakultaet der Ludwig Maximillians Universitaet, Muenchen, Bayern. Dr Landsberger was a Diplomate of the American Board of Pediatrics and a member of the Academy of Medicine, the American Academy of Pediatrics, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Alvah Leon Lord, MD, St Petersburg, Florida. Died December 31, 1986; age 91. Dr Lord was a 1920 graduate of the State University of New York Health Science Center at Buffalo. Dr Lord was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

John Edward Lowry, MD, Laguna Hills, California. Died January 15, 1987; age 89. Dr Lowry was a 1923 graduate of Howard University College of Medicine, Washington. Dr Lowry was a member of the Academy of Medicine, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Harold Lusskin, MD, Hollywood, Florida. Died December 2, 1986; age 91. Dr Lusskin was a 1919 graduate of Columbia University College of Physicians and Surgeons, New York. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

John Joseph Magovern, MD, Rockville Centre. Died November 25, 1986; age 70. He was a 1943 graduate of the State University of New York Health Science Center at Brooklyn. Dr Magovern was a Fellow of the American College of Radiology and a Diplomate of the American Board of Radiology. His memberships included the Radiological Society of New York State, the Academy of Medicine, the Radiological Society of North America, Inc, the Society of Nuclear Medicine, the American Academy of Compensation Medicine, Inc, the Nassau County Medical Society, and the Medical Society of the State of New York.

Richard Philip Messina, MD, Brentwood. Died December 27, 1986; age 48. He was a 1966 graduate of Facolta di Medicina e Chirurgia dell'Universita di Bologna. Dr Messina was a member of the American Ophthalmological Society, the Suffolk County Medical Society, and the Medical Society of the State of New York.

J. Lowry Miller, MD, Douglaston. Died December 12, 1986; age 84. Dr Miller was a 1927 graduate of the University of Pennsylvania School of Medicine, Philadelphia. He was a Diplomate of the American Board of Dermatology, Inc, and a member of the Academy of Medicine, the New York State Dermatological Society, the American Dermatological Association, the American Academy of Dermatology, the Society for Investigative Dermatology, the New York County Medical Society, and the Medical Society of the State of New York.

Frank Daniel Myers, MD, Slate Hill. Died August 27, 1986; age 78. Dr Myers was a 1933 graduate of New York University College of Medicine, New York. He was a member of the Medical Society of the County of Orange and the Medical Society of the State of New York.

Francis V. Oderkirk, MD, Victor. Died September 19, 1986; age 83. Dr Oderkirk was a 1931 graduate of the University of Buffalo School of Medicine. Dr. Oderkirk was a member of the Academy of Medicine, the Ontario County

Medical Society, and the Medical Society of the State of New York.

Angelo J. Perfetto, MD, Garden City. Died October 13, 1986; age 62. He was a 1950 graduate of the State University of New York Health Science Center at Brooklyn. Dr Perfetto was a Fellow of the American College of Pathologists, a Diplomate of the American Board of Surgery, and a Diplomate of the American Board of Pathology. His memberships included the New York Pathological Society, the American Society of Clinical Pathologists, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Abraham Max Rabiner, MD, Harbor Island, Florida. Died November 26, 1986; age 94. Dr Rabiner was a 1916 graduate of Albany Medical College of Union University. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Henry Rosenberg, MD, New York. Died November 3, 1986; age 90. Dr Rosenberg was a 1929 graduate of the Université de Paris Faculté de Médecine. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Nathaniel Ross, MD, New York. Died November 29, 1986; age 82. He was a 1929 graduate of Columbia University College of Physicians and Surgeons, New York. Dr Ross was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the Academy of Medicine, the American Psychoanalytic Association, the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Leon Rudorfer, MD, Brooklyn. Died January 3, 1987; age 76. He was a 1938 graduate of Facolta di Medicina e Chirurgia dell'Universita di Bologna. Dr Rudorfer was a Fellow of the American Psychiatric Association and a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Geriatrics Society, the Pan American Medical Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Filomeno Sanchez, MD, Scarsdale. Died December 22, 1986; age 56. Dr Sanchez was a 1956 graduate of Facultad de Medicina de la Universidad Nacional Autonoma de Mexico. Dr Sanchez was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Emanuel A. Schiffman, MD, Brooklyn. Died July 1, 1986; age 87. Dr Schiffman was a 1921 graduate of Fordham University School of Medicine, New York. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Leonard Schwarz, MD, Brooklyn. Died December 8, 1986; age 73. Dr Schwarz was a 1937 graduate of New York University College of Medicine, New York. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Amarendra Sengupta, MD, Rochester. Died January 6, 1987; age 54. Dr Sengupta was a 1956 graduate of Calcutta Medical College, Calcutta University, Calcutta, West Bengal. Dr Sengupta was a Fellow of the American College of Surgeons and the American College of Chest Physicians, a Diplomate of the American Board of Surgery and the American Board of Thoracic Surgery (Affiliated with the American Board of Surgery), and a member of the American Association for Thoracic Surgery, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

William Francis Sharkey, MD, Somers. Died January 14, 1987; age 79. He was a 1932 graduate of Georgetown University School of Medicine, Washington, DC. Dr Sharkey was a Fellow of the American Board of Gastroenterology. His memberships included the American Geriatrics Society, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

William T. Sherman, MD, New Rochelle. Died December 3, 1986; age 77. He was a 1937 graduate of Facolta di Medicina e Chirurgia dell'Universita di Bologna. Dr Sherman was a Fellow of the American Academy of Family Practice and the American College of Angiology. His memberships included the American Geriatrics Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

Morton William Shomstein, MD, Delray Beach, Florida. Died December 8, 1986; age 67. Dr Shomstein was a 1945 graduate of Chicago Medical School. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

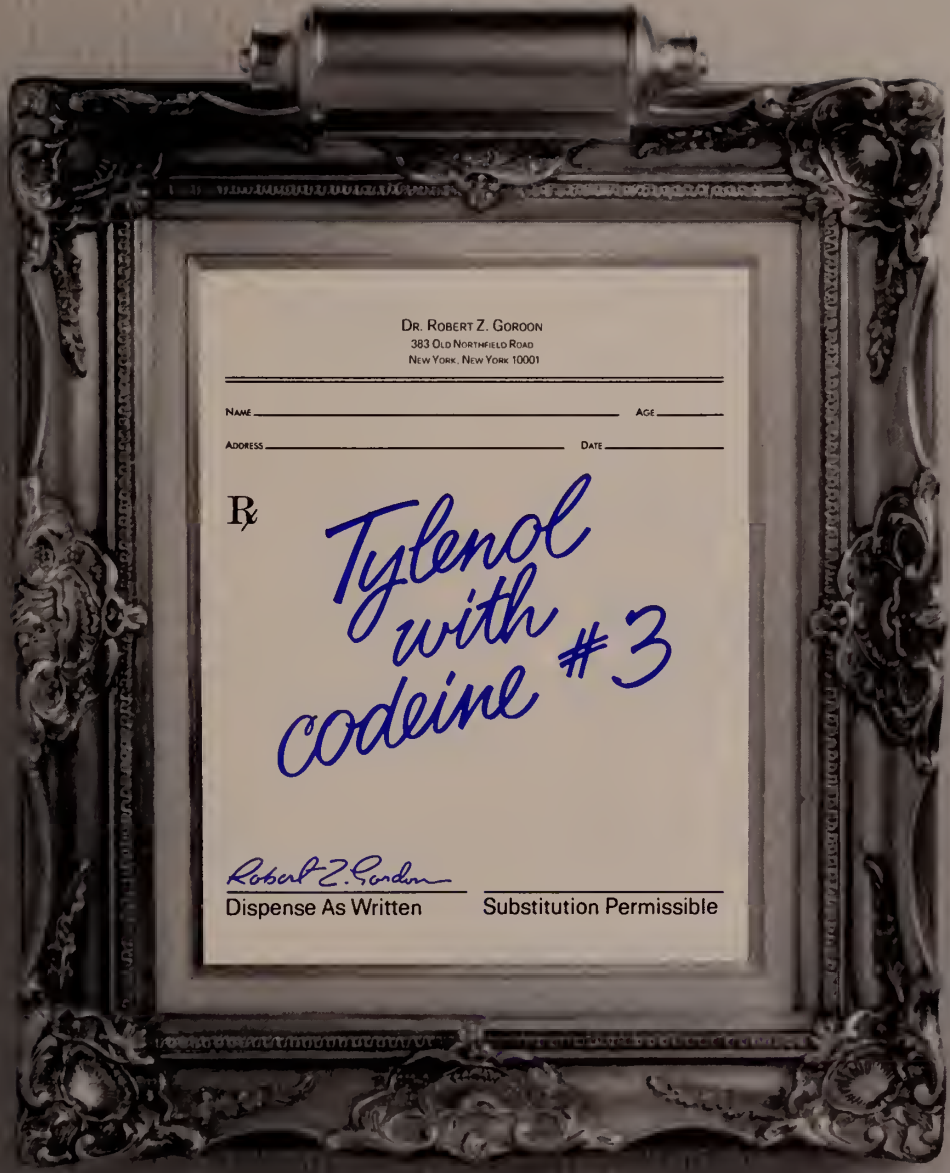
Alden J. Townsend, MD, Dansville. Died November 20, 1986; age 82. Dr Townsend was a 1934 graduate of Albany Medical College of Union University. He was a member of the Medical Society of the County of Livingston and the Medical Society of the State of New York.

Renato Paul Valt, MD, Glen Head. Died November 30, 1986; age 55. He was a 1958 graduate of Università Degli Studi di Genova, Facoltà di Medicina e Chirurgia. Dr Valt was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Roy E. Wallace, MD, Seneca Falls. Died December 2, 1986; age 78. Dr Wallace was a 1935 graduate of New York Medical College, Flower and Fifth Avenue Hospitals, New York. He was a Fellow of the American Academy of Family Practice. His memberships included the American Society of Anesthesiologists, Inc, the Seneca County Medical Society, and the Medical Society of the State of New York.

Kent D. Williams, MD, North Miami, Florida. Died December 26, 1986; age 81. Dr Williams was a 1933 graduate of the University of Rochester School of Medicine-Dentistry, Rochester. He was a member of the Medical Society of the County of Niagara and the Medical Society of the State of New York.

Woon Sang Yu, MD, Syracuse. Died December 2, 1986; age 54. Dr Yu was a1958 graduate of Seoul National University College of Medicine, Seoul. Dr Yu was a Diplomate of the American Board of Radiology and a member of the Radiological Society of North America, Inc, the Onondaga County Medical Society, and the Medical Society of the State of New York.



Dr. Robert Z. Gordon
383 Old Northfield Road
New York, New York 10001

Name ____________ Age ______

Address ____________ Date ______

℞

Tylenol with codeine #3

Robert Z. Gordon

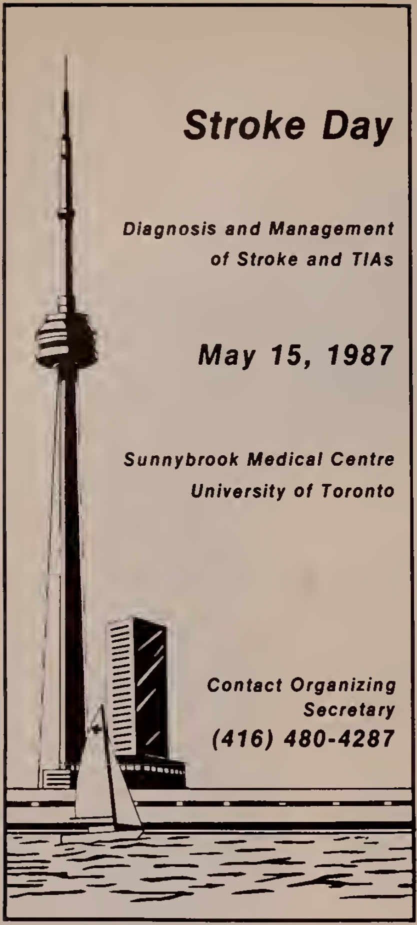
Dispense As Written

Substitution Permissible

CREATE A SIGNED ORIGINAL
to be sure your patients get...

Tylenol®
ACETAMINOPHEN
with codeine
phosphate
TABLETS Ⓒ III ELIXIR Ⓒ V

Tablets: Contain Codeine Phosphate*: No. 3 — 30 mg;
No. 4 — 60 mg — plus Acetaminophen 300 mg.
Elixir: Each 5 mL contains 12 mg Codeine Phosphate* plus
120 mg Acetaminophen (Alcohol 7%).



Stroke Day

Diagnosis and Management of Stroke and TIAs

May 15, 1987

Sunnybrook Medical Centre
University of Toronto

Contact Organizing Secretary
(416) 480-4287

June 13, 1987
7TH ANNUAL
ADVANCES IN GASTROENTEROLOGY
Bally's Park Place Hotel
Atlantic City, New Jersey

Sponsored by the University of Pennsylvania Gastrointestinal Department and the Continuing Medical Education Department of the Underwood Memorial Hospital,
Woodbury, New Jersey

Accreditation: Category 1 credit offered

Information:
Registration Supervisor
SLACK Incorporated
6900 Grove Road
Thorofare, New Jersey
08086-9447
(609) 848-1000

In New York City, for the Newest and Finest Medical Space, Remember:

THE 4 ESSENTIAL REQUIREMENTS

1. LOCATION!
Liberty Residences at Battery Park City

2. LOCATION!
Windsor Pavilion
(3rd Avenue & 32nd Street)

3. LOCATION!
205 East 64th Street (3rd Avenue)

AND

4. LOCATION!
Normandie Pavilion
(3rd Avenue & 95th Street)

For Further Information Contact Lester Schwalb
(212) 708-0846
Milstein Properties Corp.

FIGHT CANCER. EAT YOUR VEGETABLES.

There's strong evidence your greengrocer has access to cancer protection you won't find in any doctor's office.

Like broccoli, peaches, spinach, tomatoes, citrus fruits and various other types of fruits and vegetables. They may help reduce the risk of some forms of cancer.

Write for more information.

AMERICAN CANCER SOCIETY®

Cover to cover . . . . always good reading.

NEW YORK STATE
JOURNAL OF MEDICINE

MEDICAL MALPRACTICE
AN OVERVIEW

Contents

NEW YORK STATE
JOURNAL OF MEDICINE

CIGARETTE SMOKING,
TOBACCO, AND HEALTH
INTERNATIONAL PERSPECTIVES

Contents

NEW YORK STATE
JOURNAL OF MEDICINE

Contents

NEW YORK STATE
JOURNAL OF MEDICINE

Contents

In 1987, the *New York State Journal of Medicine* promises to provide timely and stimulating commentary, challenging research papers, analysis of clinical trends, debate on topical issues, and in-depth coverage of medical news. The journal will make a welcome gift to a colleague or student in the United States or abroad.

A free copy of the July 1985 issue, a special issue on smoking, will be included with each subscription while the supply lasts.

SUBSCRIPTION ORDER FORM Clip and mail

Circulation Department
New York State Journal of Medicine
420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042

Name____

Address____

____

	1 Year	2 Years	3 Years
U.S. & Canada	☐ $25	☐ $45	☐ $60
Foreign	☐ $30	☐ $55	☐ $75

Payment required with order.

EQUIPMENT—CONT'D

LIQUIDATION MEDICAL EQUIPMENT: Coulter Counter Model D-2. Hewlett Packard Ekg page writer, Fakuda portable Ekg, Digitech chemistry analyzer, Seralyzer chemistry analyzer, small centrifuge, x-ray cassettes and hangers. All at 50 to 60% less than dealers costs. Perfect functionally. Dr. Thomas Forschner: phone (718) 332-4654 or 896-0373.

REAL ESTATE FOR SALE OR RENT

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

EAST SIXTIES, PROFESSIONAL BUILDING. The preferred location for internists, cardiologist, rheumatologist. 7 treatment rooms, 2 reception areas to share with three other internists. Share NYC licensed lab, radiology, thermography. Full or part time. (212) 838-2860.

5th AVENUE/11th ST. Newly renovated, fully equipped medical office to share in elegant townhouse on historic 11th St. Ideal for primary care internists, FP, or OB/GYN. Call (212) 529-4330 10–5.

OFFICE SPACE AVAILABLE TO SHARE—1,500 sq. ft. in Medical Service Building at St. Agnes Hospital, White Plains. Call (914) 428-7480.

WESTCHESTER NEW YORK RESIDENCE, 26 minutes to NYC. Substantial English Georgian 6 br., sun rm., artist alcove, circa 1920, stripped, replastered, refinished mahogany, brick-stone-slate, 1/3 green acre, giant oaks. Central a/c, manageable-safe-secure-secluded. Turn of century area. $850K, Dr. Skalafuris (914) 668-9108, (216) 832-5077 or (202) 462-3344.

MEDICAL SPACE AVAILABLE in prestigious Stony Brook, Long Island. Close proximity to several hospitals including Stony Brook University, St. Johns Smithtown, Smithtown General and V.A. Medical Center. Many established practices presently in buildings. Conveniently located at 1212 and 1239 Route 25A. Suites available from 600 square feet and up. Competitive rates under new ownership and management. Princeton International Properties Corp. (212) 686-9474.

OFFICE SPACE AVAILABLE: Throgs Neck section of Bronx. Modern medical complex, 1500 square feet, part time/full time. Call (212) 409-9494.

MISCELLANEOUS

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuarial certification and employee statements of participation—partnership agreements and professionals incorporation—No insurance required—references upon request—Wachstock and Dienstag Attorneys at Law, 122 Cutter Mill Road, Great Neck, NY 11021 (516) 773-3322.

ARE YOU HAVING A PROBLEM RELATING TO CHEMICAL DEPENDENCE? If your answer is affirmative, contact Susan Kaplan, Attorney-at-Law, (212) 877-5998. Practice limited to legal problems directly involving your license to practice medicine—with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professionals), and as an Assistant District Attorney in Nassau County. Susan Kaplan, Esq., 165 West End Avenue, Suite 27P, New York, N.Y. 10023.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta, GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

FIRM OF GERALD GOLDBERG, CPA-ATTORNEY, formerly with IRS, announces his availability to prepare taxes and plan under changing tax environment, accounting, tax appeals, legal representation or practice transfers. (212) 406-1156 and (516) 565-6260.

PROFESSIONAL MISCONDUCT ATTORNEYS. William L. Wood Jr., formerly Executive Director of the New York State Office of Professional Discipline and Anthony Z. Scher, formerly Director of Prosecutions, announce the opening of their law office, Wood & Scher. Our recent tenure as chief enforcement officers has given us experience which will allow us to represent physicians in professional misconduct proceedings, malpractice licensure, license restoration, controlled drug proceedings, reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practitioners. Wood & Scher, Attorneys at Law, One Chase Road, Scarsdale, New York 10583. Telephone 914-723-3500.

MISCELLANEOUS—CONT'D

HAVE YOU BEEN CONTACTED BY A REPRESENTATIVE FROM THE OFFICE OF PROFESSIONAL MEDICAL CONDUCT? If the answer is yes, contact Susan Kaplan, Attorney-at-Law, (212) 877-5998. Practice limited to the crucial legal problems directly affecting your license to practice medicine—with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professions), and as an Assistant District Attorney in Nassau County. Susan Kaplan, Esq., 165 West End Avenue, Suite 27P, New York, N.Y. 10023.

PLANNING A MEDICAL FACILITY? Save time/money with custom-built, turn-key facilities and equipment, in 90 days. Free estimates. Lease or purchase. Call: Son Corporation collect (316) 263-4557.

TUNA THERAPY SEMINARS: Does your brain need a rest, your body need exercise, are your lungs in need of fresh air? Giant tuna fishing from Montauk Point, New York, aboard Montauk's finest charter boat "Dawn". Charters available for 1 to 4 serious medical anglers. Capt. Greg Beacher, RD-2, Box 16, Benson Drive, Montauk, NY 11954, (516) 668-9056.

COMPUTERIZE YOUR BILLING—Remarkably easy-to-use software automatically prints bills, statements, insurance forms. Menu-driven. Reports ageing balances, referral sources, income by time period, procedure & more. Installs automatically. IBM and compatibles. Solo practice only $399. Group practice, only $499. Free information. Demonstration disk with complete manual, $10. Call or write REM Systems, Inc., Dept. NY4, 281 Dug Hill Road, Brownsboro, AL 35741 (205) 534-3457. VISA, MC Accepted.

PORTABLE RADIOLOGY SERVICE: abdominal + pelvic ultrasound, cardiac echos including 2D + m-mode. portable x-rays. NYC and suburbs. Schedule weekly/bi-weekly in your office or visits to your patient's home. New portable hi-tech equipment. Hi-quality like standard office images, far superior to hospital portable images. No lead shielding needed for x-rays. University Radiology Service, (212) 928-5321 or (718) 339-5363.

Essential reading for every physician

NEW YORK STATE
JOURNAL OF MEDICINE

THE WORLD CIGARETTE PANDEMIC

NEW YORK STATE
JOURNAL OF MEDICINE

THE WORLD CIGARETTE PANDEMIC
Part II

PRIMUM NON NOCERE

NEW YORK STATE
JOURNAL OF MEDICINE

CIGARETTE SMOKING,
TOBACCO, AND HEALTH
INTERNATIONAL PERSPECTIVES

The December 1983, July 1985 and September 1986 issues of the **New York State Journal of Medicine** constitute the most comprehensive coverage on the subject of smoking ever published by a medical journal.

SEND A COPY TO COLLEAGUES AND MEDICAL STUDENTS in the US and abroad who might not normally receive the Journal. Or order an extra copy for your patients and yourself.

Extra press runs have enabled the New York State Journal of Medicine to offer single or bulk copies of its issues on smoking. As the official publication of the Medical Society of the State of New York since 1900, the Journal is one of the foremost independent peer-reviewed general medical journals in the country. Providing timely and stimulating commentary, challenging research papers, analysis of clinical trends, debate on topical issues, and in-depth coverage of medical news, the Journal makes important reading—and a welcome gift—to physicians and students in the United States or abroad. A free copy of the July 1985 issue will be included with each subcription.

The Following, Reduced Rates Are Now Being Offered On The Smoking Issues

Quantity	**Per Copy***
1–24	$4.00
25–49	$3.50
50–99	$3.00
100–149	$2.50
150–500	$2.00

ORDER FORM

Name______________________________

Address______________________________

City/State/Zip______________________________

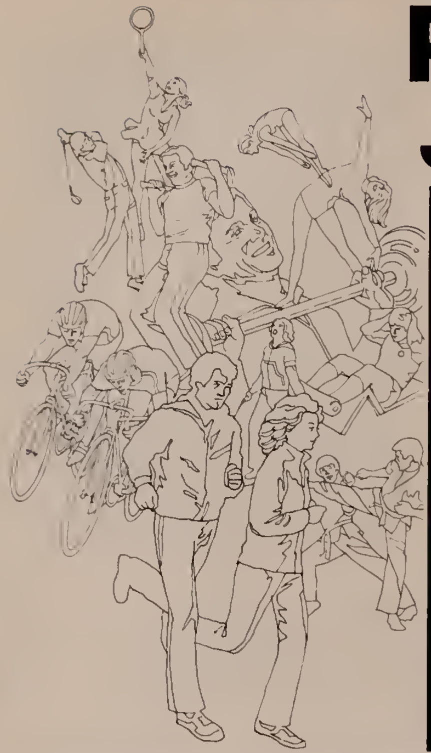
Please send _______ copies of December 1983, _______ copies of July 1985, _______ copies of September 1986 issue of the Journal.

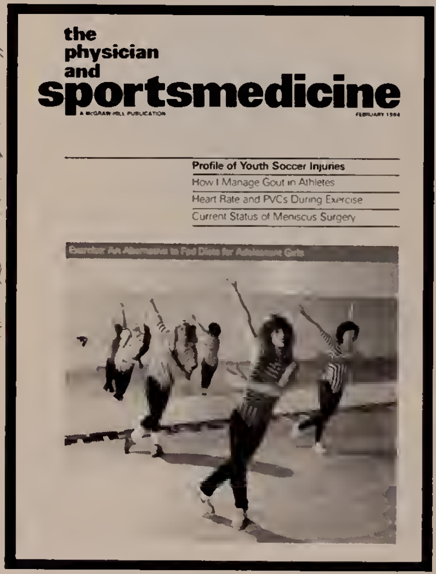
Enter my subscription to the Journal. ☐ $25.00 U.S. & Canada; ☐ $33.00 Foreign

Total amount enclosed $ ________ ***Note:** Domestic postage included. Please add New York state & local sales tax where applicable, or sales tax exemption certificate. Foreign postage additional.

Mail to: Circulation Department, **New York State Journal of Medicine**
420 Lakeville, Rd., P.O. Box 5404, Lake Success, N.Y. 11042.

OVER 66,000 FAMILY PHYSICIANS READ THIS JOURNAL




the physician and sportsmedicine
A McGRAW-HILL PUBLICATION
FEBRUARY 1984
Profile of Youth Soccer Injuries
How I Manage Gout in Athletes
Heart Rate and PVCs During Exercise
Current Status of Meniscus Surgery
Exercise: An Alternative to Fad Diets for Adolescent Girls

Practical information on the medical aspects of fitness and exercise.

Tennis elbow: Joint resolution by conservative treatment.

Hypertrophic cardiomyopathy and the athlete.

Effects of sunscreen use during exercise in the heat.

Overuse injuries to the knee in runners.

How I manage ingrown toenails.

McGraw Hill

Doctor . .

Do you, or a physician you know, have a problem with alcohol, drugs, or emotional illness?

If so, please contact the Medical Society of the State of New York's Committee for Physicians' Health, whose function is to assist, *confidentially.*

Telephone: 516-488-7777.

Index to Advertisers

Ayerst Laboratories	21A, 22A, 23A, 24A
Bankers Trust Company	10A
Ciba Pharmaceutical Company	7A, 8A
St. Clare's Hospital	5A
Classified Advertising	6A, 10A, 16A, 18A
Knoll Pharmaceuticals	11A, 12A, 13A
Eli Lilly and Company	14A
Marion Laboratories	1A, 2A
McGraw Hill Publications	4A, 20A
McNeil Laboratories	15A
Milstein Properties Corp.	16A
Roche Laboratories	3rd & 4th Cover
Upjohn Company	2nd Cover

To show you how many hypertensives stayed on INDERAL® LA (PROPRANOLOL HCl) after a major nationwide trial...

...we had
to find
just the
right room.

60,073 patients (90%) who started on INDERAL® LA stayed on INDERAL LA.[1]*

Surprising? Not really.

Because most patients on INDERAL LA (propranolol HCl) don't even know it's working.

A recent double-blind, placebo-controlled, crossover study in 138 hypertensive patients[2] revealed that INDERAL LA has a side effects profile unsurpassed by atenolol or metoprolol — which shows how well-tolerated once-daily INDERAL LA can be.

Sole therapy or concomitant therapy? Fifty-nine percent of the time, INDERAL LA stood on its own.

The patients in the nationwide compliance trial were no different from yours. Generally when the antihypertensive regimen is complicated, compliance may become a problem. So, the effectiveness of INDERAL LA as once-daily monotherapy is a big plus. Of the remaining hypertensives in the program, 36% were controlled merely with the addition of a diuretic to INDERAL LA.

For the noncompliant patients in your practice, INDERAL LA may well be the answer.

Almost 20,000 of the patients in the nationwide compliance trial were identified as having been noncompliant with their previous antihypertensive therapy. Their physicians reported that 88% showed improved compliance when placed on once-daily INDERAL LA.

Control, comfort, and compliance

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl) LONG ACTING CAPSULES

Like conventional INDERAL Tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree, and bronchial asthma.

*After a 30-day trial with INDERAL LA, physicians reported that 90% of the patients would remain on INDERAL LA.

The one you know best keeps looking better

Please see next page for brief summary of prescribing information.

The one you know best keeps looking better

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules

CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol Over a twenty-four (24) hour period blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product INDERAL LA can provide effective beta blockade for a 24-hour period

The mechanism of the antihypertensive effect of INDERAL has not been established Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use Effects on plasma volume appear to be minor and somewhat variable INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential The significance of the membrane action in the treatment of arrhythmias is uncertain

The mechanism of the antimigraine effect of propranolol has not been established Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain

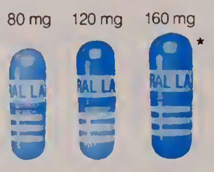
Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient But there are also situations in which sympathetic stimulation is vital For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm.

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl)

LONG ACTING CAPSULES



Propranolol is not significantly dialyzable

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension; it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope INDERAL LA also improves exercise performance The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock; 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible)

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e g , dobutamine or isoproterenol However, such patients may be subject to protracted severe hypotension Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers

DIABETES AND HYPOGLYCEMIA Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes In these patients, it may be more difficult to adjust the dosage of insulin

THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm Propranolol does not distort thyroid function tests

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker In one case, this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. *General* Propranolol should be used with caution in patients with impaired hepatic or renal function INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that INDERAL may interfere with the glaucoma screening test Withdrawal may lead to a return of increased intraocular pressure

Clinical Laboratory Tests Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia vertigo, syncopal attacks, or orthostatic hypotension.

Carcinogenesis, Mutagenesis, Impairment of Fertility Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

Pregnancy Pregnancy Category C INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose

There are no adequate and well-controlled studies in pregnant women INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers. INDERAL is excreted in human milk Caution should be exercised when INDERAL is administered to a nursing woman.

Pediatric Use Safety and effectiveness in children have not been established

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy

Cardiovascular. bradycardia, congestive heart failure, intensification of AV block, hypotension; paresthesia of hands, thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type

Central Nervous System lightheadedness, mental depression manifested by insomnia, lassitude, weakness, fatigue, reversible mental depression progressing to catatonia, visual disturbances, hallucinations, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics

Gastrointestinal: nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis

Allergic. pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress

Respiratory: bronchospasm

Hematologic. agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported

Miscellaneous. alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained INDERAL LA should not be considered a simple mg for mg substitute for INDERAL INDERAL LA has different kinetics and produces lower blood levels Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval

HYPERTENSION—*Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic. The dosage may be increased to 120 mg once daily or higher until adequate blood-pressure control is achieved The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage of 640 mg may be required. The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks.

ANGINA PECTORIS—*Dosage must be individualized* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS).

MIGRAINE—*Dosage must be individualized* The initial oral dose is 80 mg INDERAL LA once daily The usual effective dose range is 160-240 mg once daily The dosage may be increased gradually to achieve optimum migraine prophylaxis. If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued It may be advisable to withdraw the drug gradually over a period of several weeks.

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use

*The appearance of these capsules is a registered trademark of Ayerst Laboratories

---

REFERENCES:

1. INDERAL LA National Compliance Evaluation Program Data on file, Ayerst Laboratories
2. Ravid M, Lang R, Jutrin I: The relative antihypertensive potency of propranolol, oxprenolol, atenolol, and metoprolol given once daily *Arch Intern Med* 1985;145 1321-1323

6412/187

Ayerst®

AYERST LABORATORIES
New York, NY 10017

© 1986 Ayerst Laboratories

YOUR ROCHE REPRESENTATIVE WOULD LIKE YOU TO HAVE SOMETHING THAT WILL...

ROCHE ®

... improve patient satisfaction with office visits

... improve patient compliance with your instructions

... reduce follow-up calls to clarify instructions

The new Roche product books

- Offer a supplement to, not a substitute for, patient contact
- Support your specific instructions to the patient
- Provide a long-term reinforcement of your oral counseling

Because you are the primary source of medical information for your patients, we invite you to look over the Roche Product Booklets shown below and ask your Roche representative for a complimentary supply of those applicable to your practice.

You, your medical problem and your treatment with
DALMANE
BACTRIM™
brand of trimethoprim and sulfamethoxazole/Roche
You, your medical problem and your treatment with
LIBRIUM
You, your medical problem and your treatment with
VALIUM
You, your medical problem and your treatment with
LIMBITROL
ROCHE MEDICATION ME EDUCATION

ROCHE
MEDICATION
ME
EDUCATION

Medicines that matter from people who care

COMMITTED TO EXCELLENCE

ROCHE

PRESIDENT'S ACHIEVEMENT AWARD

Presenting the winners of the 1987 Roche President's Achievement Awards

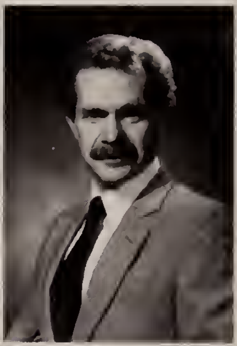
Hoffmann-La Roche is pleased to honor these outstanding sales representatives, chosen for their unparalleled dedication to the health-care field, professionalism and consistent high level of performance. Please join us in congratulating these exceptional individuals.

Gene E. Barone

Alan Berk

Joseph Kessler

Ralph Kressel


Sheldon Maginsky

Thomas J. O'Donnell

Alan Roiphe

Frederick G. Ruder, III

Firoz S. Singaporewala

Turn to the preceding page and find out how your award-winning Roche representative can help both you *and* your patients.

STACKS

NEW YORK STATE
JOURNAL OF MEDICINE

MAY 1987 • Volume 87, Number 5

MEDICAL SOCIETY OF THE STATE OF NEW YORK ORGANIZED 1807

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA
M. D. S.
MAY 14 1987

ACQUIRED IMMUNODEFICIENCY SYNDROME

COMMENTARIES

Acquired immunodeficiency syndrome—1987 251
PASCAL JAMES IMPERATO, MD

AIDS: The eleventh year 255
ERNEST DRUCKER, PHD

AIDS in New York City: Moving ahead on effective public health approaches 257
STEPHEN C. JOSEPH, MD, MPH

The prevention of AIDS 259
VICTOR W. SIDEL, MD

Fear of AIDS 260
GERALD FRIEDLAND, MD

RESEARCH PAPERS

Demography of HIV infections among civilian applicants for military service in four counties in New York City 262
DONALD S. BURKE, MD; JOHN F. BRUNDAGE, MD; WILLIAM BERNIER, MD; LYTT I. GARDNER, PHD; ROBERT R. REDFIELD, MD; JEFFREY GUNZENHAUSER, MD; JAMES VOSKOVITCH, MD; JOHN R. HERBOLD, DVM

Local policy responses to the AIDS epidemic: New York and San Francisco 264
PETER S. ARNO, PHD, ROBERT G. HUGHES, PHD

The effect of group education on improving attitudes about AIDS risk reduction 272
RONALD O. VALDISERRI, MD; DAVID W. LYTER, MD; LAWRENCE A. KINGSLEY, DRPH; LAURA C. LEVITON, PHD; JANET W. SCHOFIELD, PHD; JAMES HUGGINS, MSW; MONTO HO, MD; CHARLES R. RINALDO, PHD

Hospital workers and AIDS: Effect of in-service education on knowledge and perceived risks and stresses 278
LYDIA O'DONNELL, EDD, CARL R. O'DONNELL, SCD, MPH

Cyclooxygenase inhibition and improved oxygenation in patients with pulmonary complications of AIDS 280
STEVEN H. FOSTER, MD; ROBERT C. GARRETT, MD; HENRY M. THOMAS, III, MD

REVIEW ARTICLES

Intravenous drug use and the heterosexual transmission of the human immunodeficiency virus: Current trends in New York City 283
DON C. DES JARLAIS, PHD; ERIC WISH, PHD; SAMUEL R. FRIEDMAN, PHD; RAND STONEBURNER, MD, MPH; STANLEY R. YANCOVITZ, MD; DONNA MILDVAN, MD; WAFAA EL-SADR, MD; ELIZABETH BRADY, MA; MARY CUADRADO, MA

AIDS in Africa: Evidence for heterosexual transmission of the human immunodeficiency virus 286
THOMAS C. QUINN, MD

AIDS in adolescents: A rationale for concern 290
KAREN HEIN, MD

Education and contact notification for AIDS prevention 296
DEAN F. ECHENBERG, MD, PHD

SPECIAL ARTICLE

AIDS care in New York City: The comprehensive care alternative 298
JACK A. DEHOVITZ, MD, MPH, VIRGINIA PELLEGRINO, MSC, MPH

CASE REPORTS

Coagulase-negative staphylococcal sepsis in drug-dependent newborns who may be HIV-positive 301
FRANCISCO MEDINA MEJIA, MD, STEPHEN R. KANDALL, MD

An unusual presentation of lymphoma in a homosexual man 302
SANFORD DUBNER, MD, KEITH S. HELLER, MD

Oral candidiasis and AIDS 303
JEFFREY S. GELWAN, MD; BURTON M. GOLD, MD; HENRY J. SHIH, MD; CRESCENS PELLECCHIA, MD

Herpes simplex pericarditis in AIDS 304
ROBIN S. FREEDBERG, MD; AARON J. GINDEA, MD; DOUGLAS T. DIETERICH, MD; JEFFREY B. GREENE, MD

Fatal hemoptysis in a patient with AIDS-related complex and pulmonary aspergilloma 306
GERARD T. LOMBARDO, MD; NANARAO ANANDARAO, MD; CHING-SHEN LIN, MD; ANDRE ABBATE, MD; WILLIAM H. BECKER, MD

NEWS BRIEFS 309

BOOKS RECEIVED 313

OBITUARIES 314

GUIDELINES FOR AUTHORS 318

MEDICAL MEETINGS AND LECTURES 8A

Motrin® 800 mg TABLETS
ibuprofen

Extra strength
Convenience
Economy

Upjohn
A Century of Caring
1886·1986

©1986 The Upjohn Company

J-6138 January 1986

Give your angina patients what they're missing...

CARDIZEM®: FEW SIDE EFFECTS

diltiazem HCl/Marion

90 mg GREATER DOSAGE FLEXIBILITY

NEW 120 mg

- ***Antianginal action includes dilatation of coronary arteries, a decrease in vascular resistance/afterload, and a reduction in heart rate***
- ***Proven efficacy when used alone in angina[1]***
- ***Compatible with other antianginals[2,3]****
- ***A safe choice for angina patients with coexisting hypertension, asthma, COPD, or PVD[4,5]***

***See Warnings and Precautions.**

Please see brief summary of prescribing information on the next page.

CARDIZEM® diltiazem HCl/Marion FEW SIDE EFFECTS IN ANTIANGINAL THERAPY

60 mg tid or qid

Brief Summary
Professional Use Information

CARDIZEM®
(diltiazem HCl) 30 mg and 60 mg Tablets

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. ***Cardiac Conduction.*** *CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1,243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.*
2. ***Congestive Heart Failure.*** *Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt).*
   Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. ***Hypotension.*** *Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.*
4. ***Acute Hepatic Injury.*** *In rare instances, significant elevations in enzymes such as alkaline phosphatase, CPK, LDH, SGOT, SGPT, and other symptoms consistent with acute hepatic injury have been noted. These reactions have been reversible upon discontinuation of drug therapy. The relationship to CARDIZEM is uncertain in most cases, but probable in some. (See PRECAUTIONS.)*

PRECAUTIONS

General. *CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.*

Drug Interaction. *Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)*

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. *A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.*

Pregnancy. *Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.*

There are no well-controlled studies in pregnant women, therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. *Diltiazem is excreted in human milk. One report suggests that concentrations in breast milk may approximate serum levels. If use of CARDIZEM is deemed essential, an alternative method of infant feeding should be instituted.*

Pediatric Use. *Safety and effectiveness in children have not been established.*

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences as well as their frequency of presentation are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%). In addition, the following events were reported infrequently (less than 1%):

Cardiovascular:	*Angina, arrhythmia, AV block (first degree), AV block (second or third degree — see conduction warning), bradycardia, congestive heart failure, flushing, hypotension, palpitations, syncope.*
Nervous System:	*Amnesia, gait abnormality, hallucinations, insomnia, nervousness, paresthesia, personality change, somnolence, tinnitus, tremor.*
Gastrointestinal:	*Anorexia, constipation, diarrhea, dysgeusia, dyspepsia, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH (see hepatic warnings), vomiting, weight increase.*
Dermatologic:	*Petechiae, pruritus, photosensitivity, urticaria.*
Other:	*Amblyopia, dyspnea, epistaxis, eye irritation, hyperglycemia, nasal congestion, nocturia, osteoarticular pain, polyuria, sexual difficulties.*

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: alopecia, gingival hyperplasia, erythema multiforme, and leukopenia. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established. ***Issued 7/86***

See complete Professional Use Information before prescribing.

***References:* 1.** *Pepine CJ, Feldman RL, Hill JA, et al: Clinical outcome after treatment of rest angina with calcium blockers: Comparative experience during the initial year of therapy with diltiazem, nifedipine, and verapamil. Am Heart J 1983; 106(6):1341-1347* **2.** *Shapiro W: Calcium channel blockers: Actions on the heart and uses in ischemic heart disease. Consultant 1984;24(Dec):150-159* **3.** *Johnston DL, Lesoway R, Humen DP, et al: Clinical and hemodynamic evaluation of propranolol in combination with verapamil, nifedipine and diltiazem in exertional angina pectoris: A placebo-controlled, double-blind, randomized, crossover study. Am J Cardiol 1985;55:680-687.* **4.** *Cohn PF, Braunwald E: Chronic ischemic heart disease, in Braunwald E (ed): Heart Disease: A Textbook of Cardiovascular Medicine, ed 2. Philadelphia, WB Saunders Co, 1984, chap 39* **5.** *Schroeder JS: Calcium and beta blockers in ischemic heart disease: When to use which. Mod Med 1982;50(Sept):94-116.*

Another patient benefit product from
PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC.
KANSAS CITY, MO 64137

0246M6

NEW YORK STATE
JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
ELIZA H. CALDWELL, MD
JOSEPH F. MURATORE, MD
JOHN T. PRIOR, MD
GITA S. SINGH*
STANFORD WESSLER, MD

**Medical student*

Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Consulting Editor and Book Review Editor	RICHARD B. BIRRER, MD
Consulting Editor	NAOMI R. BLUESTONE, MD, MPH
Consulting Editor	CARL POCHEDLY, MD
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Managing Editor	CAROL L. MOORE
Advertising Director	ROBERT MILLER
Consulting Medical Writer	VICKI GLASER
Editorial Assistant	MILDRED J. ARFMANN
Secretary	ELIZABETH J. SOMERS
Librarian	ELLA ABNEY
Assistant Librarian	ELEANOR BURNS

ASSOCIATE EDITORIAL BOARD 1987

MICHAEL E. BERLOW, MD
RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MYLES S. GOMBERT, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
JAMES M. MORRISSEY, ESQ
STEPHEN NORDLICHT, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. United Media Associates, Inc, 16 Bruce Park Ave, Greenwich, CT 06830. Telephone: (203) 661-9702.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

SAMUEL M. GELFAND, MD,	*President*
JOHN A. FINKBEINER, MD,	*Past-President*
CHARLES D. SHERMAN, JR, MD,	*President-Elect*
JOSEPH F. SHANAPHY, MD,	*Vice-President*
JOHN H. CARTER, MD,	*Secretary*
GEORGE LIM, MD,	*Assistant Secretary*
MORTON KURTZ, MD,	*Treasurer*
ROBERT A. MAYERS, MD,	*Assistant Treasurer*
CHARLES N. ASWAD, MD,	*Speaker*
SEYMOUR R. STALL, MD,	*Vice-Speaker*

Councilors

Term Expires 1988	
JAMES H. COSGRIFF, JR, MD,	Erie
RICHARD A. HUGHES, MD,	Warren
ANTONIO F. LASORTE, MD,	Broome
SIDNEY MISHKIN, MD,	Nassau
Term Expires 1989	
ROBERT E. FEAR, MD,	Suffolk
STANLEY L. GROSSMAN, MD,	Orange
THOMAS D. PEMRICK, MD,	Rensselaer
RALPH E. SCHLOSSMAN, MD,	Queens
Term Expires 1990	
DAVID M. BENFORD, MD,	New York
DUANE M. CADY, MD,	Onondaga
WILLIAM A. DOLAN, MD,	Monroe
ROBERT E. GORDON, MD,	Kings
Resident Councilor (representing the resident physician membership)	
KATHLEEN E. SQUIRES, MD,	New York
Student Councilor (representing the medical student membership)	
MICHAEL PACIOREK,	Onondaga

Trustees

RICHARD D. EBERLE, MD, (*Chairman*)	Onondaga
EDGAR P. BERRY, MD,	New York
JAMES M. FLANAGAN, MD,	Wayne
ALLISON B. LANDOLT, MD,	Westchester
DANIEL F. O'KEEFFE, MD,	Warren
BERNARD J. PISANI, MD,	New York
VICTOR J. TOFANY, MD,	Monroe

Executive Vice-President	DONALD F. FOY
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Vice-President Emeritus	GEORGE J. LAWRENCE, JR, MD
Director, Division of Policy Coordination	IRMA A. ERICKSON
Director, Division of Scientific Publications	PASCAL JAMES IMPERATO, MD
Director, Division of Communications	EDWARD A. HYNES
Director, Division of Computer Services	IVAN H. NAJMAN
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Governmental Affairs	GERARD L. CONWAY, ESQ
General Counsel	DONALD R. MOY, JD
Director, Division of Management and Meeting Services	ROBERT MILLER
Director, Division of Membership Support Services	EUNICE M. SKELLY
Director, Division of Scientific and Educational Activities	PETER B. FARNSWORTH, MD
Director, Division of Socio-Medical Economics	MARIO V. MENGHINI
Medical Director, Committee for Physicians' Health	BARTON A. HARRIS, MD
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York. Copyright © 1987, Medical Society of the State of New York. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.

There's never been a better time for her...

and PREMARIN®

Proven benefits beyond relief of vasomotor symptoms

No other estrogen proven effective for osteoporosis

Only conjugated estrogens tablets have established efficacy in both osteoporosis[1] and vasomotor symptoms* at 0.625 mg/day. No other estrogen, oral or transdermal, has established clinical evidence or minimum effective dose in both indications.

No estrogen proven safer

PREMARIN is the most extensively tested estrogen, with an unsurpassed record of long-term safety.
And clinical evidence shows a significantly reduced risk of endometrial hyperplasia when cycled with a progestin.[2]

PREMARIN®
(conjugated estrogens tablets)

Most trusted for more reasons

*PREMARIN is indicated for moderate-to-severe vasomotor symptoms.

Please see following page for brief summary
of prescribing information.

For moderate-to-severe vasomotor symptoms and for osteoporosis

PREMARIN®
(conjugated estrogens tablets)

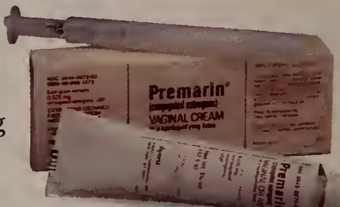
0.3 mg 0.625 mg 0.9 mg 1.25 mg 2.5 mg

The appearance of these tablets is a trademark of Ayerst Laboratories.

For atrophic vaginitis

PREMARIN®
(conjugated estrogens)

Vaginal
Cream
0.625 mg/g



BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION AND PATIENT INFORMATION, SEE PACKAGE CIRCULARS.)*

PREMARIN® Brand of conjugated estrogens tablets, USP
PREMARIN® Brand of conjugated estrogens Vaginal Cream, in a nonliquefying base

> 1 ESTROGENS HAVE BEEN REPORTED TO INCREASE THE RISK OF ENDOMETRIAL CARCINOMA
> Three independent, case-controlled studies have reported an increased risk of endometrial cancer in postmenopausal women exposed to exogenous estrogens for more than one year. This risk was independent of the other known risk factors for endometrial cancer. These studies are further supported by the finding that incidence rates of endometrial cancer have increased sharply since 1969 in eight different areas of the United States with population-based cancer reporting systems, an increase which may be related to the rapidly expanding use of estrogens during the last decade. The three case-controlled studies reported that the risk of endometrial cancer in estrogen users was about 4 5 to 13 9 times greater than in nonusers. The risk appears to depend on both duration of treatment and on estrogen dose In view of these findings, when estrogens are used for the treatment of menopausal symptoms, the lowest dose that will control symptoms should be utilized and medication should be discontinued as soon as possible When prolonged treatment is medically indicated, the patient should be reassessed on at least a semi-annual basis to determine the need for continued therapy Although the evidence must be considered preliminary, one study suggests that cyclic administration of low doses of estrogen may carry less risk than continuous administration; it therefore appears prudent to utilize such a regimen Close clinical surveillance of all women taking estrogens is important. In all cases of undiagnosed persistent or recurring abnormal vaginal bleeding, adequate diagnostic measures should be undertaken to rule out malignancy There is no evidence at present that "natural" estrogens are more or less hazardous than "synthetic" estrogens at equi-estrogenic doses
> 2 ESTROGENS SHOULD NOT BE USED DURING PREGNANCY
> The use of female sex hormones, both estrogens and progestogens, during early pregnancy may seriously damage the offspring It has been shown that females exposed in utero to diethylstilbestrol, a nonsteroidal estrogen, have an increased risk of developing, in later life, a form of vaginal or cervical cancer that is ordinarily extremely rare This risk has been estimated as not greater than 4 per 1,000 exposures. Furthermore, a high percentage of such exposed women (from 30% to 90%) have been found to have vaginal adenosis, epithelial changes of the vagina and cervix Although these changes are histologically benign, it is not known whether they are precursors of malignancy Although similar data are not available with the use of other estrogens, it cannot be presumed they would not induce similar changes Several reports suggest an association between intrauterine exposure to female sex hormones and congenital anomalies, including congenital heart defects and limb-reduction defects. One case-controlled study estimated a 4.7-fold increased risk of limb-reduction defects in infants exposed in utero to sex hormones (oral contraceptives, hormone withdrawal tests for pregnancy, or attempted treatment for threatened abortion). Some of these exposures were very short and involved only a few days of treatment The data suggest that the risk of limb-reduction defects in exposed fetuses is somewhat less than 1 per 1,000. In the past, female sex hormones have been used during pregnancy in an attempt to treat threatened or habitual abortion. There is considerable evidence that estrogens are ineffective for these indications, and there is no evidence from well-controlled studies that progestogens are effective for these uses If PREMARIN is used during pregnancy, or if the patient becomes pregnant while taking this drug, she should be apprised of the potential risks to the fetus, and the advisability of pregnancy continuation

DESCRIPTION: PREMARIN (conjugated estrogens, USP) contains a mixture of estrogens, obtained exclusively from natural sources, blended to represent the average composition of material derived from pregnant mares' urine It contains estrone, equilin, and 17α-dihydroequilin, together with smaller amounts of 17α-estradiol, equilenin, and 17α-dihydroequilenin as salts of their sulfate esters. Tablets are available in 0.3 mg, 0 625 mg, 0.9 mg, 1 25 mg, and 2.5 mg strengths of conjugated estrogens Cream is available as 0 625 mg conjugated estrogens per gram

INDICATIONS AND USAGE: PREMARIN (conjugated estrogens tablets, USP) Moderate-to-severe *vasomotor* symptoms associated with the menopause (There is no evidence that estrogens are effective for nervous symptoms or depression without associated vasomotor symptoms and they should not be used to treat such conditions) Osteoporosis (abnormally low bone mass). Atrophic vaginitis. Kraurosis vulvae Female castration.

PREMARIN (conjugated estrogens) Vaginal Cream is indicated in the treatment of atrophic vaginitis and kraurosis vulvae

PREMARIN HAS NOT BEEN SHOWN TO BE EFFECTIVE FOR ANY PURPOSE DURING PREGNANCY AND ITS USE MAY CAUSE SEVERE HARM TO THE FETUS (SEE BOXED WARNING)

Concomitant Progestin Use: The lowest effective dose appropriate for the specific indication should be utilized Studies of the addition of a progestin for 7 or more days of a cycle of estrogen administration have reported a lowered incidence of endometrial hyperplasia Morphological and biochemical studies of the endometrium suggest that 10 to 13 days of progestin are needed to provide maximal maturation of the endometrium and to eliminate any hyperplastic changes. Whether this will provide protection from endometrial carcinoma has not been clearly established There are possible additional risks which may be associated with the inclusion of progestin in estrogen replacement regimens (See PRECAUTIONS) The choice of progestin and dosage may be important; product labeling should be reviewed to minimize possible adverse effects

CONTRAINDICATIONS: Estrogens should not be used in women (or men) with any of the following conditions: 1 Known or suspected cancer of the breast except in appropriately selected patients being treated for metastatic disease 2 Known or suspected estrogen-dependent neoplasia 3 Known or suspected pregnancy (see Boxed Warning). 4 Undiagnosed abnormal genital bleeding 5 Active thrombophlebitis or thromboembolic disorders 6 A past history of thrombophlebitis, thrombosis, or thromboembolic disorders associated with previous estrogen use (except when used in treatment of breast or prostatic malignancy)

WARNINGS: Estrogens have been reported to increase the risk of endometrial carcinoma (see Boxed Warning) However, a recent large, case-controlled study indicated no increase in risk of breast cancer in postmenopausal women. A recent study has reported a 2- to 3-fold increase in the risk of surgically confirmed gallbladder disease in women receiving postmenopausal estrogens

Adverse effects of oral contraceptives may be expected at the larger doses of estrogen used to treat prostatic or breast cancer or postpartum breast engorgement, it has been shown that there is an increased risk of thrombosis in men receiving estrogens for prostatic cancer and women for postpartum breast engorgement Users of oral contraceptives have an increased risk of diseases, such as thrombophlebitis, pulmonary embolism, stroke, and myocardial infarction Cases of retinal thrombosis, mesenteric thrombosis, and optic neuritis have been reported in oral contraceptive users An increased risk of postsurgery thromboembolic complications has also been reported in users of oral contraceptives If feasible, estrogen should be discontinued at least 4 weeks before surgery of the type associated with an increased risk of thromboembolism, or during periods of prolonged immobilization Estrogens should not be used in persons with active thrombophlebitis, thromboembolic disorders, or in persons with a history of such disorders in association with estrogen use They should be used with caution in patients with cerebral vascular or coronary artery disease Large doses (5 mg conjugated estrogens per day), comparable to those used to treat cancer of the prostate and breast, have been shown to increase the risk of nonfatal myocardial infarction, pulmonary embolism, and thrombophlebitis When doses of this size are used, any of the thromboembolic and thrombotic adverse effects should be considered a clear risk

Benign hepatic adenomas should be considered in estrogen users having abdominal pain and tenderness, abdominal mass, or hypovolemic shock Hepatocellular carcinoma has been reported in women taking estrogen-containing oral contraceptives. Increased blood pressure may occur with use of estrogens in the menopause and blood pressure should be monitored with estrogen use. A worsening of glucose tolerance has been observed in patients on estrogen-containing oral contraceptives For this reason, diabetic patients should be carefully observed Estrogens may lead to severe hypercalcemia in patients with breast cancer and bone metastases

PRECAUTIONS: Physical examination and a complete medical and family history should be taken prior to the initiation of any estrogen therapy with special reference to blood pressure, breasts, abdomen, and pelvic organs, and should include a Papanicolaou smear As a general rule, estrogen should not be prescribed for longer than one year without another physical examination being performed Conditions influenced by fluid retention, such as asthma, epilepsy, migraine, and cardiac or renal dysfunction, require careful observation Certain patients may develop manifestations of excessive estrogenic stimulation, such as abnormal or excessive uterine bleeding, mastodynia, etc Prolonged administration of unopposed estrogen therapy has been reported to increase the risk of endometrial hyperplasia in some patients. Oral contraceptives appear to be associated with an increased incidence of mental depression. Patients with a history of depression should be carefully observed Pre-existing uterine leiomyomata may increase in size during estrogen use The pathologist should be advised of estrogen therapy when relevant specimens are submitted. If jaundice develops in any patient receiving estrogen, the medication should be discontinued while the cause is investigated Estrogens should be used with care in patients with impaired liver function, renal insufficiency, metabolic bone diseases associated with hypercalcemia, or in young patients in whom bone growth is not yet complete If concomitant progestin therapy is used, potential risks may include adverse effects on carbohydrate and lipid metabolism

The following changes may be expected with larger doses of estrogen:

a Increased sulfobromophthalein retention.

b. Increased prothrombin and factors VII, VIII, IX, and X; decreased antithrombin 3, increased norepinephrine-induced platelet aggregability

c. Increased thyroid binding globulin (TBG) leading to increased circulating total thyroid hormone, as measured by PBI, T_4 by column, or T_4 by radioimmunoassay Free T_3 resin uptake is decreased, reflecting the elevated TBG, free T_4 concentration is unaltered.

d Impaired glucose tolerance.

e. Decreased pregnanediol excretion.

f. Reduced response to metyrapone test

g Reduced serum folate concentration.

h. Increased serum triglyceride and phospholipid concentration

As a general principle, the administration of any drug to nursing mothers should be done only when clearly necessary since many drugs are excreted in human milk

Long-term, continuous administration of natural and synthetic estrogens in certain animal species increases the frequency of carcinomas of the breast, cervix, vagina, and liver. However, in a recent, large case-controlled study of postmenopausal women there was no increase in risk of breast cancer with use of conjugated estrogens

ADVERSE REACTIONS: The following have been reported with estrogenic therapy, including oral contraceptives: breakthrough bleeding, spotting, change in menstrual flow, dysmenorrhea; premenstrual-like syndrome; amenorrhea during and after treatment; increase in size of uterine fibromyomata, vaginal candidiasis, change in cervical erosion and in degree of cervical secretion, cystitis-like syndrome, tenderness, enlargement, secretion (of breasts); nausea, vomiting, abdominal cramps, bloating, cholestatic jaundice, chloasma or melasma which may persist when drug is discontinued, erythema multiforme; erythema nodosum, hemorrhagic eruption, loss of scalp hair, hirsutism, steepening of corneal curvature, intolerance to contact lenses, headache, migraine, dizziness, mental depression, chorea, increase or decrease in weight, reduced carbohydrate tolerance, aggravation of porphyria, edema; changes in libido

ACUTE OVERDOSAGE: May cause nausea, and withdrawal bleeding may occur in females

DOSAGE AND ADMINISTRATION:

PREMARIN® Brand of conjugated estrogens tablets, USP

1. *Given cyclically for short-term use only* For treatment of moderate-to-severe *vasomotor* symptoms, atrophic vaginitis, or kraurosis vulvae associated with the menopause (0.3 mg to 1 25 mg or more daily). The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible Administration should be cyclic (eg, three weeks on and one week off) Attempts to discontinue or taper medication should be made at three- to six-month intervals

2 *Given cyclically* Osteoporosis Female castration. Osteoporosis —0 625 mg daily Administration should be cyclic (eg, three weeks on and one week off). Female castration—1 25 mg daily, cyclically Adjust upward or downward according to response of the patient For maintenance, adjust dosage to lowest level that will provide effective control.

Patients with an intact uterus should be monitored for signs of endometrial cancer and appropriate measures taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding

PREMARIN® Brand of conjugated estrogens Vaginal Cream

Given cyclically for short-term use only For treatment of atrophic vaginitis or kraurosis vulvae

The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible.

Administration should be cyclic (eg, three weeks on and one week off).

Attempts to discontinue or taper medication should be made at three- to six-month intervals

Usual dosage range: 2 g to 4 g daily, intravaginally, depending on the severity of the condition

Treated patients with an intact uterus should be monitored closely for signs of endometrial cancer and appropriate diagnostic measures should be taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding

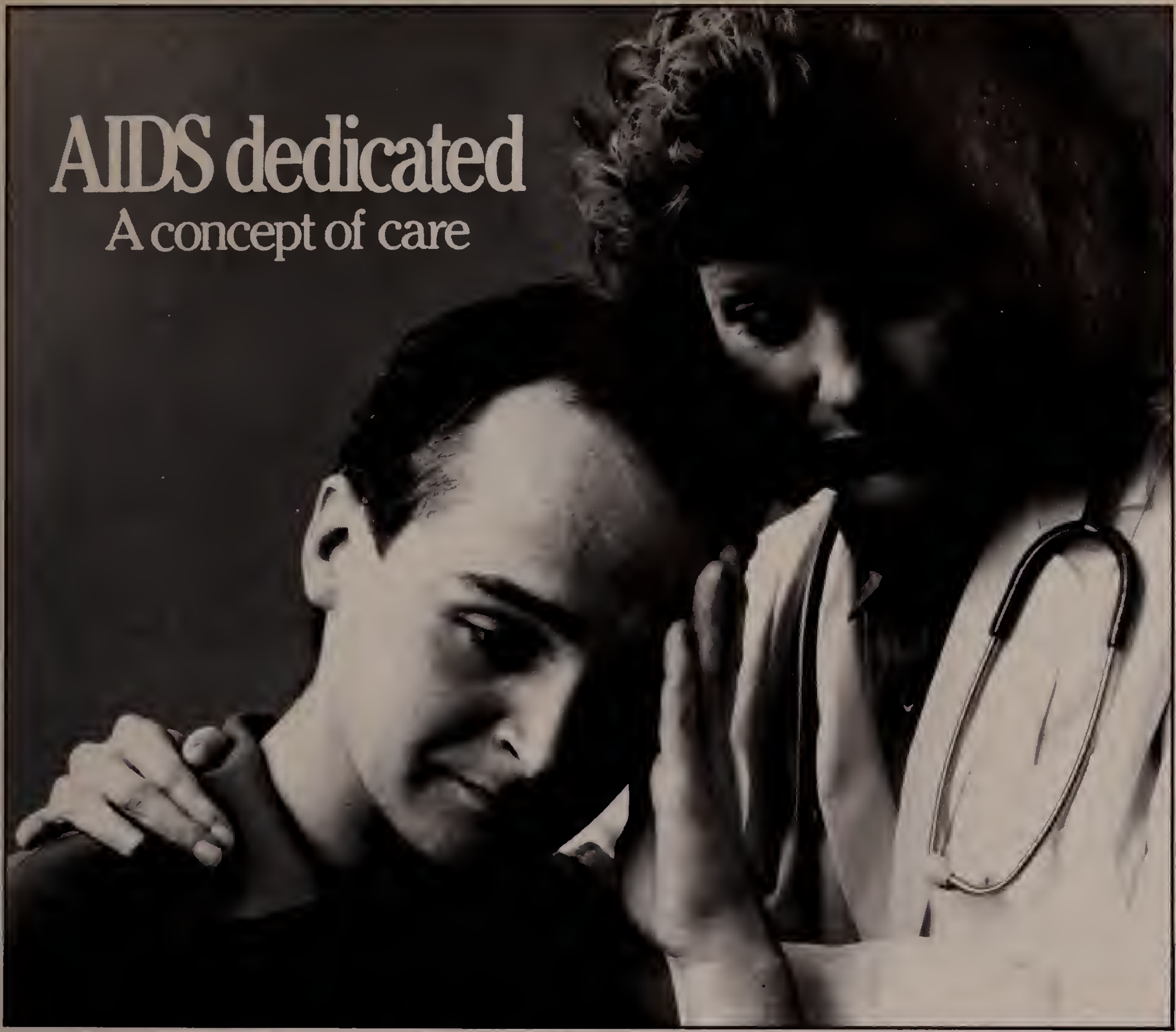
References:

1. Lindsay R, Hart DM, Clark DM. The minimum effective dose of estrogen for prevention of postmenopausal bone loss *Obstet Gynecol* 1984,63 759-763 **2.** Studd JWW, Thom MH, Paterson MEL, et al The prevention and treatment of endometrial pathology in postmenopausal women receiving exogenous estrogens, in Pasetto N, Paoletti R, Ambrus JL (eds): *The Menopause and Postmenopause* Lancaster, England. MTP Press Ltd, 1980, chap 13.

© 1987 Ayerst Laboratories
All rights reserved

6437/587

Ayerst® AYERST LABORATORIES
New York, NY 10017



Photography by Ken Nahoum

AIDS is a complex disease, assaulting the psyche as well as the body. The Spellman Center for the Treatment of Persons With AIDS treats both. A comprehensive medical facility, Spellman is devoted to the care of those suffering from Acquired Immune Deficiency Syndrome.

Respect for individual dignity guides the Spellman program. Staffed by trained personnel dedicated to working with AIDS patients, the Center includes family and friends in the management of the patient's health crisis. Spellman also operates a 24-hour information hotline at 1-800-433-AIDS.

For criteria on admission and referrals, please contact the Medical Director, at **1-212-307-0735**. Spellman is a division of St. Clare's Hospital and Health Center, designated the first AIDS treatment center by New York State.

The Spellman Center
for the Treatment of Persons With AIDS
St. Clare's Hospital and Health Center

415 W 51st St
New York, NY 10019

An Affiliate of New York Medical College

An AZT Clinical Site

Medical experts,
providers of care

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices unless the information is submitted at least three months prior to the event.*

JUNE 1987

AROUND THE STATE

BUFFALO

June 7–20. **Ninth Summer Program in Methods of Immunologic Research and Diagnosis.** The Ernest Witebsky Center for Immunology, State University of New York Health Science Center at Buffalo. Contact: James F. Mohn, MD, Director, The Ernest Witebsky Center for Immunology, 210 Sherman Hall, Buffalo, NY 14214. Tel: (716) 831-2848.

MANHATTAN

June 1–3. **Practical Obstetrical Ultrasound.** 18 Cat 1 Credits. *June 11–14.* **Emergency Medicine 1987.** 27½ Cat 1 Credits. *June 22–25.* **Selected Topics in Pediatrics.** 22 Cat 1 Credits. NYU Medical Center, New York City. Contact: NYU Medical Center Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

June 4–6. **1987 Sigmund Schutz Professorship.** *June 13.* **Workshop: Diagnosis and Management of Patients with Oculo-Plastic Problems.** Contact: Martha Klapp, Manhattan Eye, Ear & Throat Hospital, 210 East 64th St, New York, NY 10021. Tel: (212) 605-3762.

June 8–12. **Update Your Medicine 1987.** The New York Hospital-Cornell Medical Center. Contact: Office of CME, 1300 York Ave at 69th St, New York, NY 10021. Tel: (212) 472-6119.

June 12. **Pharmacological and Toxicological Approach to Critical Emergency Department Problems.** New York City Poison Control Center. Contact: Susan Callaghan-Montella, RN, Poison Control Project, Bellevue Hospital Center, Emergency Dept, 27th St and First Ave, New York, NY 10016. Tel: (212) 561-6561.

June 12–13. **Neurological Applications of Implanted Drug Pumps.** Sheraton Centre. *June 22–24.* **Biomedical Ethics: An Anglo-American Dialogue.** Omni Park Central Hotel. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd St, New York, NY 10021.

June 15–19. **Critical Care Board Review.** 30 Cat 1 Credits. Grand Hyatt Hotel. Info: American College of Physicians, Registration Services, 4200 Pine St, Philadelphia, PA 19105. Tel: (800) 523-1546.

SYRACUSE

June 2. **Current Concepts in Obstetrics, Endocrinology and Infertility.** 6 Cat 1 Credits. Sheraton University Inn, Syracuse. Contact: Program Coordinator, Continuing Education, SUNY Health Science Center, 750 East Adams St, Syracuse, NY 13210. Tel: (315) 473-4304.

AROUND THE NATION

CALIFORNIA

June 4–5. **16th Annual Neurosurgical Postgraduate Course and the 10th Annual Neurosurgical Nurses' Conference.** 15 Cat 1 Credits. Hotel Meridien, San Francisco. Contact: University of California, Extended Programs in Medical Education, Room U-569, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

June 5–7. **16th Annual Scientific Assembly of the American College of Emergency Physicians.** Newporter Resort, Newport Beach. Contact: CAL/ACEP, 505 N Sepulveda Blvd, #12–14, Manhattan Beach, CA 90266. Tel: (213) 475-4039.

June 7–10. **Critical Care Examination Review Course.** 30 Cat 1 Credits. Hotel Del Coronado, San Diego. Contact: American College of Chest Physicians, PO Box 93826, Chicago, IL 60673. Tel: (312) 698-2200.

June 11–13. **Gynecologic Oncology and Breast Disease.** Fairmont Hotel, San Francisco. The American College of

(*continued on p 18A*)

LIBRIUM®
chlordiazepoxide HCl/Roche Ⓒ
5-mg, 10-mg, 25-mg capsules
Before prescribing, please consult complete product information, a summary of which follows:
Indications: Management of anxiety disorders; short-term relief of anxiety symptoms, acute alcohol withdrawal symptoms, preoperative apprehension and anxiety. Usually not required for anxiety or tension associated with stress of everyday life. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended.
Contraindications: Known hypersensitivity to the drug.
Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage. Withdrawal symptoms (including convulsions) reported after abrupt cessation of extended use of excessive doses are similar to those seen with barbiturates. Milder symptoms reported infrequently when continuous therapy is abruptly ended. Avoid abrupt discontinuation; gradually taper dosage.

Usage in Pregnancy: **Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically. Due to isolated reports of exacerbation, use with caution in patients with porphyria.
Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.
Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral—Adults:* Mild and moderate anxiety disorders and symptoms, 5 or 10 mg *t.i.d.* or *q.i.d.*; severe states, 20 or 25 mg *t.i.d.* or *q.i.d.* *Geriatric patients*: 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)
Supplied: Librium® (chlordiazepoxide HCl/Roche) *Capsules,* 5 mg, 10 mg and 25 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in boxes of 4 reverse-numbered cards of 25, and in boxes containing 10 strips of 10. Libritabs® (chlordiazepoxide/Roche) *Tablets,* 5 mg and 10 mg—bottles of 100 and 500; 25 mg—bottles of 100. With respect to clinical activity, capsules and tablets are indistinguishable.

ROCHE®
Roche Products Inc.
Manati, Puerto Rico 00701

Do not substitute

Librium®

brand of

chlordiazepoxide HCl/Roche ⒸIV

Nobody does it better.

ROCHE®

Copyright © 1986 by Roche Products Inc. All rights reserved.

Please see adjacent page for summary of product information.

Classified Advertising

PHYSICIANS WANTED

UNIVERSITY PHYSICIAN, STUDENT HEALTH SERVICES. Princeton University Health Services, a comprehensive program serving a student population of 8,000 seeks a full-time physician effective July 1, 1987. Responsible for providing full spectrum of primary care to students. Requires experience in adolescent and young adult medicine with strong clinical skills and special interest in Athletic Medicine. Some evenings and weekend responsibilities including athletic team coverage. Board certified or eligible in internal medicine, pediatrics or family medicine. Salary and benefits competitive. Send resume with references to: L. A. Pyle, Jr., M.D., Director, McCosh Health Center, Princeton University, Princeton, New Jersey 08544. Equal Opportunity/Affirmative Action Employer.

PHYSICIANS needed for 75 children's private residential camps July and/or August. Contact: Association of Independent Camps (JM), 43 West 23rd Street—Sixth Floor, New York, N.Y. 10010 (212) 645-6620.

DO YOU WANT TO SHARE A THRIVING DERMATOLOGY PRACTICE: Do you enjoy year around outdoor recreation? You should contact us—a progressive multi-specialty group of 68 physicians in a pleasant, growing community. We offer a liberal financial package and outstanding benefits. Send curriculum vitae and references to: Michael T. Anderson, M.D., Willmar Medical Center, 101 Willmar Ave. SW, Willmar, MN 56201.

EMERGENCY MEDICINE POSITIONS—Full/part time emergency medicine physicians sought by multi state professional association for openings in metropolitan NY, PA, MD, DC, FL, New England and throughout U.S. Contact or send CV to Liberty Healthcare Corporation, 399 Market Street, Suite 400, Philadelphia, PA 19106, (215) 592-7400 or outside Pa (800) 331-7122.

ARIZONA-BASED PHYSICIAN RECRUITMENT firm has quality opportunities coast to coast. Available positions in most primary care and surgical specialties to include OB/GYN, Orthopedics, ER, and ENT. "Quality Physicians for Quality Clients since 1972." Call 602-990-8080; or send CV to: Mitchell & Associates, Inc., PO Box 1804, Scottsdale, AZ 85252.

INTERNIST: Immediate opening. NY State license, board certified or eligible for same. 500 bed JCAH Accredited Hospital serving Southwestern NY State. New challenging programs. Opportunities for innovation, professional growth and promotion. Salary range $61,176 to $75,932. Generous NY State fringe benefits. Additional earnings available through extra service program. Housing available. Located 30 miles from Buffalo, two hours from Toronto. Semi-rural setting. Many fine schools, colleges, recreational and cultural facilities nearby. Send curriculum vitae and two recent professional reference letters to: Dr. Jean Jackson, Director of Clinical Services, Gowanda Psychiatric Center, Helmuth, NY 14079. The NY State Office of Mental Health is an AA/EOE Employer.

PHYSICIANS WANTED—CONT'D

PEDIATRICIAN, Board eligible/certified, to work in a community health center in the Finger Lakes Region of New York State. The Center is an established program with a record of excellence. Associated with the University of Rochester, this can be an exciting opportunity. Send CV to Constantino Fernandez, MD, Acting Medical Director, Jordan Health Center, 82 Holland Street, P.O. Box 876, Rochester, NY 14603, EOE, M/F.

INTERNIST OR G.P. WANTED for partnership in old established Queens Practice. Yugoslavian preferred, but not a must. (718) 728-1244.

INTERNIST with an interest in Gastroenterology needed to join a Cardiologist/Internist in rural Louisiana town starting July, 1987. Attractive first year salary, benefits and early partnership. If interested, send C.V. to: Manzoor H. Qazi, M.D., 1101A Port Arthur Terrace, Leesville, LA 71446.

PEDIATRICIAN... Board Certified or Board Eligible for rapidly growing area in Orange County. Excellent opportunity with strong community hospital committed to support and establishment of practice. Cross coverage available. For additional information send CV to: Mary Ann Hamburger, Mary Ann Hamburger Associates, 74 Hudson Avenue, Maplewood, New Jersey 07040.

POSITIONS WANTED

ROUTINE X-RAY DIAGNOSIS: Mature, U.S. trained, Board Certified Radiologist will have time available commencing summer of 1987, to participate in radiographic installation in New York City Metropolitan area. Not interested in large volume, salary commensurate. Private practice coverage would also be suitable. Reply Dept. 441 c/o NYSJM.

PRACTICES AVAILABLE

GENERAL PRACTICE FOR SALE in Woodside, Queens, established for 33 years, located in an apartment house. Physician retiring. (718) 429-6541, 3–5 PM on Mondays, Tuesdays or Fridays.

FLORIDA FAMILY/MEDICAL PRACTICE—N.E. Fort Lauderdale. Physician retiring, established 20 years. High gross, desirable demographic patient profile. Fully equipped including x-ray. Top location, terms (305) 946-2472 evenings. Excellent opportunity to get started, by-pass long years of building.

PRACTICE FOR SALE. Excellent opportunity to join and buy into large active 30 year old I.M. and F.M. practice. Bergen County. Plan early partnership and retirement after introduction. Please send curriculum and needs. Reply Dept. 442 c/o NYSJM.

EQUIPMENT

50% OFF PREVIOUSLY OWNED MEDICAL—Laboratory, x-ray, ultrasound equipment. We buy, sell, broker, repair. Medical Equipment Resale & Repair, 24026 Haggerty, Farmington Hills, MI 48018, 1-800-247-5826, (313) 477-6880.

EQUIPMENT—CONT'D

DISCOUNT HOLTZER SCANNING SERVICES starting at $35.00. Spacelabs holter recorders available from $1,275.00. Smallest and lightest holters update. Fast service (24–48 hours) turnover. Hook-up kits starting at $4.95. Special introductory offer of three free tests at the purchase or lease of the holter. Stress test electrodes available at 34 cents. Scanning paper available at $18.95. Cardiologist overread available at $15.00. If interested call (301) 870-3626.

REAL ESTATE FOR SALE OR RENT

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

EAST SIXTIES, PROFESSIONAL BUILDING. The preferred location for internists, cardiologist, rheumatologist. 7 treatment rooms, 2 reception areas to share with three other internists. Share NYC licensed lab, radiology, thermography. Full or part time. (212) 838-2860.

5th AVENUE/11th ST. Newly renovated, fully equipped medical office to share in elegant townhouse on historic 11th St. Ideal for primary care internists, FP, or OB/GYN. Call (212) 529-4330 10–5.

OFFICE SPACE AVAILABLE TO SHARE—1,500 sq. ft. in Medical Service Building at St. Agnes Hospital, White Plains. Call (914) 428-7480.

OFFICE SPACE AVAILABLE: Throgs Neck section of Bronx. Modern medical complex, 1500 square feet, part time/full time. Call (212) 409-9494.

CENTRAL AVENUE, VALLEY STREAM, LONG ISLAND spacious office for rent, 6 rooms plus shared waiting room, corner, move-in condition. (516) 872-6690, (516) 825-1427.

EAST NORTHPORT: Office space for medical practitioner or group. Can provide from 700 to 8000 sq. ft. if necessary. Housed in large white colonial building in East Northport, NY midway between Huntington and Smithtown, Long Island. Almost any sort of financial arrangement can be worked out. Please contact: Robert Smirnow, D.D.S., 7 Pulaski Road, East Northport, L.I., New York 11731, (516) 261-1068.

MANHATTAN, 7 EAST 68th STREET, medical office sub-lease. Lots of rooms, R.M.S., busy office with referrals, flex hours. (212) 879-4260.

PRIME MIDTOWN MEDICAL SPACE East 37th Street between Lexington Ave. and Third Ave. Private street entrance, near eastside hospitals. Convenient to all transportation, flexible lease. Waiting room, separate exam rooms. Perfect for surgical or medical specialties. Call (212) 473-3049 Dr. Fox.

H.M.S.S.

The AMA
Hospital Medical Staff Section
Ninth Assembly

JUNE 18-22, 1987
PALMER HOUSE
CHICAGO

Represent your medical staff.

For Information Contact:

Department of Hospital Medical
Staff Services
American Medical Association
535 North Dearborn Street
Chicago, Illinois 60610
Phone (312) 645-4747 or 645-4753

AMA

News from DISTA about a new dosage form of cephalexin

ANNOUNCING NEW

Keflet™ TABLETS cephalexin

All the advantages of cephalexin in a convenient tablet form

- Backed by over 15 years of clinical experience
- Smaller tablet is specially shaped and coated for easier swallowing
- May enhance patient compliance, particularly among the elderly
- Tablet dosage form may be appreciated by patients of all ages

NEW Keflet Tablets are available as:

250-mg
Tablets

500-mg
Tablets

Keflet is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-sensitive patients.

© 1987 DISTA PRODUCTS COMPANY

Brief Summary. Consult the package literature for prescribing information.

Indications and Usage: Keflet™ Tablets (cephalexin, Dista) are indicated for the treatment of the following infections when caused by susceptible strains of the designated microorganisms.

Respiratory tract infections caused by *Streptococcus pneumoniae* and group A β-hemolytic streptococci (Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. Keflet is generally effective in the eradication of streptococci from the nasopharynx; however, substantial data establishing the efficacy of Keflet in the subsequent prevention of rheumatic fever are not available at present.)

Otitis media due to *S pneumoniae*, *Haemophilus influenzae*, staphylococci, streptococci, and *Neisseria catarrhalis*

Skin and skin-structure infections caused by staphylococci and/or streptococci

Bone infections caused by staphylococci and/or *Proteus mirabilis*

Genitourinary tract infections, including acute prostatitis, caused by *Escherichia coli*, *P mirabilis*, and *Klebsiella* sp

Note—Culture and susceptibility tests should be initiated prior to and during therapy. Renal function studies should be performed when indicated.

Contraindication: Keflet is contraindicated in patients with known allergy to the cephalosporin group of antibiotics

Warnings: BEFORE CEPHALEXIN THERAPY IS INSTITUTED, CAREFUL INQUIRY SHOULD BE MADE CONCERNING PREVIOUS HYPERSENSITIVITY REACTIONS TO CEPHALOSPORINS AND PENICILLIN. CEPHALOSPORIN C DERIVATIVES SHOULD BE GIVEN CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS

SERIOUS ACUTE HYPERSENSITIVITY REACTIONS MAY REQUIRE EPINEPHRINE AND OTHER EMERGENCY MEASURES

There is some clinical and laboratory evidence of partial cross-allergenicity of the penicillins and the cephalosporins. Patients have been reported to have had severe reactions (including anaphylaxis) to both drugs.

Any patient who has demonstrated some form of allergy, particularly to drugs, should receive antibiotics cautiously. No exception should be made with regard to Keflet

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening

Treatment with broad spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C difficile*. Other causes of colitis should be ruled out

Usage in Pregnancy—Safety of this product for use during pregnancy has not been established

Precautions: *General*—Patients should be followed carefully so that any side effects or unusual manifestations of drug idiosyncrasy may be detected. If an allergic reaction to Keflet occurs, the drug should be discontinued and the patient treated with the usual agents (eg, epinephrine or other pressor amines, antihistamines, or corticosteroids).

Prolonged use of Keflet may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug

Keflet should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended

Indicated surgical procedures should be performed in conjunction with antibiotic therapy

As a result of administration of Keflet, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Broad spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis

Usage in Pregnancy—Pregnancy Category B—The daily oral administration of cephalexin to rats in doses of 250 or 500 mg/kg prior to and during pregnancy, or to rats and mice during the period of organogenesis only, had no adverse effect on fertility, fetal viability, fetal weight, or litter size. Note that the safety of cephalexin during pregnancy in humans has not been established

Cephalexin showed no enhanced toxicity in weanling and newborn rats as compared with adult animals. Nevertheless, because the studies in humans cannot rule out the possibility of harm, Keflet should be used during pregnancy only if clearly needed

Nursing Mothers—The excretion of cephalexin in the milk increased up to 4 hours after a 500-mg dose; the drug reached a maximum level of 4μg/mL, then decreased gradually, and had disappeared 8 hours after administration. Caution should be exercised when Keflet is administered to a nursing woman.

Adverse Reactions: *Gastrointestinal*—Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely. The most frequent side effect has been diarrhea. It was very rarely severe enough to warrant cessation of therapy. Dyspepsia and abdominal pain have also occurred. As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely

Hypersensitivity—Allergic reactions in the form of rash, urticaria, angioedema, and, rarely, erythema multiforme, Stevens-Johnson Syndrome, or toxic epidermal necrolysis have been observed. These reactions usually subsided upon discontinuation of the drug. Anaphylaxis has also been reported

Other reactions have included genital and anal pruritus, genital moniliasis, vaginitis and vaginal discharge, dizziness, fatigue, and headache. Eosinophilia, neutropenia, thrombocytopenia, and slight elevations in SGOT and SGPT have been reported

PV 2211 DPP

[111786]

Additional information available to the profession on request from

DISTA

Dista Products Company
Division of Eli Lilly and Company
Indianapolis, Indiana 46285
Mfd by Eli Lilly Industries, Inc
Carolina, Puerto Rico 00630

720073

NEW YORK STATE
JOURNAL OF MEDICINE

May 1987 • Volume 87, Number 5

COMMENTARIES

Acquired immunodeficiency syndrome—1987

The epidemic of acquired immunodeficiency syndrome (AIDS) is now moving through its seventh documented year in the United States. Unlike other epidemics of this century, this one has posed unique problems because of the nature and characteristics of the causative viral agent, the relatively long incubation period from infection to clinical signs and symptoms, and the long period of communicability. It has appeared both in high-technology societies such as those in the United States and Europe, and in developing areas, exemplified by sub-Sahara Africa. Vaccines for prevention and effective therapeutic agents for cure are still years away, forcing reliance on older methodologies of prevention and on high-technology supportive interventions for keeping patients with the disease alive. These basic characteristics plus the current social determinants of the disease have presented Americans with a number of social, medical, political, and economic issues that are now the focus of much public discussion and, in some instances, heated debate.

The articles presented in this issue of the *Journal* are uniquely devoted to AIDS. They cover a broad range of topics and opinions contributed by a national authorship and reflect the complexity of the challenges posed by the disease. On the positive side, they demonstrate how inexorable has been the advance of our knowledge about AIDS and the high degree of societal commitment to dealing with the disease and all of the problems associated with it. From the perspective of 1987 this represents a marked advance forward from 1981 when the disease was first reported in the United States. It is useful to briefly look backward to remind ourselves about just how far we have come. For if the past is any predictor of the future, our advances in the several years ahead should be no less dramatic.

On June 5, 1981, the Centers for Disease Control (CDC) reported five cases of *Pneumocystis carinii* pneumonia among sexually active homosexual men.[1] The report stated that "All of the above observations suggest the possibility of a cellular-immune dysfunction related to a common exposure that predisposes individuals to opportunistic infections such as pneumocystosis and candidiasis."[1] Within a short period of time the disease that had only been observed among homosexual men was found among other "at risk" groups, including intravenous (IV) drug users, recent Haitian immigrants, hemophiliacs, sexual partners of those who had the disease, recipients of blood transfusions, and infants of mothers with the disease or at risk for it.[2] In late 1982 the CDC promulgated a surveillance definition for AIDS.[3] The epidemic was underway.

Early on, several groups of medical scientists began what would prove to be a fruitful search for the causative agent of the disease. In 1983 research produced suggestive evidence that the agent might be a human retrovirus, which was later isolated and called by a variety of names, including LAV (lymphadenopathy-associated virus), HTLV-III (human T-cell lymphotropic virus type III), and ARV (AIDS-associated retrovirus).[4,5] For a few years the combined acronyms HTLV-III/LAV were used in the medical literature to describe the virus. The term human immunodeficiency virus (HIV) was later proposed as a generic term as the various early isolates of the retrovirus were found to be closely related.[6] This term is now widely used for the causative agent of AIDS.

As of December 1986, 28,098 cases of AIDS had been reported in the United States.[7] By February 1987 the number had risen to 31,036 (personal communication, CDC, USPHS, February 27, 1987). The CDC estimates that 1.5 million Americans are infected with the virus. As of December 1986, 56% of adults (15,757) and 60% of children with the disease had died.[7] Of those who were diagnosed before January 1985, 79% had died by December 1986.[7]

The epidemiologic evidence gathered to date demonstrates that susceptibility to the disease may be general. The incubation period varies from a minimum of a few months after exposure to several years. Transmission primarily occurs from person to person through sexual contact, through the sharing of contaminated IV injection equipment, through the transfusion of blood or blood products, and transplacentally. Issues such as the role, if any, of either superinfection or reinfection in the clinical course of the disease have not yet been elucidated. Communicability probably exists from the start of the asymptomatic period through the development of the opportunistic diseases that characterize the clinical course of AIDS. The disease is now widely spread throughout the world and from the vantage point of 1987 it is likely that this spread will continue. The absence of cases in specific geographic areas may be more reflective of the failure to detect the disease and report it than to zero incidence.

In many countries of the third world and particularly in sub-Sahara Africa there is a reluctance to acknowledge the disease's existence. Yet this region of the world is the most affected by it. Denial has its roots in a complex web involving national pride, fear of being stigmatized and suffering economic repercussions (such as the loss of tourism), and concern about the local, social, and political consequences of admitting to a major problem for which remedial resources are unavailable. In some African countries such as Kenya, where studies of female prostitutes have shown that close to 60% are infected with HIV, citizens have been told to ignore what national leaders characterize as "malicious propaganda emanating from outside the country."[8] Yet in other countries such as Zaire there has been a frank recognition of the problem and constructive steps have been taken to deal with it.[9] In Kenya, with 600,000 foreign tourists a year, concern runs high about the economic, social, and political consequences of a reactive fall-off of visitors fearful of AIDS. Yet even there, where the disease is progressively spreading, positive public health efforts are being undertaken. Among these are universal screening of all blood donations for AIDS and large-scale health education programs.[8]

In this issue of the *Journal* Quinn[10] provides an excellent overview of the problem of AIDS in Africa. He cogently notes the risk factors associated with the predominately heterosexual mode of transmission: sexual contacts with prostitutes, promiscuity, being a prostitute, and being a sexual partner of an infected person. However, the virus is also transmitted in Africa by blood transfusions and the use of contaminated injection equipment for medical purposes.[10,11] The primarily heterosexual transmission of HIV in Africa has been viewed by some observers as a portent of epidemiological things to come in Europe and the United States. The obvious caveats of course are that the epidemiology of AIDS in Africa is at the midpoint of 1987 incompletely understood, and that there may be other determinants in the African AIDS scenario that are not always present in the United States and Europe. These include large numbers of sexual partners and frequent unprotected prostitute contacts among heterosexual African AIDS patients, the widespread use of unsterile needles and syringes in clinics and hospitals, and the poorly understood process of HIV transmission enhancing the role of concurrent or antecedent sexually transmitted diseases.[11]

Yet it is clear that 1987 represents the year in the AIDS epidemic when heterosexual transmission became a major focus of concern. Whether or not AIDS will become a major problem in the general heterosexual American population is at present something of an open question. Largely confined now to homosexual males and IV drug abusers, who account for approximately 90% of all cases diagnosed as of late 1986,[7] AIDS seems unlikely to move in a major way out of these high-risk-behavior groups into the general heterosexual population over the narrow bridges of bisexuality, transplacental spread from infected mothers, and heterosexual transmission from IV drug abusers and other infected persons. For in the infection-recipient groups on the other side of these bridges the crucial variable of promiscuity is often lacking. The suburban housewife who is infected with AIDS by a bisexual husband is not likely to have subsequent sexual contacts with a score or more of men as are her husband's male partners. Thus this form of heterosexual transmission leads the virus into a dead end from which it does not usually escape. Transmission from high-risk-behavior groups via blood transfusions will be minimal in the US given the relative safety of the blood supply.

However, based on what is already known about the epidemiology of AIDS, it is probable that the number of HIV infections will increase among heterosexuals. This increase is likely to occur first in heterosexual subset populations that are closest to the existing transmission bridges from currently infected high-risk-behavior groups. The degree and rapidity of spread outside of this center will be directly related to the frequency of sexual contacts and the number of sexual partners, variables likely to be affected by public health education activities now underway.

Hein[12] in this issue of the *Journal* underscores the vulnerability of certain groups of teenagers who, because of early age of first intercourse, choice of sexual partners, rates of sexually transmitted diseases, current contraceptive practices, and other factors, are at high risk for acquiring HIV infections. Many of these teenagers are on the other side of the AIDS transmission bridge from IV drug abusers and form a potential part of the epidemiologic center of the epidemic. Many of them are found in inner city areas and should be the immediate focus of intense public health education activities.

The data on HIV infection rates among civilian applicants for military service presented by Burke et al[13] in this issue of the *Journal* may or may not sustain concern about spread to the general heterosexual population depending on one's interpretation of the data. One can cogently argue that the civilian applicants for military service who were screened are not representative of the universe population of which they are a part but rather may represent subsets whose prior behaviors may have put them at significant risk for HIV infection. If they are truly representative of the universe heterosexual population then one should see similar levels of HIV infections in this larger group. Yet initial studies to date of the larger universe population conducted on donated blood have only shown HIV prevalence rates of 0.38%.[14] There are limitations to the data generated from surveys of blood donated to blood banks just as there are limitations to data on HIV prevalence gathered at sexually transmitted disease clinics. What is needed are carefully designed ongoing surveys of the general heterosexual population. These were called for by some at the February 1987 conference on AIDS sponsored by the CDC in Atlanta, Georgia.[15] If such surveys are ever undertaken, they could provide useful insights into the epidemiology of the disease in the heterosexual population. Meanwhile, current predictions as to what is likely to occur in the heterosexual population are largely derived from existing data which are far from complete.[16]

Far more complete are data on high-risk-behavior groups: homosexual men and IV drug abusers. Concerning the latter, Des Jarlais and his colleagues[17] provide useful insights in this issue of the *Journal* into the relationship between IV drug abuse and the heterosexual transmission of HIV. Of significance is the finding that HIV-infected prostitutes in New York City seem to have rarely transmitted the infection to clients. Des Jarlais and his colleagues put forth as one of the reasons for this the very credible explanation that prostitutes in the city have employed "safe sex" procedures, particularly the use of condoms, for a number of years as protection against other sexually transmitted diseases.[17] They also underscore the close linkage between IV drug abuse and heterosexual transmission of HIV infection in the city.[17]

The possible effectiveness of condoms in preventing the transmission of HIV infection has also been a prominent issue of the 1987 agenda for AIDS. There has been much heated discussion over the proposal to advertise condoms on television, a proposal supported by the Surgeon General of the US Public Health Service, C. Everett Koop, MD.[18] Network television and some local stations have avoided these ads out of concern for offending the beliefs and convictions of a significant proportion of the American public that sees in them the potential for approving and promoting extramarital sexual relations, particularly among young people. Religious groups and the Secretary of Education, William Bennett, have advocated abstinence as the safest and best means of preventing HIV infection. The heated polemics that have characterized the discussion of this issue have centered more on moral, religious, and social issues than on the scientific one, namely, the effectiveness of condoms in preventing transmission of HIV. To date there are no extensive data documenting the level of prevention afforded by condoms against transmission under a variety of use circumstances.

The year 1987 has witnessed a strong call for public health education as one of the best means for preventing the transmission of HIV infections. Yet there are areas of disagreement about content, the debate over condom use being the best example. The debate over prevention strategies continues and has tended to center more in the arena of heterosexual transmission than in those of homosexual and IV drug abuse transmission, where high prevalence rates of HIV infection should serve as a strong incentive for behavior modification.

Two papers in this issue of the *Journal* describe health education intervention strategies. Valdiserri and his colleagues[19] describe small group intervention sessions with homosexual and bisexual men that show promise for reducing high-risk behavior. Echenberg[20] describes two intervention strategies developed in San Francisco for both high- and low-prevalence groups. In addition to mass health education, the program employed by the San Francisco Department of Public Health utilizes contact notification in the low-prevalence group. This latter strategy is a very useful adaptation of that which has long been used to trace sexually transmitted disease partners.[20] Both the program in Pittsburgh and the one in San Francisco demonstrate that public health education can go a long way at this stage of the epidemic.

How local communities deal with the AIDS epidemic in terms of public policy is clearly illustrated in the paper by Arno and Hughes[21] in this issue of the *Journal*. They show how the contrasting public policy responses in New York City and San Francisco are attributable to a number of factors, prominent among which are the patient mix, the character of the public health and health care systems in each city, differences in the magnitude of the epidemic, and the role of risk groups and the local media and press in molding local response. This communication focuses on another prominent issue that has received much attention in 1987, namely, that of the medical care costs involved in caring for patients with AIDS. Related to individual patient care costs and those of the aggregate is the issue of the impact of the epidemic on the health care system. The epidemic has already had a significant effect on the number and mix of beds in many hospitals. As the numbers of patients with AIDS rise, pressures build to transfer them to municipal, generally teaching, hospitals, because of failure to receive adequate reimbursement from third-party payers. Educational programs for house staff and medical students have also been affected in teaching institutions by the number of inpatients with AIDS. Institutions struggle with their responsibility to treat AIDS patients while simultaneously trying to maintain a mix of patients with other disease states that helps assure both the comprehensiveness of educational content and its quality.[22] As the number of patients with AIDS continues to grow there will be a need to provide a broad range of ambulatory and inpatient services, as discussed by DeHovitz and Pellegrino in this issue.[23] To meet these new service requirements health care planners must rethink pre-epidemic strategies and plans so as to permit rapid service responses to changing needs. Based on CDC prevalence estimates for AIDS, Scitovsky and Rice[24] estimate that the personal medical care costs for the disease will rise from the $630 million spent in 1985 to $8.5 billion in 1991. They estimate that indirect costs attributable to productivity loss among victims will rise to $55.6 billion in 1991, from $3.9 billion in 1985. Nonpersonal costs for research, education, screening, and general support services are expected to rise from $542 million for 1986 to $2.3 billion in 1991.[24] As increasing numbers of patients with AIDS enter the medical care system, there will be an increased need for in-service education programs for hospital workers. The results of one such program are described by O'Donnell and O'Donnell[25] in this issue.

The year 1987 witnesses a continued growth in our knowledge of the clinical course of AIDS and of means for dealing with the disease's many complications. Foster et al[26] describe the therapeutic use of meclofenamate in patients with AIDS-related pulmonary involvement. Their study demonstrates that as necessity develops, as it frequently does in this disease, newer modalities are found and applied. However, therapeutic interventions have thus far been palliative at best. Much hope was attached to the introduction of azidothymidine (AZT) in late 1986.[27] At this writing it is too early yet to say how truly effective this drug will be as a therapeutic agent. Although AZT is easy to administer, its untoward effects require careful patient monitoring. DeHovitz[28] makes an important point in noting that this increased monitoring will translate into more frequent patient-physician contact which in turn will escalate medical care costs. Real breakthroughs in the development of other effective anti-HIV agents and a vaccine do not appear imminent.

As of this writing no other issue has seized the center of the debate platform as has that of mandatory testing for AIDS. The point of contention is not the testing itself but rather the confidentiality issue that is inextricably linked to it.[15] Instances have been reported where the confidential nature of positive AIDS tests has been breached, resulting in dismissal from employment; loss of housing; and cancellation of life, disability, and health insurance.[29] This issue was widely debated at the February 1987 conference on AIDS sponsored by the CDC, at which many cogent arguments against mandatory testing were put forth.[15] On the face of it mandatory testing appears to be an effective way of identifying those infected with HIV and of encouraging them not to engage in behaviors that would spread it. If the test were mandatory for all hospital admissions and for patients attending sexually transmitted disease clinics, significant numbers of HIV-infected individuals could be identified, their behavior modified, and the spread of the disease curtailed. In the absence of a cure or vaccine this appears to be a reasonable approach to prevention of spread.

However, there is some evidence that mandatory testing would drive those who are infected underground and away from preventive interventions, thus defeating the purpose of such testing.[30] The true magnitude of this impact has yet to be accurately documented. Given the current distribution of AIDS, which primarily occurs among homosexual men and IV drug abusers, it would probably not be cost effective to screen the 37 million Americans who are admitted to hospitals each year. The inducements for so doing would include a major change in the epidemiology of the disease in which very significant numbers of the general heterosexual population would become infected. Such an epidemiologic development does not appear imminent from the perspective of 1987. Also, a marked increase in the total number of cases might prompt reconsideration of mandatory testing.

A consensus has developed among public health officials in the United States in 1987 that voluntary testing linked to counseling both before and after the test holds hope for inducing people to avoid high-risk behaviors.[31] It is this belief that underpins New York State's newly expanded program for counseling and testing. The degree of effectiveness of this approach in preventing the spread of HIV infection has yet to be demonstrated. Meanwhile, as long as the risks of discrimination in employment, housing, and insurance remain linked to possible breaches in confidentiality about positive test results, there will continue to be strong opposition to mandatory testing.[32,33] These concerns were heightened by the US Justice Department's ruling in June 1986 which denied HIV carriers the protection afforded by a federal law prohibiting discrimination against the handicapped.[31] On March 3, 1987, the US Supreme Court ruled that recipients of federal monies could not discriminate against those who are physically or mentally impaired by contagious diseases.[34] Although the decision was handed down in the case of a Florida elementary school teacher who was dismissed in 1979 because of active tuberculosis, it affects those with AIDS. The immediate beneficiaries are children who are excluded from school because of AIDS and adults with the disease who are employed by employers receiving federal monies.[34] The critical issue of whether or not the federal 1973 Rehabilitation Act protects carriers of HIV who are asymptomatic was left undecided.[34] Although this decision affords less than complete protection to AIDS victims, it represents a major step forward.

In 1987, Louis Harris and Associates published the results of a survey of 227 leading biomedical researchers and medical scientists concerning future projections about AIDS.[35] The survey group projected that a million Americans will have developed AIDS by the year 2000. Among a subset of specialists knowledgeable about prevention and treatment, 46% predicted that a safe and effective vaccine would be available between 1990 and 1999.[35] Of this same subset of scientists, only 28% thought that there would be an effective cure for the disease before the end of the century. Of the entire group of 227, only 19% thought that AIDS could be eliminated by the end of the century. Predicting the course of any epidemic disease is extremely difficult because the expected often fails to happen and the unexpected occurs with regularity. There are many unforeseen variables and surprise developments that lay ahead on the road of AIDS prevention and treatment. From the vantage point of 1987, it is impossible to predict what they will be. What is more certain is that American society will have to continue to deal with the myriad problems associated with AIDS, many of which are comprehensively addressed in this issue of the *Journal*.

PASCAL JAMES IMPERATO, MD
Editor

Professor and Chairman
Department of Preventive Medicine
and Community Health
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

1. *Pneumocystis* pneumonia. *MMWR* 1981; 30:250-252.
2. Institute of Medicine, National Academy of Sciences: *Confronting AIDS, Directives for Public Health, Health Care and Research*. Washington, DC, National Academy Press, 1986, p 37.
3. Update on acquired immune deficiency syndrome (AIDS)—United States. *MMWR* 1982; 31:507-514.
4. Barre-Sinoussi F, Chermann JC, Rey F, et al: Isolation of a T-lymphotropic retrovirus from a patient at risk for acquired immune deficiency syndrome (AIDS). *Science* 1983; 220:868-871.
5. Gallo RC, Salahuddin SZ, Popovic M, et al: Frequent detection and isolation of cytopathic retroviruses (HTLV-III) from patients with AIDS and at risk for AIDS. *Science* 1984; 224:500-503.
6. Coffen J, Haase A, Levy JA, et al: Human immunodeficiency virus. *Science* 1986; 232:697.
7. Update: Acquired immunodeficiency syndrome—United States. *MMWR* 1986; 35:757-760, 765-766.
8. Brooke J: Kenya is reacting bitterly as AIDS is highlighted. *NY Times*, February 24, 1987, p B4.
9. Brooke J: AIDS danger: Africa seems of two minds. *NY Times*, January 3, 1987, p 13.
10. Quinn TC: AIDS in Africa: Evidence for heterosexual transmission of the human immunodeficiency virus. *NY State J Med* 1987; 87:286-289.
11. Imperato PJ: The epidemiology of the acquired immunodeficiency syndrome in Africa. *NY State J Med* 1986; 86:118-121.
12. Hein K: AIDS in adolescents: A rationale for concern. *NY State J Med* 1987; 87:290-295.
13. Burke DS, Brundage JF, Bernier W, et al: Demography of HIV infections among civilian applicants for military service in four counties in New York City.

NY State J Med 1987; 87:262-264.
14. Schorr JB, Berkowitz A, Cumming PD: Prevalence of HTLV-III antibody in American blood donors. *N Engl J Med* 1985; 313:384-385.
15. Altman LK: Mandatory tests for AIDS opposed at health parley. *NY Times*, February 25, 1987, pp I,16.
16. DeHovitz JA: A perspective on the heterosexual transmission of the acquired immunodeficiency syndrome. *NY State J Med* 1986; 86:117-118.
17. Des Jarlais DC, Wish E, Friedman SR, et al: Intravenous drug use and the heterosexual transmission of the human immunodeficiency virus: Current trends in New York City. *NY State J Med* 1987; 87:283-286.
18. Werner LM: Koop urges TV condom ads to fight AIDS. *NY Times*, February 11, 1987, pp 1, A10.
19. Valdiserri RO, Lyter DW, Kingsley LA, et al: The effect of group education on improving attitudes about AIDS risk reduction. *NY State J Med* 1987; 87:272-278.
20. Echenberg DF: Education and contact notification for AIDS prevention. *NY State J Med* 1987; 87:296-297.
21. Arno PS, Hughes RG: Local policy responses to the AIDS epidemic: New York and San Francisco. *NY State J Med* 1987; 87:264-272.
22. Wachter RM: The impact of the acquired immune deficiency syndrome on medical residency training. *N Engl J Med* 1986; 314:177-179.
23. DeHovitz JA, Pellegrino V: AIDS care in New York City: The comprehensive care alternative. *NY State J Med* 1987; 87:298-300.
24. Scitovsky AA, Rice DP: Estimates of the direct and indirect costs of acquired immunodeficiency syndrome in the United States, 1985, 1986 and 1991. *Public Health Reports* 1987; 102:5-17.
25. O'Donnell L, O'Donnell CR: Hospital workers and AIDS: Effect of in-service education on knowledge and perceived risks and stresses. *NY State J Med* 1987; 87:278-280.
26. Foster SH, Garrett RC, Thomas HM 3d: Cyclooxygenase inhibition and improved oxygenation in patients with pulmonary complications of AIDS. *NY State J Med* 1987; 87:280-282.
27. Barnes DM: Promising results halt trial of anti-AIDS drug. *Science* 1986; 234:15-16.
28. DeHovitz JA: Azidothymidine and human immunodeficiency virus infection. *NY State J Med* 1987; 87:7-8.
29. Altman LK: Privacy called vital to AIDS screening. *NY Times*, March 1, 1987, p E26.
30. Mandatory testing for AIDS? *US News and World Report* 1987: 102:62.
31. A new strategy against AIDS. *NY Times*, March 1, 1987, p E24.
32. Boffey PM: Homosexuals applaud rejection of mandatory tests for AIDS. *NY Times*, February 26, 1987, p B7.
33. Boffey PM: Laws urged to protect identities in AIDS testing. *NY Times*, February 25, 1987, p A18.
34. Taylor S Jr: Rights of disease victims backed; those with AIDS could benefit. *NY Times*, March 4, 1987, pp A1, A21.
35. Schmeck HM Jr: Million victims of AIDS predicted by year 2000. *NY Times*, March 4, 1987, p A20.

AIDS: The eleventh year*

In his novel *The Plague*, Albert Camus observed that "Epidemics like wars always seem to take people by surprise."[1] A number of the papers in this issue of the *Journal* address the latest "surprise" of acquired immunodeficiency syndrome (AIDS)—the significance of heterosexual transmission in the United States. While our finest biomedical scientists struggle to understand and gain some control over this "new" virus, the known risk groups are being decimated by disease and the number of new cases, many involving heterosexual transmission, seems to grow inexorably, and ultimately will certainly affect all of society.

In June 1986, the US Public Health Service held a meeting on AIDS in Berkeley Springs, West Virginia.[2] Among the predictions coming from that meeting were that, by 1991, only four years from now, the cumulative number of cases of AIDS will exceed a quarter of a million. It was also predicted that in 1991, the tenth year of the epidemic, we in the US will have to care for 150,000 people who are sick with AIDS, approximately ten times the number in care today—a number already straining health care resources in many cities.

Finally, it was predicted that by 1991, in the absence of a dramatic breakthrough in treatment, at least 54,000 more people will have died of AIDS, approximately the same number of Americans who died in the 15 years of the Vietnam War. This will bring the cumulative number of AIDS deaths in the US to 179,000 in the decade since AIDS was first reported in 1981.

By some cruel irony of history, the year 1992, the 11th of AIDS in the US, will also be the 500th anniversary of Columbus' first voyage across the Atlantic. This historical event, important both to our civilization and to the modern world, is also one of profound biological significance. The year 1492 is full of meaning because it marks the end of the almost total isolation of a huge portion of humanity, a natural quarantine imposed by two great oceans for over 20,000 years. The historian Alfred Crosby calls this event and its aftermath *The Columbian Exchange*,[3] the moment in which two long-separated worlds were reunited, with enormous consequences for both. Aside from all the gold and the silver, the slaves and the soldiers, potatoes and tobacco, tomatoes and maize (all the things which we were schooled to associate with the discovery of the New World), there was also a great biological mingling and the reestablishment of a global system of health and disease which has shaped our world ever since.

The Europeans brought smallpox, typhus, and cholera to the New World, and these killed more native Americans than all the Conquistadores' muskets and cannons. And, in exchange, on the very first ship of Columbus' expedition returning to Barcelona in 1493 traveled the syphilis treponema, a deadly stowaway. Whether it was in one of the crew members or in one of the Indians returned as captives to Europe does not really matter. Syphilis, unknown in Europe at that time, would, within a dozen years, spread to the rest of the Old World and take a fearful toll.

Five hundred years ago, when it first struck Europeans, syphilis was, for them, a new disease, as AIDS is for us today. It appears to have been far more virulent and far more dangerous than the syphilis we see now. The first risk groups for syphilis were soldiers and sailors, and warfare provided the means for its rapid spread via sexual transmission to the entire population of 16th century Europe and beyond.

---

* This article is based on the opening address presented at the "Symposium on the Heterosexual Transmission of AIDS," sponsored by the Department of Epidemiology and Social Medicine, Montefiore Medical Center and Albert Einstein College of Medicine, and held October 21, 1986, at the New York Academy of Medicine.

Crosby says of the spread of syphilis in Europe:

> Charles the 8th of France crossed the Alps into Italy with an army of about 50,000 soldiers (of French, Italian, Swiss, German and other origins) bound for the capture of Naples. The army trailed its column of the usual camp followers and engaged in the usual practices of rape and sack. But, Ferdinand and Isabella, anxious to prevent the establishment of French power in Italy, sent Spanish troops. So Charles packed his bags and marched back to France and the whole process of battle, rape and sack was repeated in reverse. Syphilis, hitherto spreading slowly and quietly across Europe, flared into epidemic in Italy during this invasion. Charles arrived back at Lyon in November of 1495 and disbanded his army. Its members, with billions of treponemas in their blood streams, scattered back to their homes in a dozen lands or off to new wars. With the dispersal of this army, the lightning spread of syphilis across Europe and the rest of the world became inevitable.[3]

Syphilis appeared in Germany by the summer of 1495; in Switzerland and France later that same year; in Holland, England, Greece, and Hungry in 1496. It reached Russia by 1499, and by 1505 it had reached China, 11 years after its first appearance in Spain.[3] As we consider the 11th year of AIDS, are there parallels to draw?

At the second International Conference on AIDS held in Paris in June 1986, one could see the conjunction of the work of many investigators studying the heterosexual transmission of AIDS and the latest data on the prevalence of infection in cities around the world. The most startling data concerned the high levels of infection among intravenous drug users in Europe: Barcelona, 70%; Edinburgh, 42%; Bologna, 52%; Milan, 30%; Toulouse, 58%; Paris, 65%; Vienna, 35%; Belgrade, 48%—levels which must grow given the lack of drug treatment and the continued illegality of providing sterile needles for drug users in all of these cities. These are rates of infection which have ominous implications, not only for the addicts themselves but, given the usual heterosexual character of the group, for all of their many sexual partners. Indeed, intravenous drug abusers are potentially a bridge to the heterosexual population at large.

The major studies on heterosexual transmission discussed in Paris—especially the work from the University of Miami,[4] Montefiore Medical Center in the Bronx, NY,[5] and Walter Reed Army Hospital in Washington, DC,[6] had all been presented at other scientific meetings and some had already appeared in print. Further, if one chose to apply the findings, reports of the experience with AIDS in Haiti and Africa also seemed to shed light on heterosexual transmission. However, the time lag associated with publication seemed poorly attuned to the pace of a rapidly growing epidemic and threatened to create a serious gap in understanding both within the scientific community and between that community and the general public. For these reasons some exchange of information and viewpoints more in "real time" seemed appropriate. Finally, there was clearly a need for dialogue between those conducting the primary research and surveillance, those whose responsibility it is to care for the patients, and those involved in shaping public policy aimed at prevention. These were the reasons for organizing the Symposium from which some of the papers in this issue of the *Journal* flowed.

Several different surveys have indicated low levels of public confidence in our communications about AIDS. People feel that they are not being told "the whole story." This lends some urgency to the need to both establish the truth about sexual transmission of AIDS and to fully communicate it to the public in the clearest possible terms and as soon as possible. One cannot leave a vacuum to be filled by anxieties and fears and those who pander to them. While we may be dimly aware of the dangers associated with rousing public fears about AIDS, few of us have had the experience of a epidemic of this sort, and we do not fully understand the hazards of doing the reverse—which is to remain silent while the process slips beyond our control. Indeed, in many areas this may already have happened.

Modern epidemiology can help if one uses it as the imaging and teaching tool that it can be. In discussing John Snow's work on cholera, Wade Hampton Frost of Johns Hopkins wrote in 1936 that epidemiology is "something more than the total of its established facts and includes chains of inference which *extend beyond* the bounds of direct observation."[7] We have before us today a good deal of direct observation about AIDS and, more importantly, new forms of knowledge about this disease, based on new technologies, which extend our vision very far indeed. These allow us, in essence, to see into the future of this epidemic. The studies included in this issue of the *Journal* amass much useful evidence on the question of the heterosexual transmission of AIDS and reveal something of the means and the potential magnitude of that route of infection. These studies cross international boundaries and differences of race and social class. They do, I believe, deal a blow to the concept of "risk groups," as opposed to "risk behavior," for the acquisition of this disease. For a time these groups served as a guide to help focus our preliminary research. However, in retrospect, they also appear to have been as much a psychological maneuver as a public health strategy, a maneuver which lulled many into believing they were not at risk and thereby ultimately failed to protect us.

The purpose of some of the papers in this issue of the *Journal* is to examine the notion that AIDS is a disease which has human sexuality at its fulcrum, not a disease of homosexuals or drug addicts, Haitians or Africans, although these groups have suffered significantly from its spread. These papers provide a basis for evaluating this judgment and a basis for considering some of its implications for our future.

ERNEST DRUCKER, PhD
Associate Professor
Department of Epidemiology and
Social Medicine
Montefiore Medical Center/
Albert Einstein College of Medicine
Bronx, NY 10467

---

1. Camus A: *The Plague*. New York, Alfred A. Knopf Inc, 1947.
2. US Public Health Service: Coolfont Conference on AIDS. Washington, DC, June 1986.
3. Crosby AW Jr: *The Columbian Exchange: Biological and Cultural Consequences of 1492*. Westport, Conn, Greenwood Press, 1975, pp 149–151.
4. Fischl MA, Dickinson GM, Scott GB, et al: Evaluation of heterosexual partners, children, and household contacts of adults with AIDS. *JAMA* 1987; 257:640-644.

5. Harris C, Small CB, Klein RS, et al: Immunodeficiency in female sexual partners of men with the acquired immunodeficiency syndrome. *N Engl J Med* 1983; 308:1181–1184.
6. Redfield RR, Markham PD, Salahuddin SZ, et al: Frequent transmission of HTLV-III among spouses of patients with AIDS-related complex and AIDS. *JAMA* 1985; 253:1571–1573.
7. Frost WH (ed): Introduction, in *Cholera*. Baltimore, Md, Johns Hopkins University Press, 1936, p ix.

AIDS in New York City: Moving ahead on effective public health approaches

The relentless tragedy of acquired immunodeficiency syndrome (AIDS) is nowhere more starkly felt than in New York City. As of the end of February 1987, 9,188 people in New York City had been diagnosed with AIDS; 58% have died. AIDS is the leading cause of death in New York City for men 25 to 44 years of age, and for women aged 25 to 29.[1]

Half a million people in New York City are estimated to be infected with human immunodeficiency virus (HIV) today. Homosexual and bisexual men make up the largest percentage of cases, though the proportion of AIDS cases among this group has fallen from 73% in 1981 to 55% in 1986, while the proportion of cases among intravenous (IV) drug abusers has risen from 22% in 1981 to 36% in 1986. From 50% to 60% of the city's estimated 200,000 heroin users are thought to be HIV-infected.[1]

IV drug abusers have been, and will likely continue to be, the major source of the spread of HIV infection to women and children in New York City. Of the 932 women diagnosed with AIDS in New York City since 1981, 80% have been IV drug abusers or the sex partners of IV drug abusers. One hundred seventy-eight cases of AIDS have occurred in New York City in children, the vast majority of these having been infected perinatally by their mothers. Ninety-two percent of mothers who infected their children were IV drug abusers or the sex partners of IV drug abusers.

AIDS is also having an increasing impact on New York City's minority communities. More than half of all AIDS victims in New York City are black or Hispanic. Eighty-six percent of male IV drug users with AIDS are black or Hispanic; 91% of mothers of children who have AIDS are black or Hispanic.[1]

By the end of 1991, more than 40,000 people will have developed AIDS in New York City alone; 30,000 will have died. These projections are based on a five-year horizon; no one knows the ten- or 20-year clinical outlook for those now infected. Nor do we know what long-term burdens will be posed by HIV-associated illnesses, such as tuberculosis (TB) or lymphoma. The impact of HIV immunodeficiency on the trends of other important diseases is under study and as yet is incompletely understood.

Against this background, and as the epidemic spreads in New York City, a number of public health policy issues are emerging as critical.

First, intensive public education efforts need to be implemented, without creating irrational anxiety and hysteria. Presently, the only feasible way to alter the course of the AIDS epidemic is by educating the public, training health and social service professionals, and offering counseling for personal risk reduction and antibody testing. Health professionals, educators, and the media must discuss AIDS issues appropriately, facing the facts about AIDS squarely, with honesty about our areas of uncertainty, and in explicit, understandable language.

Second, vigorous public health actions need to be increased while resisting ineffective measures of social control. Pressures are mounting for measures such as universal or mandatory AIDS antibody screening programs, as well as for isolation and quarantine of AIDS victims. At a conference sponsored by the Centers for Disease Control (CDC) held in Atlanta in February 1987, virtually all public health officials from the areas of highest AIDS prevalence agreed that forcing people to learn their serostatus, when no treatment is available, and when confidentiality may not be assured, would be unwise and counterproductive.

Third, increases in available resources for research and education must support the broad range of clinical, public health, and social service needs. The federal government has been very reluctant to recognize the true dimensions of and propose an adequately funded federal response to AIDS. It must do much more to support the unprecedented demands for AIDS treatment, human services, public health initiatives, and human rights resources, without sacrificing the research and education efforts that are so necessary.

To translate these policy issues into measures for controlling the spread of AIDS, in the current absence of effective treatment, we must rapidly increase confidential, voluntary counseling and testing. The New York City Health Department is moving aggressively to make voluntary, confidential HIV antibody counseling and testing much more widely accessible. The Department has published a counseling and testing policy containing guidelines for health professionals involved in the prevention and treatment of HIV infection as well as education of those at risk.[2] The policy states that anyone should be able to know his or her antibody status, provided the test results will be kept confidential, testing is voluntary, and pre- and post-test counseling is available. We strongly recommend that physicians actively consider whether their patients may be at risk of AIDS infection, discuss risk-avoiding behavior as part of routine medical care of all patients, and offer counseling and, if appropriate, antibody testing to patients at risk.

To support the confidential, voluntary aspects of counseling and testing, we have adopted a course of action known as contact notification. With this approach, the Health Department is urging, and directly and actively assisting when asked, people who are seropositive to notify their contacts.

We must increase access to substance abuse treatment programs for IV drug addicts. This is one of the most critical options for halting the spread of AIDS among addicts and to women and children. Currently, New York City has long waiting lists for methadone maintenance programs; drug-free rehabilitation programs are full to capacity. The federal government must consider drug rehabilitation programs as one of our important priorities, and provide more federal dollars through states and communities so that more addicts may find treatment for their addiction.

The Department of Health, in conjunction with the Divison of Substance Abuse Services of New York State, has developed a proposal to explore an additional strategy for reducing the spread of HIV among drug users: a research project to determine the effect of the availability of clean needles to a small, carefully selected and monitored group of IV drug addicts awaiting entry into drug treatment programs. If approved by the State Health Commissioner, the research study will serve as the basis for evaluating the efficacy of this approach in halting the further spread of the AIDS virus, while avoiding promotion of IV substance abuse.

Efforts to expand our knowledge base must be increased. Research needs to be expanded to investigate the nature of HIV and its transmission. Health Department researchers are collecting data for two studies, a survey of hospital patients suspected of having AIDS to identify associated risk factors, and a study of risk factors for transmission of AIDS in heterosexual couples in which one partner contracted AIDS from a blood transfusion. In another project, Department researchers and five participating hospitals are studying HIV transmission from infected mother to child. A series of blind and anonymous serosurveys is planned, to shed greater light on current infection patterns, especially among women of childbearing age.

Massive public health education risk-reduction efforts must be increased. For the present, the most effective way to reduce the spread of HIV infection is a multifaceted prevention and risk-reduction strategy directing education at the general public, plus targeting information and outreach for people engaged in high-risk behavior. Such a strategy is currently being undertaken by the Health Department's program of AIDS education and counseling. The Health Department has launched a $1 million campaign to promote latex condom use in all appropriate circumstances. The campaign includes a plan to distribute a million condoms next year along with educational material. The Department has also been working with two advertising agencies to develop multimedia public health advertising campaigns that will begin in the spring of this year.

The false dichotomy between civil liberties and public health must be eliminated. Stands on issues such as contact tracing and mandatory antibody testing have at times been portrayed as sacrificing public health in the name of civil liberties. Yet the approaches taken in New York City are based on sound public health principles; virtually all public health professionals agree that public health would be hindered by control measures such as mandatory testing. HIV-infected people—already facing devastating discrimination in housing, employment, and insurance—would not cooperate with our counseling, education, and testing if they feared that society was considering measures that could lead to quarantine, isolation, and additional forms of discrimination.

Funding at the federal, state, and local levels must be increased. Last year, the National Academy of Sciences called for a $2 billion AIDS research and education effort. The Academy recommended that $1 billion a year be newly appropriated for extensive basic and applied biomedical investigations of the disease.[3] Another $1 billion would go for a massive continuing education campaign to increase public awareness of the ways of protecting against infection. The money would also be applied to other necessary public health measures, such as screening the blood supply, voluntary confidential testing, and increased efforts in treatment and prevention of IV drug use. The Academy's call must herald an increase in the national commitment against the massive problem of AIDS.

The epidemic, and our understanding of it, is growing and changing constantly. Responsible policy must be continually reexamined against these changing conditions, and modified if necessary.

Education, health promotion, and risk reduction—these will remain the critical weapons in the fight against AIDS for at least the next several years. Those of us in a position to influence policy must do all we can to advance these weapons against this mounting health problem in New York City, across the United States, and around the world.

STEPHEN C. JOSEPH, MD, MPH
Commissioner of Health
City of New York
New York, NY 10013

---

1. Analysis of New York City AIDS surveillance data, New York City Department of Health, 1987.
2. New York City Department of Health HIV counseling and testing policy, March 1987.
3. Institute of Medicine, National Academy of Sciences: *Confronting AIDS: Directions for Public Health, Health Care and Research.* Washington, DC, National Academy Press, 1986, pp 33–34.

The prevention of AIDS*

The papers in this special issue of the *Journal* dealing with acquired immunodeficiency syndrome (AIDS) constitute an urgent call to action. The problem is: What action? In this rapidly changing field, controversy surrounds detailed proposals, but the principles on which action should be based are, or at least ought to be, unchanging.

First of all, there must be a massive effort in public education. Other countries do it well. Sweden, which I will use as an example several times, offers intense education on sexual health and other health issues in the public schools, beginning at the earliest possible age.[1] That public education process continues, using every conceivable medium of information, throughout the lifetime of every citizen. We in the United States permit massive advertising for cigarettes and other forms of self-destruction and encourage television programs and films that implicitly encourage sex without responsibility, but do not use the same channels to reach our people about the perils that surround us and how they can be avoided.

There must be a massive expansion of relevant prevention and treatment services. Industrialized countries like Sweden, with approximately the same GNP per capita as the US, provide significantly more public services through higher rates of taxation, the proceeds of which are applied to human rather than military purposes.[2] Condoms, to take a highly relevant if relatively inexpensive example, are supplied extremely cheaply at multiple easily-accessible sites, including public vending machines.[3] Counseling and other preventive services are far more accessible than in the United States. Treatment services, including immediate availability for every drug user or other at-risk person who wants to use them, are viewed as an urgent public responsibility. We, as a rich country, can do no less.

There must be a far greater role in the formulation of public policy for those most affected and those most at risk. As an example, Sweden, after an extensive public education campaign, conducted a national referendum on the future of nuclear power; an extraordinarily large part of the electorate, compared to our levels of voting, took part.[4] More locally, most Swedish health service policies are set at the county level and almost all health service funding is collected and controlled at the local level.[2] We must begin, in our own heterogeneous nation—sharply divided between those who have wealth and power and those who do not—to find ways to involve people much more directly in the development and implementation of policies that directly affect their lives. For example, the needle-exchange program in Amsterdam is a coordinated project developed jointly by public health authorities and the organized "Association of Drug Addicts."[5] Greater involvement of those directly afflicted will not only lead to more effective policies but the resulting reduction in feelings of alienation and powerlessness will have reverberations for improvement of health and well-being throughout our society.

Any society that wishes to use repressive measures, such as various forms of quarantine, against its people must justify those actions not only on utilitarian grounds but also in terms of fairness or justice. In short, the society must earn the right to use such measures. To use the example of Sweden yet again, the Associated Press reported in February 1986 that an intravenous drug user "who tested positive for the virus believed to cause AIDS was involuntarily detained in a Stockholm hospital after continuing to share needles with other drug users and to have sex without taking precautions to protect his partner."[6] Without taking a position on the complex question of the usefulness, not to speak of the desirability, of involuntary detention in our society, the more important point is that Sweden may have earned the right to limit civil liberties in certain ways for the protection of others. The extraordinary nature of its health and human services, its emphasis on services for minorities and for others in its population who are least well off, its other social concerns for its people, and the many other areas in which it is working for the public health, may give it that right. We in the United States are only beginning to earn the right to selectively limit civil liberties for the protection of others from AIDS. We still permit tobacco advertising and smoking in public places; we still do not require passive restraints in automobiles; we fail to protect the public health in any number of appropriate ways. For us to single out certain vulnerable groups or certain areas of human behavior for Draconian restriction is morally unacceptable.

To again cite a specific example, in October 1985 the New York State Public Health Council adopted an addition to the state health code that defined "high risk sexual activity" as anal intercourse and fellatio, labelled any commercial facilities in which such activity takes place "a public nuisance dangerous to the public health," and permitted their closing by health authorities.[7] I was the only member of the Council to vote against the regulation. My dissent was based on two grounds. First, many knowledgeable groups argued that it was precisely in those kinds of establishments that some of the best education on sexual health issues was taking place, and that the recent fall in surrogate measures of "high risk" sexual activity, such as the incidence of gonorrhea, demonstrated that. Second, and more important, intravenous drug use appeared to represent a greater risk factor for AIDS transmission than fellatio or anal intercourse, particularly in New York City, and it is increasingly clear that vaginal intercourse is an important mode of transmission. Yet here was a measure that was restrictive in only one area of risk, not surprisingly directed against people and acts that some powerful people find repugnant, but which did nothing to attempt to deal with more important risk factors. Similar issues are raised by involuntary blood testing for antibodies (which it has recently been suggested should be applied as broadly as for all those applying for marriage licenses and for all those admitted to hospitals), and of course by job, school, or insurance denial for those who are antibody-positive. Even if such actions could be shown—as they have not so far been—to be rational they present extremely difficult problems. The point is that if our society wants to limit civil liberties in certain areas for the protection of public health, it must do so equitably and justly. Picking out certain groups for special restriction—particularly if they are socially and politically weak and vulnerable—or subjecting large groups to testing which is of questionable predictive or therapeutic value, may be a greater danger to our society than is AIDS.

---

* This article is based on a presentation at the "Symposium on the Heterosexual Transmission of AIDS," sponsored by the Department of Epidemiology and Social Medicine, Montefiore Medical Center and Albert Einstein College of Medicine, and held October 21, 1986, at the New York Academy of Medicine.

The nature of AIDS and the recent evidence on the routes of its communicability pose an enormous challenge to our society. The question is whether we will meet that challenge using methods that improve human rights and the quality of life for all—in short, whether we will use the crisis to move toward being a more democratic and just nation, or whether our response will further erode the already bruised and tearing fabric of our society.[8,9]

VICTOR W. SIDEL, MD
Distinguished University Professor
of Social Medicine
Montefiore Medical Center/
Albert Einstein College of Medicine
Bronx, NY 10467

1. Swedish sex education and its results. *Current Sweden* 1984; no. 315.
2. Sidel VW, Sidel R: Sweden—Planned pluralism, in *A Healthy State: An International Perspective on the Crisis in United States Medical Care*, rev ed. New York, Pantheon Books, 1983, pp 118–141.
3. *Instruction Concerning Interpersonal Relations*. Stockholm, National Swedish Board of Education, 1977, p 175.
4. *Energy and Energy Policy in Sweden*. Stockholm, The Swedish Institute, 1986.
5. Buning EC, Coutinho RA, Jan Brussel GHA, et al: Preventing AIDS in drug addicts in Amsterdam. *Lancet* 1986; 1:1435.
6. HTLV-III carrier detained in Sweden. *Am Medical News*, February 7, 1986.
7. Official Compilation of Codes, Rules and Regulations of the State of New York, Title 10 (Health), Chapter 1 (State Sanitary Code), Part 24 (AIDS), Subpart 24-2 (Prohibited Facilities).
8. Sidel R, Sidel VW: Toward the twenty-first century, in Sidel VW, Sidel R (eds): *Reforming Medicine: Lessons of the Last Quarter Century*. New York, Pantheon Books, 1984, pp 267–284.
9. Sidel VW: The fabric of public health. *Am J Pub Health* 1986; 76:373–378.

Fear of AIDS*

Americans in general are still influenced by the fear of acquired immunodeficiency syndrome (AIDS). This fear, often uninformed and unwarranted, has an impact on many of the issues discussed in the papers published in this issue of the *Journal*. A number of these papers focus on the issue of sexual transmission, a subject which is of great concern. The fear of other routes of transmission persists, including those that either do not exist or, if they do, are really inconsequential in terms of the total AIDS epidemic. To some extent we have been hampered in our efforts to deal with the disease by an obsessive fear of transmission by nonsexual, "casual" contact. What appear to be really important factors in terms of human immunodeficiency virus (HIV) transmission are sexuality and substance abuse. Yet there is a great deal of difficulty in publicly and intelligently dealing with these particular aspects of basic human existence in the context of the AIDS epidemic. We are all sexual beings, and many in our society are abusers of either legal or illegal drugs. These behaviors are deeply embedded in our psychology and our culture, making them difficult to approach in such a way that behavior can be changed.

A basic issue that relates to the fear of AIDS, and which has diverted attention from concentrating on sex and drugs, is the continuing irrational belief among some that a virus that causes such a terrible disease in young people must be very easily transmitted. In fact the accumulated information, supported by the facts, indicates that the disease is not easily transmitted. Studies of both household contacts of AIDS patients[1] and hospital workers caring for AIDS patients[2] make this clear. In terms of heterosexual transmission, it is noteworthy that the majority of long-standing sexual contacts of infected individuals remain uninfected, even after hundreds of sexual encounters.[3]

The fear of AIDS is also related to the fact that we tend to concentrate on isolated transmission events rather than on the probability of transmission. The overall literature on AIDS and some of the papers in this issue of the *Journal* provide information from population-based studies that permit a determination of rates of risk.[1-5] Rather than observing that an event can occur, one should reflect on how frequently it occurs and from this begin to approach the factors that are associated with its occurrence.

We remain uninformed about the current patterns of

---

* This article is based on a presentation at the "Symposium on the Heterosexual Transmission of AIDS," sponsored by the Department of Epidemiology and Social Medicine, Montefiore Medical Center and Albert Einstein College of Medicine, and held October 21, 1986, at the New York Academy of Medicine.

HIV transmission. It we think in terms of infection and not disease, there is at least a five-year lag period in documenting and understanding transmission patterns. This is the result of the prolonged incubation period of the disease.[6] In order to understand what is currently occurring one must perform seroprevalence surveys in populations not known to engage in the traditional high-risk behaviors of intravenous drug abuse and homosexual sex. An illustration is the occurrence of female-to-male sexual transmission. Documentation of this form of transmission does not appear in AIDS case surveillance data but may be present in seroprevalence surveys. The virus first entered predominately male populations in US society—homosexual men, intravenous (IV) drug abusers, and those suffering from hemophilia. Therefore the pool of infected women able to transmit infection to men was quite small five years ago. Since the number of infected women is increasing, seroprevalence surveys among sexually active heterosexuals might disclose increasing rates of female-to-male transmission at present, whereas AIDS case surveys do not because they document the results of transmission events that occurred many years ago. However, because of the fear of AIDS and the resultant consequences of seropositivity, such as isolation and stigmatization, loss of jobs and health insurance, elimination of sexuality, and the burden of uncertainty about the future, it is difficult to perform seroprevalence surveys. There is little incentive for serologic testing for individuals. Seroprevalence surveys, contact tracing, and knowledge about who is infected and how transmission is occurring have all thereby been impeded. Further, to characterize current heterosexual transmission patterns, one must determine the rate of seropositivity among adolescents and sexually active young adults in geographic areas of extensive IV drug abuse, since IV drug abuse and heterosexual transmission are clearly linked. But this must be done with appropriate concern for confidentiality and the protection of individuals' rights.

The availability of chemotherapy in the future will substantially change the situation and make individual serologic testing valuable by enabling early treatment. If one looks at the history of sexually transmitted disease in this society in the last century, it is clear that it was only the availability of specific therapy with penicillin that made contact tracing for syphilis and gonorrhea a viable public health tool. To expect individuals to be tested, we must have something more to offer them than bad news, particularly if that news is accompanied by stigmatization and other social dilemmas. The possibility of offering a treatment in the future, with some degree of cautious optimism, might change this situation.

Finally, it is important to emphasize how essential it is to understand the behavior and the culture in which transmission occurs. The biologic model of disease or of virus infection inadequately explains transmission. In order to understand transmission, one must know what people do, how and where they do it, how frequently, and why. One must understand the culture in which these events take place in order to gain access to changing behavior. The papers by Hein[7] about adolescent sexuality and Des Jarlais[8] about prostitution make this clear. The same detailed understanding of patterns of IV drug use is essential to influence behavior.

To interrupt the transmission of HIV, information and education remain the most important tools.

GERALD FRIEDLAND, MD
Associate Professor
Department of Medicine
Department of Epidemiology and
Social Medicine
Montefiore Medical Center/
Albert Einstein College of Medicine
Bronx, NY 10467

1. Friedland GH, Saltzman BR, Rogers MF, et al: Lack of transmission of HTLV-III/LAV infection to household contacts of patients with AIDS or AIDS-related complex with oral candidiasis. *N Engl J Med* 1986; 314:344-349.
2. McCray E: The cooperative needlestick surveillance group, occupational risk of acquired immunodeficiency syndrome among health care workers. *N Engl J Med* 1986; 314:1127-1132.
3. Saltzman BR, Harris CA, Klein RS, et al: HTLV-III/LAV infection and immunodeficiency in heterosexual partners of AIDS patients. Presented at the International Conference on AIDS, Paris, France, June 1986.
4. Burke DS, Brundage JF, Bernier W, et al: Demography of HIV infections among civilian applicants for military service in four counties in New York City. *NY State J Med* 1987; 87:262-264.
5. Quinn TC: AIDS in Africa: Evidence for heterosexual transmission of the human immunodeficiency virus. *NY State J Med* 1987; 87:286-289.
6. Lui KJ, Lawrence DN, Morgan WM, et al: A model-based approach for estimating the mean incubation period of transfusion-associated acquired immunodeficiency syndrome. *Proc Nat Acad Sci* 1986; 83:3051-3055.
7. Hein K: AIDS in adolescents: A rationale for concern. *NY State J Med* 1987; 87:290-295.
8. Des Jarlais DC, Wish E, Friedman SR, et al: Intravenous drug use and the heterosexual transmission of the human immunodeficiency virus: Current trends in New York City. *NY State J Med* 1987; 87:283-286.

Demography of HIV infections among civilian applicants for military service in four counties in New York City*

DONALD S. BURKE, MD; JOHN F. BRUNDAGE, MD; WILLIAM BERNIER, MD; LYTT I. GARDNER, PHD; ROBERT R. REDFIELD, MD; JEFFREY GUNZENHAUSER, MD; JAMES VOSKOVITCH, MD; JOHN R. HERBOLD, DVM

ABSTRACT. During the period October 1, 1985, through July 31, 1986, serum specimens from 9,498 civilian applicants for military service from four New York City counties (New York, Kings, Queens, and Bronx) were tested for antibodies to the human immunodeficiency virus (HIV). Ninety-seven (1.03%) specimens were positive as confirmed by Western blot. Antibody prevalence was strongly associated with age. Among recruit applicants who were less than 18, 18–21, 22–25, and greater than 25 years old, HIV seroprevalence rates were 0.23%, 0.31%, 1.30%, and 2.95%, respectively. Among applicants of different racial groups, the rates of seroprevalence were as follows: whites, 19/2,553, 0.74%; blacks, 56/4,869, 1.15%; and others including Hispanic, 22/2,076, 1.06%. Rates among male applicants (84/7,938, 1.06%) and female applicants (13/1,560, 0.83%) were not significantly different ($p = 0.45$).

(NY State J Med 1987; 87:262–264)

In October 1985 the US Department of Defense instituted a program for screening all civilian applicants for military service for evidence of infection with HIV. Data for the first six months of the program for the entire United States and its territories are summarized elsewhere.[1] Of the 14 counties with the highest seroprevalence rates in the continental United States, four were contiguous counties in the New York City area (New York, Kings, Queens, and Bronx). This report is a preliminary summary of current data on demographic factors associated with HIV infection in these four counties.

PATIENTS AND METHODS

Beginning October 1, 1985, blood samples for HIV testing were obtained as part of the medical examination conducted at 71 Military Entrance Processing Stations within the United States and its trust territories. Details of the methods used to detect HIV antibodies and to process demographic data are presented elsewhere (D.S. Burke, J.F. Brundage, W. Bernier, et al, unpublished manuscript, 1987). In brief, serum samples were screened by a contracting laboratory (Damon Laboratories) with a commercial HIV enzyme-linked immunoassay system (ELISA, Electronucleonics, Inc). All repeatable, positive samples were subsequently tested by Western blot. Blots were determined to be positive if antibodies to gp41 and/or p24+p55 were detected. Applicants who had positive samples were notified by a registered letter which also requested that they return to the examination station for counseling and repeat Western blot testing. In this report, applicants whose samples tested positive on a repeat blot, and applicants whose samples were positive on the first blot but did not submit a second specimen are considered antibody positive. All other applicants, including those positive by ELISA but negative by blot, are considered antibody negative. Data are analyzed for the ten-month period October 1, 1985–July 31, 1986. Data were tested for statistical significance using chi-square analysis for contingency tables and chi-square for assessing a linear trend.

RESULTS

Seroprevalence rates among civilian applicants for military service in the counties of New York, Kings, Queens, and Bronx are presented by county in Table I. Overall, 97 of 9,498 applicants were HIV seropositive (10.3/1,000). Rates by county ranged from 4.2/1,000 (10/2,400) in Queens to 17.1/1,000 (24/1,404) in Manhat-

TABLE I. HIV Seroprevalence Rates Among Military Recruit Applicants in Four Selected New York Counties, October 1, 1985–July 31, 1986

County	No. Positive	No. Tested	(Rate per 1,000)
New York	24*	1,404	17.1†
Kings	36	3,382	10.6
Queens	10	2,400	4.2
Bronx	27	2,312	11.7
Totals	97	9,498	10.3

* HIV seropositive as determined by Western blot.
† Rate HIV Western blot positive per 1,000 tested.

From the Walter Reed Army Institute of Research (Drs Burke, Brundage, Gardner, Redfield, and Gunzenhauser), the US Military Entrance Processing Command (Drs Bernier and Voskovitch), and the Office of the Assistant Secretary of Defense (Health Affairs) (Dr Herbold).

Address correspondence to Col Donald S. Burke, Department of Virus Diseases, Walter Reed Army Institute of Research, Washington, DC 20307-5100. Requests for reprints should be addressed to the Division of Academic Affairs, Walter Reed Army Institute of Research, Washington, DC 20307-5100.

The views of the authors do not reflect the positions of the Department of the Army or the Department of Defense.

*This article is based on a presentation at the "Symposium on the Heterosexual Transmission of AIDS," sponsored by the Department of Epidemiology and Social Medicine, Montefiore Medical Center and Albert Einstein College of Medicine, and held October 21, 1986, at the New York Academy of Medicine.

TABLE II. HIV Seroprevalence Rates Among Military Recruit Applicants in New York, Kings, Queens, and Bronx Counties, by Age, Sex, and Racial Group, October 1, 1985–July 31, 1986

	White		Black		Other		All Racial Groups	
Age (yr)	M	F	M	F	M	F	M	F
<18	0/115[*]	0/6	0/124	1/41	0/104	0/11	0/343	1/58
	0.0[†]	0.0	0.0	24.4	0.0	0.0	0.0	17.2
18–21	3/1,328	0/104	6/2,048	3/582	4/1,015	0/110	13/4,391	3/796
	2.3	0.0	2.9	5.1	3.9	0.0	3.0	3.8
22–25	3/495	2/57	15/896	2/255	5/381	1/61	23/1,772	5/373
	6.1	35.1	16.7	7.8	13.1	16.4	13.0	13.4
>25	11/389	0/59	25/700	4/223	12/343	0/51	48/1,432	4/333
	28.3	0.0	35.7	17.9	35.0	0.0	33.5	12.0
Totals	17/2,327	2/226	46/3,768	10/1,101	21/1,843	1/233	84/7,938	13/1,560
	7.3	8.8	12.2	9.1	11.3	4.3	10.6	8.3
	19/2,553		56/4,869		22/2,076		97/9,498	
	7.4		11.5		10.6		10.3	

[*] Number HIV Western blot positive/number tested.

[†] Rate HIV Western blot positive per 1,000 tested.

tan. Rates among applicants in these four counties in demographic subcategories of age, racial group, and gender are presented in Table II.

Among applicants who were less than 18, 18–21, 22–25, and greater than 25 years old, HIV seroprevalence rates were 2.3, 3.1, 13.0, and 29.5 per 1,000, respectively. Among applicants of different racial groups, the rates were as follows: whites, 19/2,553, 7.4/1,000; blacks, 56/4,869, 11.5/1,000; and others including Hispanic, 22/2,076, 10.6/1,000 ($p = 0.24$).

HIV seroprevalence among male applicants (84/7,938, 10.6/1,000) was only marginally greater than among female applicants (13/1,560, 8.3/1,000) ($p = 0.45$). Male and female seroprevalence rates were generally similar among each racial group: white males, 7.3/1,000 versus white females, 8.8/1,000 ($p = 0.88$); black males, 12.2/1,000 versus black females, 9.1/1,000 ($p = 0.49$); other racial group males, 11.3/1,000 versus other racial group females, 4.3/1,000 ($p = 0.51$).

When analyzed by age, male and female HIV seroprevalence rates were similar for applicants younger than 26 years (males, 36/6,506, 5.5/1,000; females, 9/1,227, 7.3/1,000) ($p = 0.31$). Among applicants 26 years old or older, HIV seroprevalence rates were greater among males (48/1,432, 33.5/1,000) than among females (4/333; 12.0/1,000) ($p = 0.06$). Overall, there was a significant trend of increasing prevalence by age in males ($p < 0.001$) but not in females ($p = 0.21$).

DISCUSSION

Although there are considerable data regarding the demography of AIDS cases in the United States,[1] to date there has been a dearth of comparable information about the early stages of infection with HIV, the causative agent of AIDS. Studies on HIV antibody prevalence have largely been confined to selected groups in locations with high AIDS incidence rates, such as male homosexuals in San Francisco (67% positive) or intravenous drug abusers in New York City (87% positive).[2,3] Data gathered from blood bank screening programs, where persons considered to be at high risk of infection are actively discouraged from donating, present the opposite extreme (0.04% positive).[4]

Data from the US Military Entrance Processing screening program provide new perspectives on the prevalence of HIV infection and the geographic and demographic factors associated with HIV infection. During the first six months of the program, more than 300,000 civilian applicants for military service were tested at 71 stations in the US and its territories.[1] HIV seroprevalence rates among recruit applicants in specific geographic localities were found to correlate closely with cumulative AIDS incidence rates in the general population in those localities. The overall seroprevalence rate among recruit applicants in the United States was 1.5/1,000. Seroprevalence rates among applicants from the New York City area were substantially higher than rates throughout the rest of the country.

For the current report, we analyzed data on HIV seroprevalence among applicants from four contiguous counties in New York City which had unusually high HIV infection rates. In these four counties, more than 1% of all applicants had serologic evidence of HIV infection. As was noted for the US at large, seropositivity was closely associated with increasing age. However, rates for any specified age group were four to ten times greater in these four counties than in the rest of the United States.

In this analysis we did not detect substantial differences in HIV seroprevalence rates between different racial groups. This finding differs from that for the nation at large, where black applicants have a 3.6-fold higher rate and applicants of other racial groups a 1.5-fold higher rate than white applicants (D.S. Burke, J.F. Brundage, W. Bernier, et al, unpublished data, 1987).

HIV seroprevalence rates among men and women in these four counties were surprisingly similar, suggesting that infection is occurring in the male and female populations at comparable rates. This observed sex ratio of close to 1:1 differs substantially from the ratio of 13:1 reported for AIDS cases.[1] When the male-to-female infection ratio was analyzed according to age or racial groups, rates were comparable in all subcategories except for applicants over the age of 25 years, for which the male-to-female ratio was 2.7:1.

We interpret these data to show that in the four counties in central New York City, HIV infections are highly prevalent among young adults. Risk of infection is not confined to any racial group and is not appreciably greater for men than for women, at least under age 26. Data on the probable mode of infection for each HIV-infected recruit applicant is not currently available. Although exposure through intravenous drug abuse cannot be ruled out, the high rates of infection observed among women suggest that heterosexual relations may be a significant mode of transmission in urban centers in the United States.

Young persons who apply for entry into the military may not be representative of the population at large. Nonetheless, these data suggest the possibility of a substantial change in the epidemiology of AIDS in the New York area within the next five to ten years.

REFERENCES

1. Peterman TA, Drotman DP, Curran JW: Epidemiology of the acquired immunodeficiency syndrome (AIDS). *Epidemiol Rev* 1985; 7:1-21.
2. Jaffe HW, Feorino PM, Darrow WW, et al: Persistent infection with human T-lymphotropic virus type III/lymphadenopathy-associated virus in apparently healthy homosexual men. *Ann Intern Med* 1985; 102:627-628.
3. Spira TJ, Des Jarlais DC, Marmor M, et al: Prevalence of antibody to lymphadenopathy-associated virus among drug-detoxification patients in New York [letter]. *N Engl J Med* 1984; 311:467-468.
4. US Public Health Service Workshop on human T-lymphotropic virus type III antibody testing. *MMWR* 1985; 34:477-478.

Local policy responses to the AIDS epidemic: New York and San Francisco

PETER S. ARNO, PHD, ROBERT G. HUGHES, PHD

ABSTRACT. **The epidemic of acquired immunodeficiency syndrome (AIDS) has been concentrated in a few large cities. Thus far, New York City and San Francisco have reported more AIDS cases than any other cities in the world. Together they account for 40% of the total number of cases reported in the United States through the end of 1986.**

Both cities have expended an enormous amount of local resources in dealing with the epidemic, although the public policy response has been markedly different in each city. Although historically New York has had three times the AIDS caseload as San Francisco, it has consistently spent less money on public health education and other nonhospital-related health care services. The varied policy responses in each city can be attributed to several factors: differences in the magnitude of the epidemic; the patient mix; the role of risk groups in the political and economic life of each community; the scale of public health care systems, including the number of medical schools; the impact of local media; and the institutional roles of the respective health departments.

With the prospect of other cities facing an increasing number of AIDS cases, the New York and San Francisco experiences may prove useful. They indicate that each community's response to AIDS will probably reflect the underlying social, economic, and political characteristics of AIDS victims and the existing structure and organizational roles of traditional health care and community-based service providers.

(NY State J Med 1987; 87:264-272)

Forty percent of the AIDS cases reported in the United States through 1986 have occurred in New York City and San Francisco. These two cities account for more cases than any other cities in the world. Yet the ways in which the epidemic has affected these two cities and their policy responses have been quite different. Early reports indicated that the average length of hospital stay for AIDS patients was four times longer in New York, and that services for AIDS patients, ranging from hospital care to housing and counseling, were organized differently in each city.[1-4]

The concentration of cases in these two cities, coupled with a federal policy preference for shifting responsibility for domestic social problems from the national to the local level, has made the experiences reported here important to an understanding of AIDS in the United States. This paper describes the AIDS epidemic in New York and San Francisco and examines the factors that have contributed to different policy responses in the two cities. This comparison illuminates the problems other cities are likely to face as AIDS continues to spread, and identifies those factors that are important in tailoring local responses to the epidemic.

To investigate the apparent differences between the two cities, we relied on a variety of data sources as well as the organizations that have been involved in coping with the epidemic in New York and San Francisco. We reviewed available published information, including congressional testimony, scientific literature, and newspaper reports, and also unpublished agency minutes and records from involved organizations. Finally, extremely valuable information was provided by numerous public officials, leaders of private AIDS-related organizations, and volunteers from agencies in both cities who discussed the responses to AIDS in their communities.

To place these cities in perspective, it is first useful to describe some of the basic economic and demographic characteristics of each city (Table I). For example, New

---

From the Department of Health Care Administration, Bernard M. Baruch College/Mount Sinai School of Medicine, City University of New York (Dr Arno), and the School of Health Administration and Policy, Arizona State University (Dr Hughes).

Address correspondence to Dr Arno, Assistant Professor, Department of Health Care Administration, Bernard M. Baruch College/Mount Sinai School of Medicine, 17 Lexington Ave, New York, NY 10010.

This research was supported in part by fellowship grants from the Pew Memorial Trust. Earlier versions of this paper were presented at the Annual Meetings of the American Public Health Association, November 1985, in Washington, DC, and at the International Conference on AIDS in Paris, France, in June 1986.

TABLE I. Demographic and Economic Characteristics: New York City and San Francisco

	New York City	San Francisco
Total population (1,000) (1982)	7,086	692
% Black (1980)	25.2	12.7
% Hispanic (1980)	19.9	12.3
Per capita income (1981)	$8,737	$11,026
Median family income (1979)	$19,268	$24,648
% Population below poverty level (1979)	19.7	13.4

Sources: US Department of Commerce: *Statistical Abstract of the United States*, 1985; US Department of Commerce: *1980 Census of Population*, 1983.

York is a far larger city, with ten times the number of people as San Francisco. Nearly twice the proportion of New York's population is black or Hispanic. San Francisco is a wealthier city, with a per capita income 26% higher than New York's, and fewer of its residents live in poverty. These general demographic and economic factors are reflected in the AIDS patient groups in each city and have influenced local policy responses to the epidemic.

NEW YORK AND SAN FRANCISCO: DIFFERENCES IN RESPONDING TO AIDS

New York and San Francisco have responded differently to the AIDS epidemic in the amount of money spent, the distribution of these funds among programs, the length of hospital stays of AIDS patients, and the array of services available. Initial policy attention, influenced by the predominant interest in controlling health care costs in this country, was directed at the cost of care for AIDS patients in the two communities.

Precise estimates of public spending on AIDS programs during the early years of the epidemic are difficult to assess, particularly in New York, where AIDS expenditures were not tracked and programs were split among several city agencies. These figures are therefore less reliable. In addition, figures describing the amount of public money spent on AIDS do not always include the local share of Medicaid funds spent at nonpublic hospitals. Thus, it is important to recognize not only the politically sensitive budget process in which these figures are often used, but that the figures may not be strictly comparable from year to year or from one city to another. Nevertheless, a sense of the magnitude of the resources devoted to AIDS can be ascertained from agency estimates of their own expenditures.

In 1985, the acting president of the New York City Health and Hospitals Corporation (NYCHHC, the municipal hospital system) estimated that $30 million was spent on inpatient health care delivery for AIDS patients in this public hospital system during fiscal year (FY) 1985.[5] During this time, the New York City Department of Health expended an additional $1.1 million on AIDS-related activities. These consisted mainly of disease surveillance and epidemiologic investigation, health education, and laboratory services.[6] Also in New York, the Human Resources Administration spent $600,000 for social services for persons with AIDS. In total, AIDS expenditures of approximately $32 million have been identified for New York during FY 1985, but this fails to include public funds used to subsidize indigent care at private hospitals and a variety of other AIDS-related expenditures.

Expenditures of an estimated $25 million dollars for AIDS-related activities were made in San Francisco during FY 1985. The bulk of these funds ($17 million) was for inpatient care, which was financed primarily through private insurance and the Medi-Cal program.[7] The remaining $8 million was administered by the San Francisco Department of Public Health (SFDPH), more than 90% of which was derived from local taxes. Approximately 17% of these funds went for disease surveillance and epidemiologic investigations, 55% for assessments and medical care, 11% for social support services and housing, and 17% for public education and administrative coordination.[8]

As the epidemic grew, the expenditure of funds significantly increased in New York. An attempt was made by the New York City Office of Management and Budget to analyze the total amount and distribution of AIDS-related funds during FY 1986. Total spending in New York City was projected to range from $110 million to $148 million. New York City's tax levy support was estimated at approximately 38% of these figures. This included the city's contribution to inpatient medical care provided in municipal and voluntary hospitals, public health education, epidemiologic surveillance, and contracts for social service programs. The remaining balance included all other sources of reimbursement including federal and state funds and private insurers.[9]

This compares to a projected $37 million for total AIDS spending in San Francisco during this time (based on estimates).[8,10] Approximately 24% of this sum ($8.8 million) was derived from local tax revenues and was distributed to a similar range of activities as in the previous year. The remaining funds were primarily drawn from private insurers (37%) and the federal and state governments (18% and 13%, respectively).

By estimating the number of living persons with AIDS in each city during FY 1986, we calculated a per capita expenditure per AIDS patient: approximately $22,000 in San Francisco and $36,000 in New York.

A major part of overall costs in both cities is for inpatient care. The average daily cost of care in their municipal hospitals is quite similar, with estimates of $773 per day in San Francisco and $800 per day in New York—less than a 4% difference.[9,11] Yet the charges per admission are quite different: $20,320 in New York and $9,024 in San Francisco. As Table II indicates, the key difference in these charges is the number of days spent in the hospital.

The average length of stay for an AIDS patient in New York is more than twice as long as that in San Francisco.

TABLE II. Hospitalization of AIDS Patients in Municipal Facilities, 1984: New York City and San Francisco

	New York City	San Francisco
Average length of stay (days)	25.4	11.7
Average charges per day ($)	800	773
Average charges per admission ($)	20,320	9,024

Sources: Based on data derived from references 9 and 11.

In addition, it appears that at least twice the proportion of AIDS patients are hospitalized at any given time in New York as in San Francisco. According to a 1985 survey,[5] approximately 22% of all living AIDS patients in New York were hospitalized. This compares to approximately 10% in San Francisco.[7] This finding is consistent with longer hospital stays in New York in general. Reasons for regional variations in length of stay, a pattern confirmed by AIDS patients, have been addressed elsewhere.[12]

A final difference between the two cities is in the earlier development in San Francisco of a relatively coordinated set of services for AIDS patients and citizens in major risk groups—from outpatient care, to housing and counseling, to prevention through community education. Not only were these services developed earlier in San Francisco, they were strongly supported by local public funds. This contrasts with the situation in New York in 1984, where one organization, the Gay Men's Health Crisis (GMHC), provided the bulk of AIDS-related community services. Only 3% of GMHC's resources were provided by New York City.[13] In contrast, the three largest organizations providing similar services in San Francisco—the Shanti Project, the San Francisco AIDS Foundation, and Hospice of San Francisco—received 62% of their financial resources from San Francisco City and County.[14]

EXPLAINING THE DIFFERENCES

Exploring possible reasons for the differences between the two cities, we found that they fall into three sets: epidemiologic factors, preexisting social and political structural characteristics, and policy responses in local agencies.

Many of these factors are related. Epidemiologic factors and the social organization of the risk groups, for example, influence each other, and both have important effects on policy responses. Thus, although we are presenting the three sets of factors separately, it should be recognized that they are related to each other within each city.

Epidemiologic Factors. Available data on the epidemiology of AIDS in New York, San Francisco, and the United States are summarized in Table III. The figures for the United States incorporate data for the two cities and are presented for reference. New York and San Francisco account for 30.4% and 9.5%, respectively, of the nation's AIDS cases.

Significant differences between the two cities can be seen in the total number of cases, the rate of cases in the population, the proportion of patients in various risk groups, the race and sex of patients, and the number of cases in children. Although New York has historically had more than three times as many AIDS cases as San Francisco, the rate of cases per 100,000 population is three times as great in San Francisco. This difference in the rate of cases between the two cities differs from the Centers for Disease Control (CDC) data. Their rates indicate a difference of less than 4% between New York and San Francisco.[15] Two reasons explain the differences in the rates we report and those of the CDC. First, the CDC uses Standard Metropolitan Statistical Areas (SMSAs) to measure the underlying population base, while we used data only for the cities. Second, the CDC data are based on AIDS cases by place of residence, and ours are by place of diagnosis. We chose these methods because our focus is on comparing the impact of the epidemic on the health care systems in these two cities.

The lower percentage of men affected in New York is due primarily to the larger number of affected intrave-

TABLE III. Reported AIDS Cases Through December 1986: New York City, San Francisco, and the United States

	New York City	San Francisco	United States
Total cases	8,847	2,760	29,137
% of US cases	30.4	9.5	100
Cases/100,000 population[*]	124.9	398.8	12.7
% Cases male	88.3	99.1	92.6
% Cases by race/ethnicity:			
White	44.8	86.2	59.6
Black	31.2	5.8	25.0
Hispanic	23.4	6.6	14.4
Other/unknown	0.6	1.3	1.0
Number of pediatric cases[†]	166	7	410
% Cases by transmission category:			
Gay or bisexual men[‡]	61.1	97.0	72.2
Intravenous (IV) drug users	29.1	1.2	16.8
Transfusion with blood or blood products	0.9	0.8	2.0
Hemophilia/coagulation disorder	0.2	0.1	0.9
Heterosexual contact[§]	2.1	0.4	3.8
Child of parent with AIDS or at increased risk for AIDS	1.7	0.1	1.1
None of the above/other	4.9	0.4	3.2
Total	100	100	100

Sources: New York City Department of Health, Office of Epidemiologic Surveillance and Statistics; San Francisco Department of Public Health, Bureau of Communicable Disease Control; Centers for Disease Control, US AIDS Program, Center for Infectious Diseases.

[*] Cases/100,000 are based on 1982 populations for each city.

[†] Pediatric cases refer to patients under 13 years of age at time of diagnosis.

[‡] Eight, 13, and 11 percent of gay and bisexual men in New York, San Francisco, and the United States, respectively, also reported having used IV drugs.

[§] Heterosexual contact refers to sexual relations with a person with AIDS or at risk for AIDS.

nous (IV) drug users in that city, compared to the predominance of gay men in San Francisco. Female IV drug users and women infected by their sexual partners who are IV drug users represent a much larger proportion of AIDS cases in New York than in San Francisco.

AIDS cases also vary significantly by race and risk group. San Francisco's AIDS patients are more likely to be white, whereas New York's are more likely to be black or Hispanic. This reflects general demographic differences in these cities. Compared to all AIDS patients in the US, patients in San Francisco are much more likely to be homosexuals, whereas patients in New York consist of a larger proportion of IV drug users.

Heavy incidence among IV drug users affects another patient group—children. In New York, where IV drug use and sharing of contaminated needles is more common than in the rest of the country, 79% of children with AIDS were presumably infected during pregnancy by mothers who were either IV drug users themselves or who had been infected by IV drug users.[16] The proportion of IV drug-related AIDS cases among children is likely to rise because of the continued spread of AIDS among drug users and the decline in the number of cases transmitted through contaminated blood and blood products (as a result of improved screening of the nation's blood supply).

Two patient groups remain hospitalized longer than others—IV drug users and children with AIDS. Both groups are concentrated in New York City. There have been 2,570 AIDS cases among IV drug users and 166 cases of pediatric AIDS reported in New York City through 1986. This represents 52% and 40% of the nation's cases in the respective groups. Only 33 IV drug-related and seven pediatric cases were reported in San Francisco during this time. (The figures for both cities do not include approximately 8–13% of cases among homosexual or bisexual men who also report using IV drugs.)

The municipal hospitals in New York are overwhelmed with IV drug users, a group which comprises 60% of all AIDS patients hospitalized there.[9] These patients are generally indigent, have poor underlying health status, have fewer sources of social support, and are more likely to be homeless than other patient groups. The primary AIDS diagnosis for many IV drug users is *Pneumocystis carinii* pneumonia (PCP). In general, this opportunistic infection is more debilitating and requires longer and more frequent hospital stays than other AIDS-related conditions, the most common of which is Kaposi's sarcoma, which is often treated on an outpatient basis.[17] In a study of AIDS patients at San Francisco General Hospital (SFGH), the average length of stay for AIDS patients with Kaposi's sarcoma was 7.6 days, compared to 18.1 days for those with PCP.[11] Furthermore, during 1984, Kaposi's sarcoma accounted for 13.3% of the total AIDS admissions at this hospital, as compared to only 3.5% of AIDS admissions in the public hospitals in New York City.[9] All these factors increase the likelihood of a longer hospital stay and an increased direct cost of care for AIDS patients in New York.

Hospitalization of children with AIDS has not been a major problem in San Francisco, since there have been so few cases. New York, by contrast, has had to address issues arising from pediatric AIDS, including the unusually long length of stay for these patients. As discussed above, more than three quarters of the pediatric AIDS patients in New York have parents who are or have been IV drug users. Many of these parents may be ill, and some have died from AIDS. In addition, there is a lack of alternative facilities to care for children outside the hospital, and it has been extremely difficult to place children with AIDS in foster care. Again, this is consistent with the situation confronting adult AIDS patients, who, in New York's tight housing market, also face difficulty in finding available space in nursing homes, and thus are likely to have prolonged hospital stays. A one-day survey conducted in New York's municipal hospitals found nine pediatric AIDS patients awaiting nonhospital placement. All were homeless, and all were candidates for home care. Three of these patients had been on an alternate level of care status (ie, those patients who no longer require acute care hospital services and are ready for placement in nonhospital settings) for 12, 14, and 18 months.[18] Thus, overall, the AIDS epidemic is quite different in New York than in San Francisco. New York is confronted with approximately three times the number of cases, and the patients are more heterogeneous than those in San Francisco with respect to race, age, sex, income, and major risk factors. In effect, the differences in scale and diversity of patients in New York, when compared to San Francisco, present local policy makers with two quite different epidemics of the same disease.

Preexisting Social and Political Structures. The local responses to the epidemic did not occur in a vacuum. They grew out of and were influenced by the social and political structures already existing in each community. Each city's historical development—in size, number, and type of institutions providing health services; methods of payment for health care; and the roles of public agencies—set the stage for what could happen in the epidemic. We differentiate two components in the preexisting social and political structures: one is related to the community and its institutions, especially the structure of health and human service organizations; the other is related to the social and political structures of those persons in the major risk groups.

The scale and complexity of health and human service organizations is far greater in New York than in San Francisco. The difficulties associated with a larger scale in New York range from the simple logistics of epidemiologic case monitoring to the institutional complexities of developing a response in 11 acute care municipal hospitals and over 70 other private and voluntary hospitals scattered unevenly throughout five boroughs. This contrasts with the one city health care facility and 13 other hospitals in San Francisco. In addition, the existence of a single medical school in San Francisco has fostered a relatively unified response to the epidemic, while in New York there is a history of competition in selected areas among the seven medical schools, making a similar response much more difficult.

The cities are similar in having a complex role in the financing of health care for part of their populations. These roles have been illuminated by the AIDS epidemic and the enormous resources needed to cope with it. One of the major differences in the two cities is how local tax monies have been allocated. One report estimated that more than 90% of New York City AIDS funds are spent on inpatient hospital care, largely through the city government's 25% share of Medicaid expenditures, and direct subsidies to the NYCHHC and voluntary hospitals providing indigent care.[19] This results in sizable expenditures of local funds in New York because of the large number of AIDS patients who are enrolled in the Medicaid program. In comparison, in San Francisco only approximately 25% of municipal AIDS funds are spent on inpatient care. The large difference is due in part to San Francisco's local government not contributing to the Medicaid program. This reduces the cost of hospitalization to the city, while increasing the cost to the state of California.

These differences point out the importance of local-state relationships in responses to AIDS, especially in funding. In New York, the state health department has increased its influence vis-a-vis the New York City Health Department through its statutory responsibilities in Medicaid and in voluntary hospital financing in general. In addition, following the fiscal crisis of the 1970s, the state assumed other responsibilities that were formerly the province of city agencies. For example, administrative control over substance abuse programs was shifted to the state during the late 1970s, and it is these agencies that are trying to cope with the growing epidemic among IV drug users.[20]

Both cities help pay for the costs of hospitalizing medically indigent patients who are not covered by any third-party payers. This has been an especially acute problem with AIDS patients, many of whom are treated with experimental drugs because no efficacious drug regimen is available. Third-party payers, both private and public, have been reluctant to pay for hospital care when experimental drugs are used in treatment. Thus, both local governments have been forced to subsidize treatment costs. Even more important for the cities' budgets is the fragmented mix of policies that have resulted in a large number of AIDS patients joining millions of other Americans who "fall through the cracks" and have inadequate health insurance coverage. It is estimated that more than 55 million people in this country have little or no health insurance at all.[21] These patients, who are generally confined to municipal hospitals, are subsidized by local tax revenues in both cities.

The payer mix for AIDS patients in the municipal hospitals of each city is quite similar (Table IV). In the municipal hospitals, those patients not covered by private insurance, Medicaid, or Medicare usually have no other insurance coverage and are subsidized by local government. In San Francisco's nonmunicipal hospitals, Kaiser Permanente, a health maintenance organization, covers at least 50% of those patients in the "other" category, reducing the overall level of uncompensated care to approximately 13% of the total AIDS inpatient caseload in that city.

TABLE IV. Percent of AIDS Patient Discharges by Payer Source: San Francisco and New York Hospitals, 1985

	Medicaid	Medicare	Private Insurance	Other
San Francisco				
Municipal hospital	64%	2%	17%	17%
Nonmunicipal hospital	16	2	57	25
New York City				
Municipal hospital	65	1	13	21
Nonmunicipal hospital	45	3	36	16

Sources: Based on data derived from references 9 and 10 and from Kaufman and Kelly: *AIDS Reports from New York City*, memorandum to Steve Anderman, Deputy Director, Division of Health Care Financing, New York State Department of Health, December 10, 1985.

The budgets of the cities in the years preceding the epidemic also may have influenced the extent to which each committed financial resources to the epidemic. The ability of local government in San Francisco to respond to the AIDS crisis with substantial funds as early as 1983 was made easier by the existence of a budget surplus that existed during the early 1980s (but is now gone). In contrast, New York was just coming out of a protracted period of financial crisis and near bankruptcy in the late 1970s.

Another factor that existed before the epidemic and influenced local responses was the different roles of the local health departments. In San Francisco, the health department incorporated traditional public health functions, outpatient clinics, and San Francisco General Hospital under one bureaucracy. This strengthened local public health decision makers in their position that the health department should take the lead in responding to the AIDS epidemic through direct action and active coordination, funding, and support of other organizations. In New York, the health department's functions were separate from hospital services, which fell within the bureaucracy of the NYCHHC. Thus, the health department in general "should provide those services that others have not, will not, should not, or cannot provide," [22] ie, its role is to fill the gaps in the system. In an early response to the AIDS epidemic, the department called a meeting of all interested parties in the city and defined its role as "seeking not to direct, but to provide a neutral meeting ground." [22]

A further difference lies in the difficulties of coordinating the diversity of services that can be used to address the AIDS epidemic. In New York, three large, separate city bureaucracies, the Health Department, the Health and Hospitals Corporation, and the Human Resources Administration, each developed programs to deal with AIDS, with little formalized central coordination. In San Francisco, these functions were under the authority of one agency, and this allowed extensive health department organizational resources to be used to encourage the integration of community-based, ambulatory, and institutional programs.

The preexisting social and political structures of persons in the major risk groups are a second component that helps explain the differences in the two cities. In San Francisco, AIDS has predominantly affected white, middle-class, homosexual men. In New York, at least half the cases are reported among minorities, and almost a third are known IV drug users—an impoverished, highly stigmatized, and politically powerless stratum of society.

In a 1984 survey, the number of openly homosexual or bisexual men in San Francisco was estimated to be approximately 69,000, or 24% of all men aged 15 years and older.[23] The homosexual community was also found to be relatively affluent and well-educated, with 44% having pre-tax earnings of $25,000 or more, 57% having college degrees, and 21% having postgraduate degrees. A preliminary survey in New York indicates a similar socioeconomic background for homosexual men.[24] However, given the distribution of cases in other patient groups (eg, IV drug users) and by race, AIDS has afflicted a poorer and less educated population in New York.

The roles of the risk groups in the economic and political life of these communities are markedly different. The homosexual community is a well-organized, visible, and powerful political force in San Francisco, with openly gay representatives holding elective office in local government since the late 1970s. There is no comparable group of gay political power in New York. Thus, in San Francisco, the major identified risk group has been represented by organized spokesmen who were an integral component of the local political establishment even before the AIDS epidemic began.

Such visibility and political power for the major risk group in San Francisco is probably reflected in the differences in newspaper coverage of AIDS by *The New York Times* and *The San Francisco Chronicle.* Although these two papers play somewhat different roles, in that the *Chronicle* is viewed as more locally oriented than the *Times* (and therefore would be expected to put more emphasis on a locally important issue), the more extensive AIDS coverage by the *Chronicle* is still noteworthy. Between June 1982 and June 1985, 442 articles about AIDS appeared in the *Chronicle*, of which 67 were front-page stories. This compares to 226 articles published in the *Times*, of which seven were page-one news. The extensive nature of coverage by the *Chronicle*, aside from providing a degree of health education not found in New York, helped to sustain a level of political pressure on local government and health officials to respond to the AIDS crisis. The difference in coverage is also probably a reflection of the relative importance of the AIDS epidemic among other local issues. In San Francisco, AIDS was clearly a top issue; in New York, it had to compete for attention with a multitude of other issues.

Thus, the preexisting social and political structures of the institutions and major risk groups reinforced the differences in the epidemiology of AIDS in the two cities. In New York, the complexity of coping with AIDS was heightened by the heterogeneity of the risk groups and their diffuse political and social organization, by the number and diversity of organizational actors in the health and human services sector, by the roles of public agencies in financing health care and in responding to the epidemic, and by the relative importance of AIDS as a local policy issue. In San Francisco, on the other hand, both the epidemiology and the social and political factors put fewer constraints on those agencies that responded to the epidemic. Indeed, the conditions were that a well-educated, highly organized, and politically active group of citizens were at risk of AIDS in a city with relatively well-coordinated relationships among the major public health care institutions. The epidemiology and the social and political factors contributed, directly and indirectly, to some of the differences between the two cities through their influence on the cities' policy responses.

Policy Responses in New York and San Francisco. In both cities, the AIDS epidemic has generated a largely unanticipated need for a variety of medical, public health, social, and educational resources. For example, active disease surveillance and epidemiologic investigations have been necessary to track the epidemic, to establish routes of transmission, and to plan for future health care resources to service the needs of those afflicted. AIDS patients require hospital care, outpatient services, social support and, in many cases, housing and food. Members of high-risk groups need public health education, counseling, and specific guidelines regarding behavior to prevent transmission of the disease. The population outside the main risk groups needs education to understand the risks and how the disease is transmitted in order to reduce unwarranted fears, to promote support for effective programs, and to prevent further spread of the epidemic. The policy responses in the two cities, as reflected in agency behavior, followed paths that were consistent with the differences in epidemiologic factors and social and political structures.

In 1982, the San Francisco Department of Public Health (SFDPH), through its Office of Lesbian and Gay Health, began to coordinate efforts to plan and develop services to deal with AIDS. By 1983, in efforts to avoid duplication of services by a growing number of organizations and to coordinate the city and county's responses, a separate AIDS Activity Office was established within the SFDPH with the following specific purposes: to better coordinate and link the continuum of services related to the AIDS epidemic; to identify service gaps and develop plans for addressing these needs; to oversee, monitor, and support AIDS-related contract services; to anticipate funding requirements and justify new funding requests; and to develop and maintain the department's liaison with certain external funding and service agencies, community groups, and education programs. However, significant local funding for AIDS programs in San Francisco did not begin until FY 1984, when expenditures increased to $4.3 million from $180,000 the previous year.[8]

During the early years of the epidemic in New York (1981–1984), there was no centralized governmental response. Although an Office of Gay and Lesbian Health existed within the Department of Health since 1982, it was an advisory body with no policy-making authority. More importantly, it had less political power than the comparable office in San Francisco due to its location within the health department and its inability to influence other city bureaucracies that deal with AIDS in New York.

In November 1984, at the direction of the mayor, an AIDS Policy and Planning Committee was formed. Its first order of business was to send a fact-finding delegation to San Francisco (January 1985) to study that city's response and to make recommendations for improving New York's services to AIDS patients.

The delegation's foremost recommendation was to improve administrative coordination among all city agencies dealing with the AIDS crisis through the Policy and Planning Committee. This body was formalized in March 1985, and its charge was to oversee planning, coordination, monitoring, and evaluation of all the AIDS services provided by the city directly or by contract. Other recommendations called for increased resources for the surveillance efforts of the health department, and enhancement of acute and chronic medical services (including outpatient services), the provision of housing and social services, and educational programs. Most of these recommendations and other AIDS programs were implemented by the beginning of 1986, with a large increase in city appropriations during FY 1986.[9]

In San Francisco, the existence of an office within the health department devoted to gay and lesbian health issues provided a natural organizational focus for AIDS issues as the epidemic developed. The relatively small scale of the city, the recognized importance of homosexuals in local politics, the relatively high educational and income levels of the gay community, and its social organization all contributed to the development of an integrated system of outpatient and community-based services for AIDS patients. These services not only provide an important part of the care for AIDS patients, they have a financial impact on the local health care system by allowing many patients to remain outside the hospital for longer periods and, once admitted, to be discharged earlier. The range of community services available include outpatient clinics, public health education, hospice care, legal services and entitlements advocacy, psychosocial counseling, substance abuse services, and practical support and housing for AIDS patients.

While these services now exist in each city, the timing of their development and sources of funding differed. For example, outpatient clinical services for AIDS patients have been available at SFGH since September 1981. In the fall of 1982, SFGH established a special multidisciplinary clinic (infectious diseases, oncology, psychiatry, psychology, nursing, dermatology, and social work). At the University of California in San Francisco (UCSF), another outpatient clinic for AIDS patients opened in August 1984. During 1985, the clinics at SFGH and UCSF averaged approximately 1,000 patient visits per month.[8]

In New York City, the first outpatient clinic for AIDS patients was opened at Kings County Hospital in early 1984. During 1985, it was open two afternoons per week and handled between 120 and 175 patient visits per month. The first full-time AIDS ambulatory care clinic was opened in Manhattan in the spring of 1985. Slowness in providing comprehensive AIDS outpatient services in New York has probably increased the level and expense of hospitalization there.

The diversity of AIDS patients in New York makes the process of developing a useful range of services far more difficult. Developing services appropriate for both IV drug users and children with AIDS has obviously been much more of a problem for New York than for San Francisco. On the other hand, the highly successful, specialized inpatient AIDS ward at San Francisco General Hospital, where most patients are middle class gay men, has benefited from the homogeneity of its patients and extensive support from the gay community. Attempts to develop such a ward in a setting where the majority of patients are substance abusers would obviously entail difficulties. Other community-based programs in San Francisco, such as the Shanti AIDS Residence Program, which provides long-term housing for AIDS patients (and thereby helps keep patients out of the hospital), are not designed to accommodate patients with substance abuse problems, and, in fact, Shanti does not accept such patients into its program. Thus, the ability of San Francisco to develop a range of successful community-based services is due not only to good planning and a commitment of local resources, it is a reflection of the strength and importance of the gay community, whose members comprise the majority of patients and those at risk.

To support its community-based services, New York City has relied more on state and private funding and less on local funds compared to San Francisco. This may be due to the transfer of responsibility for various services from New York City to New York State government after the fiscal crisis of the 1970s, or it may be due to a paucity of local political power among the groups at risk. It may also reflect the enormous management and planning difficulties—rooted in the complexity of the epidemic in New York—of local government officials as they attempted to develop programs. For example, a city-funded program for home attendant services to persons with AIDS provided care to only a fraction of those in need. An audit of this program found that from its inception in December 1983 through February 1985 a total of only 80 clients had received service, although it was designed to serve a 200-client caseload.[25] The report elaborated a series of reasons for the program's failure to meet its goals, including poor contract planning, lack of needs assessment, enrollment limited to those who were eligible for Medicaid, excessive processing time, lack of housing, and poor community outreach.

Nevertheless, community-based AIDS organizations have played a key role in responding to the epidemic in both cities. They have provided an important and otherwise missing dimension to patient care and have been instrumental in developing and disseminating risk reduction strategies. At the same time, they have had an important financial impact on the local health care systems by keeping patients out of the hospital.

One reason government support for community-based organizations is a cost effective strategy is the heavy reliance on volunteer labor in these groups, which allows for a greater production of services per dollar expended than would be possible if government provided these services directly. However, a significant level of financial support is still necessary to develop administrative structures and to pay staff who can recruit, train, supervise, and support volunteers. According to one national survey,[26] nearly 80% of services provided by community-based AIDS groups around the country were performed by volunteers. In New York and San Francisco, the magnitude of donated labor is enormous, conservatively estimated at more than 100,000 hours in each city (Table V). The amount of donated labor at the Gay Men's Health Crisis in New York is similar to the combined total of the Shanti Project and the San Francisco AIDS Foundation. However, the ratio of unpaid staff hours to paid staff hours is twice as large in New York, indicating a greater reliance on volunteers and lesser funding by local government there.

CONCLUSIONS

The large number of AIDS cases in New York and San Francisco has had a significant impact on the local health

TABLE V. Hours Worked, Largest Community-based AIDS Organizations: New York and San Francisco, 1984–1985

	Volunteer Staff Average Total Hours/Month	Paid Staff Average Total Hours/Month	Ratio Volunteer/ Paid Hours
San Francisco			
AIDS Foundation	3,807	1,976	1.93
Shanti Project	6,852	3,900	1.76
New York			
Gay Men's Health Crisis	8,834	2,427	3.64

Sources: Based on data derived from references 13 and 14.

care systems. It is likely that as the epidemic spreads it will place increasingly greater strains on other health care systems in cities and counties with growing numbers of cases, as well as on public and private sources of health care financing. While the absolute number of AIDS cases continues to grow in metropolitan areas, there is convincing evidence that the disease is spreading across the country. According to the Centers for Disease Control, the four cities with the largest number of AIDS cases—New York, San Francisco, Los Angeles, and Miami—accounted for 73% of all reported cases in September 1982. This figure declined to 65% by September 1983, and, by February 1987, their proportion of the nation's total had fallen to 50%. In other words, the proportion of total AIDS cases not based in these four metropolitan areas has increased by more than 85% during this time period.

The availability of outpatient and community-based care facilities and programs and the relatively low charges per AIDS admission in San Francisco may give an important clue to the direction other communities should follow in a rational planning policy for the treatment of AIDS patients. The average length of hospitalization for AIDS patients in San Francisco is far shorter than in New York and in most other cities. This is due in part to the variations in patient groups, case mix, and practice patterns that are found in different regions of the country. However, an integrated system of health care delivery which is subsidized by local government in San Francisco and includes outpatient clinics, home health and hospice care, housing, and other social support services, allows patients to be discharged from the hospital earlier than in other cities where such services are not as readily available. The success of San Francisco's response to the epidemic does not, however, mean that other cities should attempt to respond in exactly the same way. New York's example illustrates how one city, confronted with complex problems, considered a variety of local conditions rather than merely adopting another city's model. Each community should develop its own response in light of its own epidemiologic circumstances and social and political structures, learning from the experiences of San Francisco and New York City.

The investment of public funds in community-based services affords better quality care for AIDS patients. It is also a rational fiscal response that helps reduce the economic impact of the epidemic by reducing the need for inpatient care. This lowers private health insurance expenditures, Medicaid outlays, and local tax revenues that must be spent when Medicaid or other third-party reimbursement is unavailable or below costs.

Aside from financial support from the public sector, the viability of community-based AIDS organizations depends on a large, steady stream of unpaid labor. If the patient mix shifts further away from homosexual men towards IV drug users, as it slowly appears to be doing, it is unclear whether the level of voluntarism can be maintained. In low incidence regions, there may not be an identified at-risk population from which to draw volunteers. Thus, unless further financial support is forthcoming, gaps in services to AIDS patients, already experienced by many of these communities, may become more severe as the epidemic continues to spread geographically.

New York City and San Francisco illustrate that the AIDS epidemic may manifest itself quite differently in various cities and can trigger diverse responses based on local conditions. With the likely prospect of other cities facing large increases in the number of those afflicted with AIDS, the experiences of New York and San Francisco may prove useful. They indicate that each community's response to AIDS will probably reflect the underlying social, economic, and political characteristics of AIDS victims and the existing structure and organizational roles of traditional health care and community-based service providers. They also indicate the intrinsic limits to the current dependency on unpaid labor and the contributions from private charity and local government, increasing the pressure for additional state and federal support for community-based services and the care of AIDS patients.

Acknowledgment. The authors thank the many people in New York City and San Francisco who generously provided their time and expertise in helping to gather and interpret the data from each community.

REFERENCES

1. Arno PS: Statement before the Subcommittee on Health and the Environment, Committee on Energy and Commerce, US House of Representatives, July 22, 1985.
2. Hardy AM, Rauch K, Echenberg D, et al: The economic impact of the first 9,000 cases of acquired immunodeficiency syndrome in the United States. Presented at the International Conference on AIDS, Atlanta, Georgia, April 17, 1985.
3. Shilts R: AIDS crisis hits an unprepared New York. *San Francisco Chronicle*, February 1985; 14:1.
4. Leishman K: San Francisco: A crisis of public health. *Atlantic Monthly*, October 1985, pp 18–41.
5. Boufford JI: Statement before Intergovernmental Relations and Human Resources Subcommittee of the Committee on Government Operations, US House of Representatives, September 13, 1985.
6. Botnick V: New York City's Response to AIDS. Memo to Edward Koch, Mayor, and Stanly Brezenoff, Deputy Mayor for Operations, City of New York, March 27, 1985.
7. West Bay Hospital Conference: *Monthly AIDS Hospital Utilization Report*. San Mateo, California, October 25, 1985.
8. San Francisco Department of Public Health: *San Francisco's Response to AIDS: Status Update*. October 8, 1985.
9. Sencer DJ, Botnick VE: *Report to the Mayor: New York City's Response to the AIDS Crisis*. Office of the Mayor, City of New York, December 1985.
10. West Bay Hospital Conference: *Quarterly AIDS Utilization Report*, San Mateo, California, March 5, 1986.
11. Scitovsky AA, Cline M, Lee PR: Medical care costs of patients with AIDS in San Francisco. *JAMA* 1986; 256:3103–3106.
12. Knickman J, Foltz AM: Regional differences in hospital utilization. How much can be traced to population differences? *Med Care* 1984; 22:971–986.
13. Gay Men's Health Crisis: 1984 Annual Report, New York, 1985.
14. Arno PS: The nonprofit sector's response to the AIDS epidemic. Community-based services in San Francisco. *Am J Public Health* 1986; 76:1325–1330.
15. Centers for Disease Control: *AIDS Weekly Surveillance Report*. US AIDS Program, Center for Infectious Diseases, Atlanta, Georgia, January 5, 1987.
16. New York City Department of Health: *AIDS Surveillance Update*. Office of Epidemiologic Surveillance and Statistics, January 1987.
17. Volberding PA: The clinical spectrum of the acquired immunodeficiency syndrome. Implications for comprehensive patient care. *Ann Intern Med* 1985; 103:729–733.

18. Boufford JI: Statement before Subcommittee on Health and Environment, Committee on Energy and Commerce, US House of Representatives, November 1, 1985.

19. New York State Comptroller. *Review of New York City's Proposed Financial Plan for Fiscal Years 1986 through 1989*. Report No. 30-86, December 11, 1985.

20. Imperato PJ: *The Administration of a Public Health Agency*. New York, Human Sciences Press, 1983, pp 100–105.

21. Farley PJ: Who are the underinsured? *Milbank Memorial Fund Quarterly/Health and Society* 1985; 63:476–503.

22. Sencer DJ: Major urban health departments: The ideal and the real. *Health Affairs* 1983; 2:88–95.

23. Research and Decisions Corporation: *Designing an Effective AIDS Prevention Strategy for San Francisco; Results from the First Probability Sample of an Urban Gay Male Community*. Prepared for the San Francisco AIDS Foundation, December 3, 1984.

24. Martin JL: *The Impact of AIDS on New York City Gay Men: Development of a Community Sample*. Presented at the Annual Meetings of the American Public Health Association, Washington, DC, November 21, 1985.

25. New York City Comptroller: *Report on the New York City Human Resources Administration of the American Red Cross AIDS Home Attendant Program*, ML 85-504, April 15, 1985.

26. US Conference of Mayors: *Local Responses to AIDS: A Report of 55 Cities*. Washington, DC, November 1984.

The effect of group education on improving attitudes about AIDS risk reduction

RONALD O. VALDISERRI, MD; DAVID W. LYTER, MD; LAWRENCE A. KINGSLEY, DRPH; LAURA C. LEVITON, PHD; JANET W. SCHOFIELD, PHD; JAMES HUGGINS, MSW; MONTO HO, MD; CHARLES R. RINALDO, PHD

ABSTRACT. Four hundred sixty-four homosexual and bisexual men, recruited from a cohort of 1,700 men enrolled in a study of the natural history of acquired immunodeficiency syndrome (AIDS), participated in a peer-led, small-group educational session promoting AIDS risk reduction. Although levels of knowledge about AIDS and human immunodeficiency virus (HIV) transmission were uniformly high prior to intervention, at least 60% of the men reported having engaged in unprotected, receptive anal intercourse with more than one partner in the preceding six months. Prior to intervention, a substantial number of the men had mixed feelings about AIDS risk reduction or endorsed negative attitudes about AIDS risk reduction. After attending the session, attitudes improved significantly in five of the six areas surveyed.

The ability of a group educational session to influence attitudes about AIDS risk reduction in a positive way suggests that this type of intervention may be effective in enabling homosexual and bisexual men to adopt low-risk sexual activities by influencing the nonhealth motives of sexual behavior, especially peer norms about safe sex. Long-term follow-up of this cohort will test for maintenance of this attitudinal change and, more importantly, will evaluate whether this attitudinal change is predictive of future changes in sexual behavior and HIV seroconversion rates. The authors stress the importance of incorporating existing health promotion research findings into the design and evaluation of AIDS risk reduction programs. (NY State J Med 1987; 87:272–278)

AIDS is the most dramatic and devastating consequence of the ongoing epidemic of human retroviral infection. In response, public health officials are attempting to prevent or decrease the sexual transmission of HIV among all risk groups, including homosexual and bisexual men, by urging the adoption of safer sexual practices. Although some officials have pointed to decreasing rates of rectal gonorrhea as evidence that homosexual men are themselves modifying high-risk behavior,[1] others have argued that such behavioral changes may have a minimal impact on the incidence of AIDS, because of the high seroprevalence rate of HIV antibody among the urban homosexual male population.[2]

There are few published evaluations of the efficacy of health promotion campaigns for AIDS risk reduction in the homosexual community. Therefore, it is important to utilize existing research and theory about health behavior derived from the design and evaluation of other risk reduction programs in designing and implementing AIDS risk reduction programs for homosexual and bisexual men. A review of the salient health promotion literature supports the hypothesis that simply acquiring more knowledge about risky activity may be inadequate to change behavior in many individuals. This has been demonstrated in efforts to encourage smoking cessation,[3–6] nutrition education,[7] seat belt use,[8] and breast self-examination.[9]

Among persons for whom information alone is not adequate to induce behavioral change, specific skills dealing with how to change and experiences that reinforce successful change may be essential. Support for this hypothesis is found in the literature that describes diabetes education,[10] smoking cessation and prevention of smoking onset,[6] and occupational safety training.[11] Specifically, the nonhealth motives of health-related behavior must be addressed if unhealthy behavior is to be modified. These

---

From the Department of Pathology (Drs Valdiserri and Rinaldo), Department of Medicine (Dr Lyter), and Division of Infectious Diseases (Dr Ho), University of Pittsburgh School of Medicine; the Department of Epidemiology (Dr Kingsley) and the Department of Health Services Administration (Dr Leviton), University of Pittsburgh School of Public Health; the Department of Psychology (Dr Schofield), University of Pittsburgh; and the Persad Center, Inc (Mr Huggins).

Address correspondence to Dr Valdiserri, University of Pittsburgh, Falk Clinic Laboratories, 3601 5th Ave, Pittsburgh, PA 15213.

Portions of this paper were presented at the International Conference on AIDS, Paris, France, June 25, 1986. This research was supported by a grant from the Centers for Disease Control (DHHS Grant No. U62/CCU3001060-01) and the Pitt Men's Study (Contract No. AI-32513, NIH).

nonhealth motives are often influenced by peer norms and existing social support systems,[10,12,13] and as such may be modified by group process.

Preliminary information concerning sexual behaviors among male homosexuals at risk for AIDS supports these assertions. In 1983, when McKusick et al[14] surveyed 655 homosexual men in San Francisco, they found that although men "were uniformly well-informed about the prescribed behavior for AIDS risk reduction," many displayed discrepancies between what they believed and their reported sexual behavior. Others have stressed the inadequacy of "warn and scare tactics" in preventing the transmission of HIV,[15] and, in terms of efficacy, some researchers have noted parallels to adolescent smoking prevention programs, in which social influence models using peer-led group discussions have been more effective than fear-based media and educational campaigns.[3,16]

Research in other areas of health promotion has documented that context may be just as important as content in terms of inducing health-related behavioral change.[17] This also implies that a group format may be more successful at promoting AIDS risk reduction among homosexual and bisexual men than media-based campaigns or individual counseling sessions, because such a format can affect group social norms. Supplying accurate information about HIV risk reduction in conjunction with social influence could promote behavioral change among homosexual men in three ways: by providing a coping mechanism for fear arousal;[18] by increasing feelings of self-efficacy;[19] or by decreasing feelings of perceived vulnerability.[20] Using peers (other homosexual men) to deliver the information may also increase efficacy, as has been observed in smoking prevention programs for adolescents.[3,21]

SUBJECTS AND METHODS

In September 1983, the University of Pittsburgh received funding from the National Institutes of Health to conduct a prospective study of the natural history of HIV infection in homosexual and bisexual men. To date, the "Pitt Men's Study" (PMS) has enrolled more than 1,700 men from western Pennsylvania, Ohio, and West Virginia in a baseline study of the epidemiology of HIV infection. From this population, 1,062 self-selected men have agreed to participate in a more extensive study that represents the Pittsburgh cohort of the Multicenter AIDS Cohort Study (MACS). Members of both groups are tested for antibody to HIV at six-month intervals and provide information about sexual behavior and other health matters via questionnaire. The MACS group also has periodic physical examinations. The results of antibody tests are not released to volunteers without their express consent.

Beginning in September 1985, these 1,700 men were invited by mail to participate in an educational session (known as the AIDS Prevention Project) as part of the process of learning their HIV antibody status. In addition to this direct mail invitation, the AIDS Prevention Project (APP) was also promoted within the male homosexual community. Volunteers already enrolled in the PMS were encouraged to participate in the APP, while men who had not joined either study were recruited for both studies simultaneously. Recruitment efforts consisted of flyers, brochures, and a poster stressing the preventability of AIDS through appropriate sexual precautions. Numerous mailings and community presentations were also undertaken stressing the importance of prevention as a practical alternative to anxiety and fear. Although earlier recruitment efforts were closely linked to the process of learning HIV antibody results, ongoing recruitment efforts have emphasized that individuals can participate in the risk reduction program even if they do not wish to learn their HIV antibody status.

Intervention. The intervention consists of an informal group educational session led by a male health educator who is also homosexual. Groups consist of five to ten participants and last 60–75 minutes, including questions after the presentation. The goals of this session are as follows: to increase individual knowledge about the transmission, incubation period, and spectrum of clinical diseases related to HIV; to increase individual understanding of the relative HIV transmission risk associated with specific sexual practices; to instruct participants in the appropriate use of condoms; and to educate participants about interpreting HIV antibody tests.

Data Collection and Analysis. Data are collected at three points in this project: before the intervention (pre-test); one to two weeks after the intervention (post-test); and four to six months after the intervention (long term). They are obtained from self-administered questionnaires that assess knowledge, attitude, and self-reported sexual behavior. Sexual behavior is reported only at pre-test and long term; attitude and knowledge are assessed at all three points. After an individual calls to schedule an appointment for the educational session, the pre-test questionnaire is mailed to him, with instructions that he is to bring the completed form with him on the day of his session. The post-test questionnaire is handed to participants immediately after they have completed a session, with the instructions that they are to return it by mail (a pre-stamped envelope is provided) within two weeks. Appointments for disclosure of antibody test results are scheduled separately, after the intervention. Antibody test results are released to individual volunteers by a study clinician. Long-term questionnaires are mailed to participants four to six months after they have completed a session, with instructions that they are to return the questionnaire, in person, at their next Pitt Men's Study visit.

The knowledge assessment instrument consists of 17 multiple choice items dealing with HIV transmission, HIV incubation period, manifestations of HIV infection, interpretation of HIV antibody test results, and currently recognized AIDS risk groups. The attitude assessment tool consists of 38 items for which responses are recorded on a 5-point Likert scale with 1 representing "strongly disagree," 3 representing "mixed feelings," and 5 representing "strongly agree;" there is also an option for "not applicable." The attitude assessment surveys feelings and beliefs about high- and low-risk sexual practices, condom use, number of sexual partners, anonymous sexual partners, perceived vulnerability to AIDS, and perceptions about the acceptance of safer sexual practices by peers. The 38 statements in this instrument are written with emphasis on the following: intention to act in a specified manner; willingness to alter sexual habits; and individual, subjective perceptions about safer sexual practices. Positive statements describe the intention to refrain from high-risk sexual practices, to engage in low-risk sexual practices, to use condoms regularly, to decrease the number of sexual partners, to refrain from anonymous sexual encounters, and to discuss AIDS prevention with sexual partners. Finally, in regard to peer acceptance, a positive response would be defined as one in which a subject agrees with statements describing the acceptance of AIDS risk reduction by homosexual friends and acquaintances. The sexual behavior questionnaire focuses on the number of partners, types and frequency of sexual practices, including anal intercourse, and the frequency of use of condoms and spermicides (as antiviral agents).

All individuals who formally declined participation in the intervention (105 subjects) completed knowledge, attitude, and sexual behavior questionnaires, so that these data are available for pretest or baseline comparison to individuals who agreed to participate. Comparable data are not available for those who failed to respond.

RESULTS

Pre-Intervention Assessment. Of the 1,700 individuals contacted

TABLE I. Comparison of Groups at Baseline (N = 1,700)

	No Response	Declined	Accepted
No. (% total)	1,050 (62%)	105 (6%)	545 (32%)
Mean age (years)	31	32	33
College degree	54%	58%	66%*
HIV seropositive	19%	22%	19%

* $p < 0.001$, chi-square test.

through May 31, 1986, 1,050 (62%) failed to respond, 105 (6%) declined to participate, and 545 (32%) accepted the invitation to participate in an educational session prior to learning their HIV antibody status. Table I shows comparative demographic data on these three groups at baseline. Although the mean age and percent of seropositivity for HIV antibody does not differ significantly across groups, individuals who agreed to participate were more likely to have a college education than those who failed to respond ($p < 0.001$).

Table II compares high-risk sexual practices of the groups at baseline—prior to intervention. Although similar data are not available for the nonresponders, it is apparent that a majority of individuals in both the "decline" and "accept" groups were still participating in high-risk sexual activities prior to this intervention. At least 60% of both groups had engaged in unprotected receptive anal intercourse with more than one partner in the six months prior to the assessment. Of the 545 men who were willing to participate in this group educational session, 464 were actually enrolled in sessions from September 1985 through May 1986.

As seen in Tables III and IV, knowledge at baseline about a variety of topics related to HIV infection or AIDS was uniformly high in both the 464 session participants and the 105 men who declined. Also, no significant differences in knowledge about HIV were noted in these two groups. Men from both groups scored lowest (77% mean correct response) on two questions about the local prevalence of HIV infection, with nearly equal percentages over- and under-estimating prevalence.

Based on their content, attitude statements were categorized and assigned to one of the following six categories: sexual practices; condom use; number of partners/anonymous partners; perceived vulnerability to AIDS; discussion of AIDS prevention with partners; and perceptions about peer acceptance of AIDS risk reduction. Tables V and VI describe the attitudes held by individuals who declined and those who participated in the intervention, at the time of the pretest. No substantial attitudinal differences were noted between the two groups. Although a majority of respondents endorsed attitudes that correspond to AIDS risk reduction, a sizable minority did not, or had "mixed feelings" on the matter. Of interest is the fact that the highest overall endorsement (combined mean percent response frequency of "strongly agree" and "agree") among both decliners and accepters was associated with the category of statements dealing with attitudes about decreasing the number of sexual partners (76% for decliners and 71% for participants).

TABLE II. Comparison of Sexual Practices Prior to Intervention

Total Number of Sex Partners, Last Six Months	Declined (N = 105)	Accepted (N = 545)
1	26%	24%
2–5	44%	48%
6–25	27%	25%
26+	3%	4%
% reporting receptive anal intercourse, last six months, >1 partner	61%	60%
Mean no. partners for RAI,* last six months (range)	1.7 (1–15)	1.9 (1–100)

* RAI, receptive anal intercourse, for those with ≥1 partner, mean is expressed as log base 10.

TABLE III. Knowledge About AIDS in Individuals Who Declined Participation (N = 105)

Subject of Multiple Choice Questions (Number of Questions)	Mean % Correct Response (Range)
Transmission of HIV (5)	90% (76–99%)
AIDS risk groups (1)	97%
HIV incubation period (1)	96%
Manifestations of HIV infection (3)	92% (91–94%)
Local AIDS prevalence (2)	77% (67–86%)
Interpretation of HIV antibody results (5)	88% (80–94%)

Compared to other categories, items related to discussing AIDS prevention with partners received the lowest overall endorsement. Individuals who declined participation scored 53% in this category, and those who agreed to participate scored 48%. This may indicate that although homosexual men may know how to reduce the risks of transmitting HIV sexually, they are uncertain of how to approach the issue with a partner.

Post-Intervention Assessment. Within two weeks after completing the intervention, participants were asked to return a follow-up questionnaire that assessed knowledge and attitude. The assessment of knowledge one to two weeks after the intervention (Table VII) demonstrated scores that were quite similar to pre-intervention ratings. This finding is not surprising given the high levels of knowledge noted prior to the intervention. The only exception is that scores improved for the two questions that deal with the local prevalence of HIV antibody and HIV-related disease.

Table VIII shows the distribution of responses to the attitudinal statements of 464 men after their participation in the group educational session. The greatest change away from "mixed feelings" and the endorsement of unhealthy behaviors occurred in the area of atti-

TABLE IV. Knowledge About AIDS in Individuals Who Participated, Prior to Intervention (N = 464)

Subject of Multiple Choice Questions (Number of Questions)	Mean % Correct Response (Range)
Transmission of HIV (5)	86% (71–99%)
AIDS risk groups (1)	95%
HIV incubation period (1)	95%
Manifestations of HIV infection (3)	92% (85–96%)
Local AIDS prevalence (2)	77% (65–90%)
Interpretation of HIV antibody results (5)	87% (75–97%)

TABLE V. Attitudes Held by Individuals Who Declined Participation (N = 105)

Subject of Attitudinal Statements (Number of Statements)	Mean % Response Frequency (Range)				
	Strongly Agree	Agree	Mixed Feelings	Disagree	Strongly Disagree
Sexual practices (10)	42% (33–63%)	29% (21–37%)	18% (8–27%)	8% (1–16%)	4% (1–9%)
Condom use (7)	42% (27–66%)	23% (14–28%)	17% (8–24%)	12% (1–20%)	7% (0–16%)
Number of partners/ anonymous partners (8)	55% (43–76%)	21% (18–25%)	13% (5–20%)	7% (0–14%)	4% (0–8%)
Perceived vulnerability to AIDS (5)	41% (24–63%)	23% (19–29%)	26% (11–38%)	6% (1–11%)	5% (3–7%)
Discussing AIDS prevention with partners (4)	33% (18–55%)	20% (7–34%)	20% (15–28%)	17% (7–28%)	12% (1–30%)
Perceptions about peer acceptance of AIDS risk reduction (4)	27% (25–30%)	46% (34–56%)	23% (10–35%)	4% (2–5%)	1% (0–2%)

tudes about condom use (mean change, 10%) and discussing AIDS prevention with a partner (mean change, 9%)—both areas in which specific behaviors should lead directly to greater prevention. The smallest change away from "mixed feelings" and the endorsement of unhealthy behaviors occurred in the category of statements about number of partners/anonymous partners.

Of major importance is the fact that the post-intervention attitude scores differed significantly from pre-intervention scores on the basis of a Wilcoxon matched-pairs signed-ranks test (Table IX). In all categories of statements except one, a majority of statements within each category demonstrated significant change in a positive direction (away from the endorsement of unhealthy behaviors and toward the endorsement of healthier behaviors) at a significance level of $p < 0.05$. However, no significant change was found in pre-intervention versus post-intervention scores for any of the eight statements that assessed attitudes about numbers of partners and anonymous contacts.

DISCUSSION

Data from the initial phase of a project designed to promote AIDS risk reduction have been analyzed to deter-

TABLE VI. Attitudes Held by Individuals Who Participated, Prior to Intervention (N = 464)

Subject of Attitudinal Statements (Number of Statements)	Mean % Response Frequency (Range)				
	Strongly Agree	Agree	Mixed Feelings	Disagree	Strongly Disagree
Sexual practices (10)	42% (29–64%)	28% (22–35%)	16% (6–25%)	9% (3–13%)	5% (3–7%)
Condom use (7)	40% (19–65%)	25% (19–33%)	22% (8–35%)	10% (1–15%)	5% (1–7%)
Number of partners/ anonymous partners (8)	47% (33–73%)	24% (17–28%)	16% (7–23%)	9% (2–15%)	6% (2–8%)
Perceived vulnerability to AIDS (5)	40% (22–64%)	27% (23–31%)	22% (7–34%)	7% (3–11%)	4% (3–7%)
Discussing AIDS prevention with partners (4)	27% (9–53%)	21% (10–24%)	24% (14–32%)	16% (6–30%)	13% (4–30%)
Perceptions about peer acceptance of AIDS risk reduction (4)	26% (23–32%)	44% (40–50%)	25% (17–32%)	3% (2–4%)	1% (1%)

TABLE VII. Knowledge About AIDS After Participation in an Educational Session (N = 464)

Subject of Multiple Choice Questions (Number of Questions)	Mean % Correct Response (Range)
Transmission of HIV (5)	94% (87–99%)
AIDS risk groups (1)	96%
HIV incubation period (1)	94%
Manifestations of HIV infection (3)	94% (92–97%)
Local AIDS prevalence (2)	84% (79–89%)
Interpretation of HIV antibody results (5)	91% (81–98%)

mine whether group educational sessions are capable of influencing knowledge and attitudes about AIDS in the immediate post-intervention period. Participants were homosexual and bisexual men recruited from an ongoing study of the natural history of AIDS. The participants agreed to attend an educational session as a prerequisite for learning their HIV antibody serostatus, which had been previously determined as part of the natural history study protocol. The study group described here consisted of 464 men who attended one of these sessions.

The fact that a majority of individuals (1,050) failed to respond to this invitation may have important implications for public health officials who wish to link AIDS risk reduction programs with disclosure of antibody results. In fact, all the data obtained from this study must be interpreted with the understanding that in the pilot phase of the program, enrollment was a prerequisite for obtaining HIV antibody results. This introduces a degree of selection bias, in that individuals who participated in the intervention may have been more motivated and therefore more capable of change than individuals who declined or failed to respond, because they did not wish to learn their antibody status.

Seroprevalence rates for all three groups—nonresponders, those who declined, and those who accepted—were equivalent, ranging from 19% to 22%. These data suggest that rates of infection are no different among those who agreed to participate in the intervention than among those who did not. The three groups were also quite similar in regard to age. The only statistically significant difference detected was that individuals who agreed to participate in the intervention were more likely to have a college degree than those who failed to respond.

An analysis of the sexual practices of the two groups (those who declined and those who accepted) prior to intervention demonstrated remarkable similarities regarding the total number of different sexual partners in the previous six months. Among both groups, nearly two thirds reported engaging in unprotected receptive anal intercourse with more than one partner in the preceding six months. This finding is particularly noteworthy when correlated with the extraordinarily high level of knowledge about AIDS and its transmission demonstrated by both groups prior to intervention. As has been noted by other investigators,[14,22] accurate knowledge of high-risk sexual activities, such as unprotected receptive anal intercourse, may not be predictive of sexual behavior.

Although a majority of individuals endorsed positive attitudes about AIDS risk reduction prior to the intervention, in all areas surveyed, there was still a sizable minority of at-risk men who did not endorse positive attitudes about AIDS risk reduction or who had "mixed feelings" on the matter. One of the goals of this intervention is a

TABLE VIII. Attitudes Held by Individuals After Participation in an Educational Session (N = 464)

Subject of Attitudinal Statements (Number of Statements)	Mean % Response Frequency (Range)				
	Strongly Agree	Agree	Mixed Feelings	Disagree	Strongly Disagree
Sexual practices (10)	48% (34–72%)	28% (21–39%)	14% (4–21%)	8% (2–11%)	4% (2–6%)
Condom use (7)	48% (23–83%)	25% (15–35%)	17% (1–36%)	7% (1–12%)	3% (1–5%)
Number of partners/anonymous partners (8)	48% (34–72%)	24% (21–27%)	15% (5–22%)	8% (1–14%)	5% (1–7%)
Perceived vulnerability to AIDS (5)	48% (24–77%)	24% (16–31%)	17% (2–27%)	6% (2–11%)	5% (2–8%)
Discussing AIDS prevention with partners (4)	34% (10–63%)	22% (13–31%)	19% (9–24%)	15% (5–31%)	10% (2–28%)
Perceptions about peer acceptance of AIDS risk reduction (4)	31% (26–34%)	44% (40–47%)	22% (17–28%)	3% (2–3%)	1% (1%)

TABLE IX. Pre-Intervention Versus Post-Intervention Attitude Change (N = 464)

Subject of Attitudinal Statements	Number of Statements	Number of Statements with Positive Change
Sexual practices	10	7* (70%)
Condom use	7	5* (71%)
Number of partners/ anonymous partners	8	0
Perceived vulnerability to AIDS	5	3* (60%)
Discussing AIDS prevention with partners	4	3* (75%)
Perceptions about peer acceptance of AIDS risk reduction	4	3* (75%)

* $p < 0.05$, by Wilcoxon matched-pairs signed-ranks test.

reduction of this minority and of the risky behaviors that may correlate with these attitudes.

Prior to intervention, the lowest percentage of agreement for both the men who declined and those who subsequently participated was found among the statements concerning the discussion of AIDS prevention with sexual partners. This may indicate that for some men, accurate knowledge about HIV transmission is, by itself, inadequate to promote risk reduction behavior. The highest percentage of endorsement prior to intervention was found in association with statements about reducing the numbers of sexual partners and anonymous contacts. This may correlate with the observations of others that many homosexual men have already responded to the AIDS epidemic by decreasing their sexual activity.[1,23,24] If this is so, it follows that homosexual and bisexual men would tend to recognize reduction in the number of sexual partners as a positive or beneficial adaptation, even before this intervention program.

Although there was little overall change in the levels of knowledge after the intervention (Table VII), this is not surprising given the extremely high levels of knowledge recorded at pre-test. Attitude scores, however, improved significantly after the educational intervention. Positive attitudinal changes were noted in response to a majority of statements dealing with sexual practices, condom use, perceived vulnerability to AIDS, the intention to discuss AIDS prevention with future sexual partners, and perceptions of peer acceptance of safer sexual practices (Table IX). Attitudes about condom use and the discussion of AIDS prevention with partners showed the greatest change away from "mixed feelings" and the endorsement of risky practices toward the endorsement of risk reduction practices.

No significant change was detected in the post-test responses to any of the eight attitudinal statements dealing with numbers of sexual partners, including anonymous contacts. Although it is possible that this intervention program is not capable of influencing these attitudes, this is unlikely since significant positive change was achieved in all other areas surveyed. An alternate explanation may be that homosexual and bisexual men who are capable of reducing or willing to reduce their sexual contacts have already done so as a result of the prominent association of AIDS with promiscuity, as reported by the media. Although this is a positive change and should continue to be reinforced by health officials, it may also promote the dangerous misconception that most sexual acts are safe if performed by individuals who are not promiscuous or with non-anonymous partners. Because of the extremely long incubation period of this virus, monogamy per se does not guarantee that an individual will not become infected.

The ability of the group educational session to positively change attitudes about AIDS risk reduction in the immediate post-intervention period provides evidence that this type of intervention may be successful at influencing the nonhealth motives of sexual behavior in homosexual and bisexual men, by affecting group norms. Research from other health promotion efforts would suggest that the ability to influence these motives would result in more effective and, perhaps, more permanent behavioral change.[10,12,13,25] Regarding AIDS risk reduction, it has been suggested that emphasizing the social acceptability of safer sexual practices might be more effective at persuading homosexual and bisexual men to change sexual behaviors than merely stressing the dangers of unsafe sexual practices.[22,25] The process of discussing sexual practices and AIDS risk reduction among a group of homosexual and bisexual men may, in fact, serve to validate the notion of safe sex for group participants.

The long-term follow-up of this cohort will test whether these immediate changes in attitude are predictive of future sexual behavior. The problems of predicting behavior from attitude are well known, and attitude may or may not be related to subsequent sexual behavior in this group. However, the pattern of results described here suggests that behavioral change is likely to follow. Furthermore, many of the attitudinal items surveyed reflect specific intentions to act, rather than global attitudes toward sex. Such specific items appear to be more highly correlated with actual behavior.[26]

The long-term follow-up of participants in this project will also determine whether attitudinal change is maintained over time, and whether there is a relationship between attitudes and subsequent sexual behavior, through both self-report and clinical indicators such as HIV seroconversion rates. The AIDS Prevention Project is ongoing; in the future, an additional intervention program is planned—one that has a skills training component in addition to the group educational session. This will enable an evaluation of the differential efficacy of each component.

Finally, these results demonstrate the importance and appropriateness of applying existing health promotion theory and research findings to the field of AIDS risk reduction.

Acknowledgments. The authors thank Mr Kerry Stoner, Mr Tony Silvestre, and Ms Diane Bell for their assistance.

REFERENCES

1. Judson FN: Fear of AIDS and gonorrhea rates in homosexual men. *Lancet* 1983; 2:159-169.
2. Handsfield HH: Decreasing incidence of gonorrhea in homosexually active men—minimal effect on risk of AIDS. *West J Med* 1985; 143:469-470.
3. McAlister AL, Perry C, Maccoby N: Adolescent smoking: Onset and prevention. *Pediatrics* 1979; 63:650-658.
4. McAlister AL, Puska P, Salonen JT, et al: Theory and action for health promotion: Illustrations from the North Karelia project. *Am J Public Health* 1982; 72:43-50.
5. Perry C, Killen J, Telch M, et al: Modifying smoking behavior of teenagers: A school-based intervention. *Am J Public Health* 1980; 70:722-725.
6. Leventhal H, Cleary PD: The smoking problem: A review of research and theory in behavioral risk modification. *Psychological Bull* 1980; 88:370-405.
7. St Pierre RG, Cook TD, Staw RB: An evaluation of the nutrition education and training program: Findings from Nebraska. *Evaluation and Program Planning* 1981; 4:335-344.
8. Ware A, Bigelow B, Sleet D: Safety belt use at the worksite. *Corporate Commentary* 1984; 1:16-27.
9. Parkinson R, Denniston RW, Baugh T, et al: Breast cancer: Health education in the workplace. *Health Educ Q* 1982; 9:61-72.
10. Steiner G, Lawrence PA: *Educating Diabetic Patients.* New York, Springer-Verlag, 1981.
11. Samways MC: Cost-effective occupational health and safety training. *Am Ind Hyg Assoc J* 1983; 44:A6-A9.
12. Levine DM: Health education for behavioral change—clinical trial to public health program. *Johns Hopkins Med J* 1982; 151:215-219.
13. Diesenhaus H: Current trends in treatment programming for problem drinkers and alcoholics, in *Alcohol and Health Monographs: Prevention, Intervention and Treatment: Concerns and Models.* Washington, Public Health Service NIAAA, 1982.
14. McKusick L, Horstman W, Coates TJ: AIDS and sexual behavior reported by gay men in San Francisco. *Am J Public Health* 1985; 75:493-496.
15. Coates TJ, Temoshok L, Mandel J: Psychosocial research is essential to understanding and treating AIDS. *Am Psychol* 1984; 39:1309-1314.
16. Thompson EI: Smoking education programs, 1960-1976. *Am J Public Health* 1976; 687:250-257.
17. Worden JK, Costanza MC, Foster RS, et al: Content and context in health education: Persuading women to perform breast self-examination. *Prev Med* 1983; 12:331-339.
18. Leventhal H, Meyer D, Gutmann M: The role of theory in the study of compliance to high blood pressure regimens, in Haynes RB, Mattson MW, Tilmer OE (eds): *Patient Compliance to Prescribed Antihypertensive Medication Regimens: A Report to the National Heart, Lung and Blood Institute,* US Dept of Health and Human Services publication No. (NIH) 81-2102, 1980.
19. Bandura A: Self-efficacy: Toward a unifying theory of behavioral change. *Psychol Review* 1977; 84:191-215.
20. Rosenstock IM: Historical origins of the health belief model. *Health Education Monographs* 1974; 2:328-335.
21. Vartiainen E, Palonen V, McAlister A, et al: Effects of two years of education intervention on adolescent smoking. *Bull WHO* 1983; 61:529-532.
22. Designing an effective AIDS prevention strategy for San Francisco. San Francisco, Research and Decisions Corporation, 1984.
23. Schechter MT, Jeffries E, Constance P, et al: Changes in sexual behavior and fear of AIDS. *Lancet* 1984; 1:1293.
24. Puckett SB, Bart M, Bye LL, et al: Self-reported behavioral change among gay and bisexual men—San Francisco. *MMWR* 1985; 34:613-615.
25. McKusick L, Conant M, Coates T: The AIDS epidemic: A model for developing intervention strategies for reducing high-risk behavior in gay men. *Sex Trans Dis* 1985; 12:229-233.
26. Fishbein M, Ajzen I: *Belief, Attitude, Intention and Behavior.* Reading, Mass, Addison-Wesley, 1975.

Hospital workers and AIDS: Effect of in-service education on knowledge and perceived risks and stresses

LYDIA O'DONNELL, EdD, CARL R. O'DONNELL, ScD, MPH

ABSTRACT. Hospital workers were surveyed in 1985 and again in 1986, after the institution of in-service training programs, regarding their knowledge about AIDS and their perceptions of the risks and stresses of AIDS patient care. The study found that in-service training was associated with reductions in workers' reported stress, perceived risks, and negative attitudes, and with improvements in knowledge and satisfaction with the quality of care provided.

(NY State J Med 1987; 87:278-280)

In the six years since acquired immunodeficiency syndrome (AIDS) was first described, clinical and impressionistic accounts have documented the existence of "AIDS-anxiety" among health-care providers.[1-6] It is often assumed that these concerns have been lessened by mounting evidence that AIDS is not easily transmitted, greater experience with the disease, and increased public awareness and acceptance of those persons who have AIDS. However, since medical personnel are being called upon to treat an increasing number of patients with AIDS, their initial apprehensions may have been compounded or supplanted by the demands of providing more AIDS-related care. Hospitals have been advised to establish AIDS committees and to devote considerable resources to AIDS-related education programs. To date, the need for such programs, given the widespread media attention and public education about the disease, is not fully documented. We report on a longitudinal study of a hospital staff's knowledge about AIDS, as well as their perceptions of the risks and stresses associated with the care of AIDS patients. We evaluated the extent to which attendance at in-service programs is associated with improvements in AIDS-related knowledge and with decreases in negative attitudes.

METHODS

In April 1985 we surveyed professional and technical employees at the major AIDS treatment center in Massachusetts, a 500-bed teaching hospital to which approximately 60 AIDS patients had been admitted by the time of the survey. The sampling pool consisted of employees in departments and on floors most likely to be providing care to patients with AIDS. A total of 227 participants, or 71% of those contacted, completed questionnaires. In the spring of 1985, the hospital established an AIDS Education Committee, which initiated a coordinated effort to provide sup-

---

From the Department of Medicine, New England Deaconess Hospital, Boston, Mass, and the Wellesley College Center for Research on Women, Wellesley, Mass.

Address correspondence to Dr Lydia O'Donnell, Pulmonary Laboratory, New England Deaconess Hospital, 185 Pilgrim Rd, Boston, MA 02215.

port and education to staff members. In-service education programs were available to hospital employees throughout the next year. Information was provided on the biology and epidemiology of AIDS, its clinical course and treatment, and infection control. There was explicit discussion of high-risk activities and risk groups. In June 1986, by which time more than 130 AIDS patients had been admitted, employees in these same areas were resurveyed. A total of 208 staff members, or 65% of those contacted, completed questionnaires. More than half of these (52%) had participated in the previous survey. Of those who did not complete the original survey, 52% were new employees. In the first survey year, 50% of the participants were registered nurses (RNs), 19.4% were technicians, 14.1% were house officers, 11.9% were licensed practical nurses (LPNs) or aides, and 8.8% were social service personnel. In the second survey year, 49.5% of the participants were RNs, 17.3% were technicians, 14.9% were house officers, 9.6% were LPNs and aides, and 8.7% were social service personnel.

Information was collected on the employees' degree of contact with AIDS patients, knowledge of modes of transmission, and (for the second survey year) attendance at in-service education programs. Employee and nursing grand rounds were charted, as well as classes with supervisors and unit teachers, infection control personnel, and the AIDS health educator. Attendance at in-service programs was voluntary, and programs were provided for all shifts. (Programs were not designed for house officers, so they were excluded from this part of the analysis.) Respondents were asked to report on how comfortable, stressed, and at-risk they felt while working with AIDS patients. They also assessed the adequacy of their knowledge and the job they performed.

Responses to individual questions were cross-tabulated by survey year, job category, and class attendance. Chi-square tests were used to assess differences in proportions. Differences were considered significant at the 0.05 probability level; all reported differences are significant unless otherwise specified.

RESULTS

From 1985 to 1986, there were overall reductions in how employees perceived the risks and stresses of care of AIDS patients, despite an increase in the number of patients seen. The percentage of those who worked with AIDS patients "sometimes" or "often" rose from 65.7% to 74.6%, and the average number of AIDS patients seen over the course of the year increased from 10.8 to 17.3. However, rather than increasing with patient load, the percentage of time staff perceived they spent with AIDS patients decreased from 8.1% to 6.6%. The percentage of those who reported that dealing with AIDS patients was one of the more stressful parts of their job decreased from 48% to 34.6%.* The percentage indicating that they were uncomfortable with AIDS patients went down from 35.5% to 26%.* More staff members felt they had sufficient knowledge of patients' physical needs (75.5% compared to 61.6%), emotional needs (52% compared to 42%), and infection control procedures (the percentage without sufficient knowledge decreased from 33.6% to 9.9%). Furthermore, in 1986 only 14.5% assessed as "not good" how their fellow workers dealt with patients' physical needs, compared to 54.5% in 1985. There was a small but not significant decrease (23.9% to 20.2%) in the percentage of those who felt at high risk of getting AIDS because of their jobs.* There was a similar decline in how many assessed as "not good" the job their fellow workers did with patients' emotional needs (42.7% to 35.5%). The percentage of staff who reported that AIDS could be transmitted in each of ten different ways (Table I) also decreased.

TABLE I. Changes In Knowledge About AIDS Transmission

Can AIDS be transmitted in the following ways?	% Answering Yes* 1985 (N = 227)	1986 (N = 208)
1. by blood transfusion	93	83
2. being stuck by a needle from AIDS patient	69	63
3. giving CPR	58	36
4. kissing an individual with AIDS	44	23
5. sharing eating utensils	38	15
6. airborne transmission	26	11
7. emptying bedpans	20	9
8. changing bed linens	14	5
9. shaking hands with AIDS patient	5	1
10. being in same room with AIDS patient	5	0

* All differences are significant at the <0.05 level except for item 2.

Twenty-two percent of the respondents had attended no classes, 32.7% attended one class, and 45.3% attended two or more classes. Attendance at in-service education programs was associated with reductions in stress, perceived risk, and discomfort around patients (Table II). Class attendance did not vary by frequency of contact with AIDS patients or by length of employment. The degree of risk, stress, and discomfort reported by nonattenders of classes was greater than that reported by class attenders, and also exceeded that reported by all those surveyed in 1985.

LPNs/aides and technicians were least likely to attend any education programs. Reductions in stress and perceptions of risk and discomfort were consistent across all job categories except that of LPNs/aides, who reported slight increases in stress and discomfort (from 35% to 45%, and from 48% to 55%, respectively). Although in the same direction as the sample as a whole, reductions were smallest for technicians. Among all respondents, those who had attended two or more classes were less likely to report that AIDS could be transmitted through a needlestick injury or by changing bedpans, giving CPR, receiving blood, or sharing eating utensils.

DISCUSSION

From 1985 to 1986, staff members showed significant improvements in knowledge about AIDS transmission and reductions in the perceived stresses and risks of providing AIDS-related care, despite a more than two-fold increase in AIDS patient admissions. These changes were not experienced by those who did not attend hospital in-service programs. A sizable minority of staff continued to experience AIDS-related difficulties.

In-service programs can make a difference in how staff members respond to the demands of AIDS patient care.

TABLE II. Hospital Employees' AIDS-related Stress, Perceived Risk, and Discomfort, as Related to Attendance at In-service Education Programs

Survey Question	Sample (N=153)*	No. of Classes Attended (%) 0	1	2+	Chi-Square
Is working with AIDS one of the more stressful parts of your job?	35.2%[†]	51.5	25.0	35.3	6.36[‡]
How much risk do you think you have of getting AIDS because of your job?	20.9%	36.3	19.2	14.7	6.41[‡]
How uncomfortable are you with AIDS patients?	28.8%	54.5	26.9	17.1	15.04[§]

* Excludes responses of 31 house officers, and 24 staff members who provided incomplete survey data (these employees had little or no contact with AIDS patients).

[†] Percentages indicate those selecting response of "3" or "4" on a 1-to-4 scale.

[‡] $p < 0.05$.

[§] $p < 0.005$.

---

* Indicated by a score of 3 or 4 on a 1-to-4 scale.

Such programs are useful despite public and media attention to AIDS and consequent reductions in initial "AIDS-anxiety." Program developers should encourage participation by all employees, and pay particular attention to the needs of technical and nursing-support staff.

Acknowledgments. The authors acknowledge the efforts of Alison Sneider, Suzanne Holloran, Janet Bath, and Julie White of the New England Deaconess Hospital AIDS Education Committee.

REFERENCES

1. AIDS: A time bomb at hospitals' door. *Hospital* 1986; 60(1):54-61.
2. Ellis M: Guidelines on AIDS in bid to quell fears. *RCN Nurs Stan* 1983; 9(6):343.
3. Mather AD: AIDS update: Halting the "epidemic of fear." *Inf Rep* 1985; 2:1-3.
4. O'Donnell L, O'Donnell C, Pleck J, et al: Psychosocial responses of hospital workers to the acquired immune deficiency syndrome. *J Appl Soc Psych* (in press).
5. Phillips JL: As a nurse, I want to find out what the facts are on AIDS. *Critical Care Update* 1983; 10(9):37.
6. Wachter RM: The impact of the acquired immunodeficiency syndrome on medical residency training. *N Engl J Med* 1986; 314:177-180.

Cyclooxygenase inhibition and improved oxygenation in patients with pulmonary complications of AIDS

STEVEN H. FOSTER, MD; ROBERT C. GARRETT, MD;[†] HENRY M. THOMAS, III, MD

ABSTRACT. Pulmonary involvement related to acquired immunodeficiency syndrome (AIDS) compromises oxygenation, by both ventilation-perfusion mismatch and increased shunt. In animal studies, meclofenamate, a cyclooxygenase inhibitor, decreases shunt and improves oxygenation. Oral meclofenamate (200 mg) was given to seven patients who had AIDS with pulmonary involvement (*Pneumocystis carinii* pneumonia or Kaposi's sarcoma) and abnormal gas exchange.

Arterial Po_2 was measured 30 and 0 minutes before and 45 and 60 minutes after administering meclofenamate, and the shunt fraction (venous admixture) was estimated. In all patients, arterial Po_2 increased, shunt decreased, and Pco_2 and pH did not change after meclofenamate therapy.

Cyclooxygenase inhibition appears to have strengthened hypoxic vasoconstriction and decreased the shunt fraction, possibly by eliminating prostacyclin-induced vasodilation in diseased lung regions. Cyclooxygenase inhibition may be of benefit in the management of hypoxemia associated with AIDS and warrants further investigation.

(NY State J Med 1987; 87:280-282)

Hypoxemia is a frequent and serious complication of the pulmonary abnormalities that occur in patients with AIDS. Supplemental oxygen may not substantially increase arterial oxygenation levels because of increased blood flow through abnormal shunt pathways in the lung.

Oxygenation is improved by the diversion of blood flow away from regions of the lung in which gas exchange is impaired. Pulmonary arteries constrict in response to regional hypoxia, but the hypoxic vasoconstriction is not strong enough to prevent substantial perfusion of hypoxic regions, with resultant hypoxemia. An additional factor that may contribute to hypoxemia in these patients is the production of prostacyclin, a potent vasodilator. Prostacyclin synthesis is continuous in the lung[1] and may be increased by hypoxia.[2,3] Its vasodilatory effect opposes the ability of hypoxic vasoconstriction to divert blood flow from hypoxic regions and may further increase perfusion of the abnormal shunt pathways.

Using an animal model of regional hypoxia, it has been shown that preventing prostacyclin-induced vasodilation by inhibiting the cyclooxygenase pathway uniformly reduces perfusion of hypoxic lung tissue by as much as 40%.[4] Reported here are the results of a preliminary study of the effects of inhibition of cyclooxygenase in seven patients with hypoxemia due to pulmonary complications of AIDS.

PATIENTS AND METHODS

All patients had the diagnosis of AIDS by Centers for Disease Control (CDC) criteria[5] and had diffuse pulmonary involvement as indicated by an abnormal chest film and a calculated venous admixture greater than 10% while breathing an inspired oxygen concentration of 40% or more. In this series, pulmonary involvement was documented by fiberoptic bronchoscopy to be caused by either *Pneumocystis carinii* pneumonia (six patients) or Kaposi's sarcoma (one patient). Exclusion criteria included allergy to nonsteroidal anti-inflammatory agents, unstable cardiorespiratory status, history of peptic ulcer disease, bleeding in the upper gastrointestinal tract, and a serum creatinine level greater than 3 mg/dL. Informed consent was obtained from the subjects after the nature of the procedures had been fully explained. The investigation was approved by the Institutional Review Board of Cornell University Medical College.

An indwelling arterial needle was placed in each of these patients. For measurement of Po_2, Pco_2, and pH, a 3-mL blood sample was drawn into a heparinized syringe and was immedi-

---

From the Will Rogers Pulmonary Research Laboratory, Cornell University Medical College, White Plains and New York, NY.

Address correspondence to Dr Thomas, Director, Will Rogers Pulmonary Research Laboratory, 785 Mamaroneck Ave, White Plains, NY 10605.

This work was supported by the Will Rogers Institute, the Stony Wold-Herbert Fund, Inc, and the Potts Foundation.

[†] Deceased.

ately transported to the laboratory. Measurements were made on a Radiometer ABL1 blood gas machine within five minutes of blood sampling. The ABL1 was calibrated with an automatic internal calibration system (Radiometer) and was within the specifications of a quality control system (General Diagnostics, Blood G.A.S. Control).

The patients were started on 40% inspired oxygen; two patients required higher concentrations of 50% and 80%. A 20-minute interval was allowed for equilibration. Arterial Po_2 was measured 30 minutes before and just prior to oral administration of 200 mg of meclofenamate. Arterial blood gas measurements were repeated at 15-minute intervals for the next hour. Vital signs were monitored. Since there were no significant differences in blood gas values 30 minutes before and just prior to meclofenamate administration, values of each variable were averaged to give a premeclofenamate value; likewise, similar values at 45 and 60 minutes after meclofenamate administration were averaged to give a postmeclofenamate value. Changes produced by meclofenamate were analyzed using the paired t-test.[6]

RESULTS

Figure 1 shows the arterial Po_2 data before and after meclofenamate administration. Arterial oxygenation increased in each patient from a mean Po_2 level of 92 ± 33 mm Hg (standard deviation) (12.2 ±0.7 kPa) to 116 ± 45 mm Hg (15.5 ±6.0 kPa) ($t = 2.6$, $p < 0.05$).

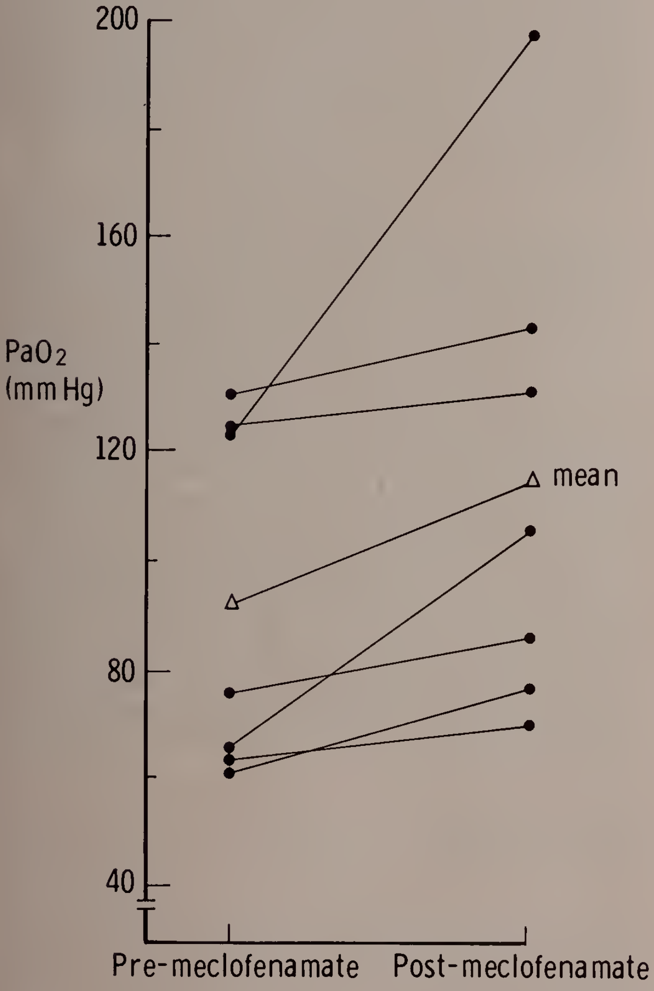


FIGURE 1. Arterial Po_2 in seven patients with pulmonary complications of AIDS, before and after administration of 200 mg meclofenamate. The mean increase is statistically significant ($t = 2.6$, $p < 0.05$).

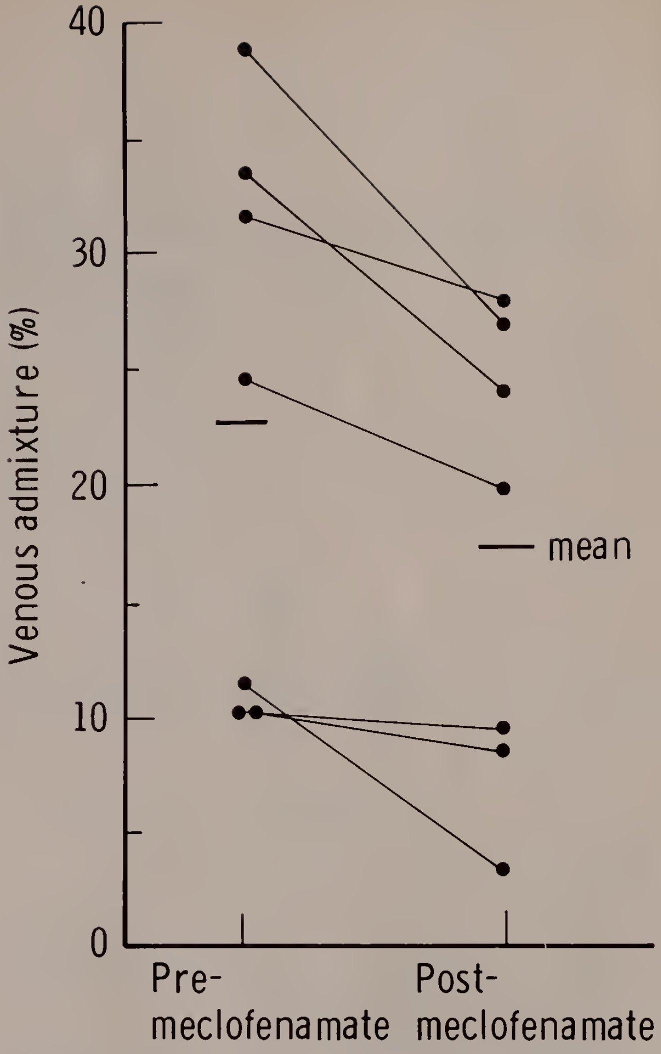


FIGURE 2. Shunt fraction (venous admixture) in seven patients with pulmonary complications of AIDS, before and after administration of 200 mg meclofenamate. The mean decrease is statistically significant ($t = 3.7$, $p < 0.01$).

To obviate any undue statistical influence of high Po_2 values on the flat portion of the oxyhemoglobin dissociation curve and to standardize the data for two patients on FIo_2 greater than 40%, the arterial oxygenation data are expressed in terms of estimated venous admixture. The shunt fraction (venous admixture) was calculated from measured values of Po_2, hemoglobin, and an assumed arteriovenous oxygen content difference. Estimated venous admixture decreased in each patient from a mean of 22.8 ±12.2% to 17.1 ±9.9% ($t = 3.7$, $p < 0.01$; Fig 2). The shunt fraction was, on average, one fourth smaller after meclofenamate therapy.

The increase in Po_2 had a time course appropriate for oral administration of meclofenamate (Fig 3). There was little increase in Po_2 at 15 minutes (+4 mm Hg; +0.5 kPa) and 30 minutes (+7 mm Hg, $p < 0.05$; +0.9 kPa), and then a substantial rise at 45 minutes (+25 mm Hg, $p < 0.10$; +3.3 kPa) and one hour (+24 mm Hg, $p < 0.05$; + 3.1 kPa).

The values of pH and Pco_2 were not affected by meclofenamate. Mean pH was 7.43 ±0.04 before and 7.43 ±0.03 after therapy; Pco_2 was 29.8 ±4.9 mm Hg (4.0 ±4.4 kPa) before and 30.7 ±4.7 mm Hg (4.1 ±0.6 kPa) after drug administration. Furthermore, heart rate, respiratory rate, blood pressure, and temperature were unchanged.

DISCUSSION

These preliminary results are encouraging. The arterial

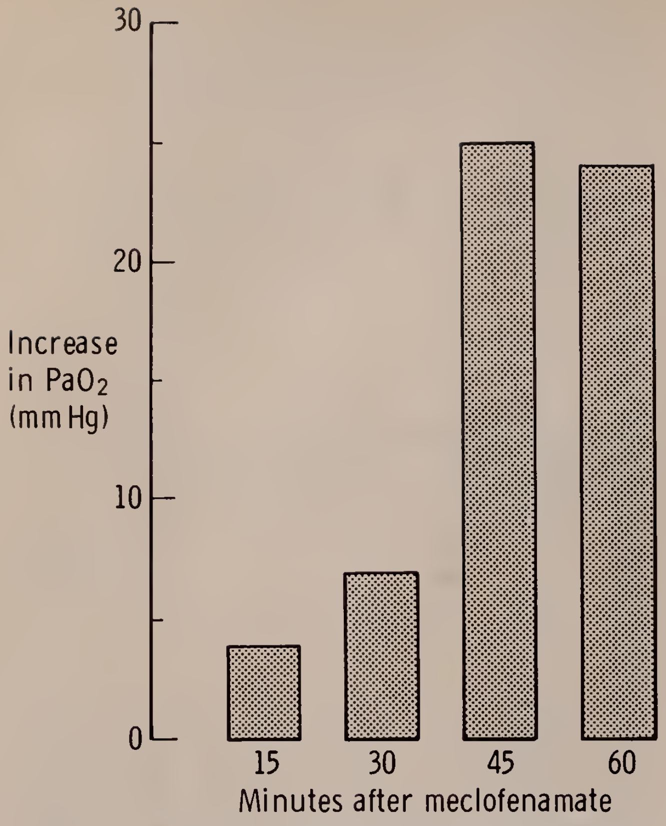


FIGURE 3. Time course of change in arterial Po_2 after administration of meclofenamate. Values are means for seven patients.

Po_2 level rose and the venous admixture fell in all seven patients. The inhibition of cyclooxygenase probably improved oxygenation by increasing the resistance to blood flow through hypoxemic lung regions.

In the absence of measurements of cardiac output and mixed venous Po_2, one cannot fully analyze the mechanism of improvement in oxygenation; for example, if cardiac output falls, shunt fraction may also fall, with a resultant elevation of Po_2.[7,8] However, in studies of anesthetized animals that received a 2-mg/kg bolus of meclofenamate, the cardiac output and mixed venous Po_2 were found to be stable.[4]

In the present study, the increase in arterial oxygenation does not appear to be due to improvement in the pathologic process in the lung: patients were selected for stability over a 24-hour period, and arterial Po_2 was then shown to be stable during the 30-minute period preceding the administration of meclofenamate.

It is possible that the effects observed are due to a property of meclofenamate not related to cyclooxygenase inhibition. However, in animal studies, several chemically unrelated cyclooxygenase inhibitors have been shown to augment the strength of hypoxic vasoconstriction and redistribute blood flow from vessels in hypoxic lung regions to vessels in normoxic lung regions.[4,9] These results suggest that in the present study, inhibition of the cyclooxygenase pathway is the most likely mechanism.

The venous admixture calculation with 40% inhaled oxygen primarily measures perfusion of shunt pathways. Diffusing capacity is frequently reduced in patients with pulmonary complications of AIDS, and it is possible that long diffusion pathways, such as those seen in the adult respiratory distress syndrome,[10,11] may produce a gas exchange abnormality almost as severe as shunt. Perfusion of low V/Q regions may also contribute to calculated venous admixture. Although the precise combination of oxygenation abnormalities in patients with AIDS is not known, reducing blood flow to regions of abnormal gas exchange appears to be beneficial.

These data suggest that in patients with AIDS who have hypoxemia, the production of vasodilator prostanoids such as prostacyclin opposes hypoxic vasoconstriction in the lung and contributes to the development of the hypoxemia. Elimination of the vasodilatory effect of prostacyclin appears to strengthen hypoxic vasoconstriction and improve oxygenation. We do not believe that these results are restricted to patients with AIDS, and they may be of value in any acute lung disease with significant shunt pathways, such as the adult respiratory distress syndrome or pneumonia. The clinical benefit to be obtained by cyclooxygenase inhibition in pulmonary complications of AIDS warrants further investigation.

REFERENCES

1. Bakhle YS, Ferreira SH: Lung metabolism of eicosanoids: Prostaglandins, prostacyclin, thromboxane, and leukotrienes, in *Handbook of Physiology, Section 3: The Respiratory System. Circulatory and Nonrespiratory Functions*. Bethesda, American Physiological Society, 1985, vol 1, pp 365-386.
2. Garrett RC, Thomas HM 3d: Relation of prostanoids to strength of hypoxic vasoconstriction in dogs with lobar atelectasis. *J Appl Physiol* 1985; 59:72-77.
3. Stephenson AH, Sprague RS, Brash DW, et al: Effects of cyclooxygenase inhibition on the renal hemodynamic response to acute hypoxia. *Fed Proc* 1986; 45:659.
4. Garrett RC, Thomas HM 3d: Meclofenamate uniformly decreases shunt fraction in dogs with lobar atelectasis. *J Appl Physiol* 1983; 54:284-289.
5. Update: Acquired immunodeficiency syndrome (AIDS)—United States. *MMWR* 1983; 32:389-391.
6. Snedecor GW, Cochran WG: *Statistical Methods*. Ames, Iowa, Iowa State University Press, 1973.
7. Smith G, Cheney FW Jr, Winter PM: The effect of change in cardiac output on intrapulmonary shunting. *Br J Anaesth* 1974; 46:337-342.
8. Breen PH, Schumacker PT, Sandoval J, et al: Increased cardiac output increases shunt. Role of pulmonary edema and perfusion. *J Appl Physiol* 1985; 59:1313-1321.
9. Hales CA, Rouse ET, Slate JL: Influence of aspirin and indomethacin on variability of alveolar hypoxic vasoconstriction. *J Appl Physiol* 1978; 45:33-39.
10. King TK, Weber B, Okinaka A, et al: Oxygen transfer in catastrophic respiratory failure. *Chest* 1974; 65:40S-44S.
11. Lamy M, Fallat RJ, Koeniger E, et al: Pathologic features and mechanisms of hypoxemia in adult respiratory distress syndrome. *Am Rev Respir Dis* 1976; 114:267-284.

Intravenous drug use and the heterosexual transmission of the human immunodeficiency virus

Current trends in New York City*

DON C. DES JARLAIS, PHD; ERIC WISH, PHD; SAMUEL R. FRIEDMAN, PHD; RAND STONEBURNER, MD, MPH;
STANLEY R. YANCOVITZ, MD; DONNA MILDVAN, MD; WAFAA EL-SADR, MD;
ELIZABETH BRADY, MA; MARY CUADRADO, MA

Women, and men who deny homosexual activity, account for slightly more than one third (3,929/7,696) of the cases of acquired immunodeficiency syndrome (AIDS) in New York City through September 1986.[1] This percentage has been rising during the course of the epidemic. Intravenous (IV) drug users account for more than half (2,261/3,929) of these heterosexual cases, and an additional 134 cases have occurred in persons known to be heterosexual partners of IV drug users. The connections between AIDS, IV drug use, and the heterosexual transmission of the human immunodeficiency virus (HIV) pose one of the more difficult public health challenges facing the city and the country. In this paper we review data relevant to two questions: potential heterosexual transmission among IV drug users, and potential transmission from IV drug users to heterosexual partners who do not inject drugs.

Before reviewing data on these two questions, however, it is useful to note the history of AIDS among IV drug users in New York City. The first cases of AIDS among IV drug users were retrospectively diagnosed as having occurred in 1980.[2] Retrospective studies of sera of IV drug users revealed that the first HIV antibody positive sample was collected in 1978.[3] The trends in IV drug use and heterosexual transmission of HIV in New York City are based on at least an eight-year history of infection among IV drug users who were also active heterosexuals. While attention to heterosexual transmission in the United States is relatively new, there is actually almost a decade of experience with opportunities for heterosexual transmission. This length of time allows some relatively firm conclusions to be drawn about the dynamics of transmission related to IV drug use and heterosexual behavior.

HETEROSEXUAL TRANSMISSION AMONG IV DRUG USERS

The national system used for classifying cases of AIDS tends to obscure both IV-drug-related and heterosexual transmission of HIV. Male IV drug users who also engage in homosexual behavior are often simply classified into the "homosexual/bisexual male" category, and heterosexuals who inject drugs are classified in the "IV drug use" category. This classification system tends to miss cases of IV-drug-related transmission among homosexual/bisexual men and cases of heterosexual transmission between persons who inject drugs. To study the question of potential heterosexual transmission among IV drug users, we examined the relationships between drug injection and sexual behavior in a cohort of IV drug users from the borough of Manhattan. This cohort has been previously described.[4]

A slightly higher rate of HIV infection was found among females compared to males—58% of the females were seropositive compared to 48% of the males. Since male-to-female sexual transmission of HIV may be more efficient than female-to-male transmission, these rates would be consistent with the existence of an added risk for heterosexual transmission above the risk of drug injection transmission. The difference, however, was not significant after controlling for the frequency of drug injection. Parallel findings were also obtained by Weiss et al[5] in New Jersey. They also found a slightly higher seroprevalence rate among females that was not statistically significant after controlling for drug injection frequency.

We examined a variety of sexual behaviors as possible predictors of HIV exposure among this cohort of IV drug users, including male homosexuality and, for the females, the number of IV-drug-using sexual partners. None of these sexual behaviors was significantly associated with HIV infection when drug injection behavior was con-

---

From the New York State Division of Substance Abuse Services (Dr Des Jarlais), Narcotic and Drug Research, Inc (Drs Wish and Friedman, Ms Brady, and Ms Cuadrado), the New York City Department of Health (Dr Stoneburner), Beth Israel Medical Center (Drs Yancovitz and Mildvan), and Manhattan Veterans Administration Medical Center (Dr El-Sadr).

Address correspondence to Dr Des Jarlais, New York State Division of Substance Abuse Services, 55 W 125th St, New York, NY 10027.

The research presented in this paper was supported primarily through grant R01 DA 03574 from the National Institute on Drug Abuse. Additional support was provided by grant 85-IJ-CX-0025 from the National Institute of Justice.

* This article is based on a presentation at the "Symposium on the Heterosexual Transmission of AIDS," sponsored by the Department of Epidemiology and Social Medicine, Montefiore Medical Center and Albert Einstein College of Medicine, and held October 21, 1986, at the New York Academy of Medicine.

trolled for. Twelve females reported engaging in prostitution in the year prior to the interview. Ten of these were HIV seropositive. While the women who reported engaging in prostitution did have a higher seroprevalence rate than those who denied engaging in prostitution, this was also not statistically significant when the frequency of drug injection was entered into the analysis. The probability level associated with prostitution in the multivariate analysis was 0.21, however, which does not lead one to completely reject a hypothesis that engaging in prostitution is associated with higher rates of seropositivity among female IV drug users. Schoenbaum and colleagues[6] did find that engaging in prostitution was a significant predictor of HIV exposure in their sample of IV drug users from the Bronx, though not as strong a predictor as the drug injection variables.

One conclusion from the available data is that, to date, heterosexual transmission among IV drug users is at most a slight added risk compared to the risk of HIV infection from the sharing of equipment for drug injection. This added risk would seem to be greater for female IV drug users who engage in prostitution. Even in this instance, extreme care must be used in drawing any conclusion about the sources of this potential added risk. Many IV-drug-using females who engage in prostitution have customers who inject drugs (D. Strug, personal communication, 1985). They also frequently have boyfriends who inject drugs, and the use of condoms and other forms of "safer sex" are particularly unlikely with these boyfriends (J. Cohen, personal communication, 1986). It is also possible that the female prostitutes have slightly different patterns of drug injection behavior that were not identified in the interviews but were the underlying reason for higher rates of seropositivity, or that the effects of frequent infection with sexually transmitted diseases make them more susceptible to heterosexual transmission.

The hierarchical risk classification system may result in the misclassification of some AIDS cases as being related to IV drug use when transmission actually occurred through heterosexual activity. Comparisons of sexual behavior and IV drug use behavior as predictors of HIV exposure, however, indicate that such misclassifications are currently infrequent in New York City.

HETEROSEXUAL TRANSMISSION FROM MALE IV DRUG USERS TO WOMEN WHO DO NOT INJECT DRUGS

Comparisons of the relative risk of developing AIDS among females who inject drugs versus females who do not inject drugs but are the heterosexual partners of male IV drug users also suggest that, to date, the risk of heterosexual transmission is relatively small compared to the risk of transmission through sharing equipment for IV drug use.

Such a comparison was first made in September 1984, at which time there had been 132 cases of AIDS among female IV drug users and 25 cases among females who did not inject drugs but were heterosexual partners of IV drug users.[7] In calculating the relative risks, one must first estimate the sex ratio among IV drug users and the ratio of male IV drug users to their heterosexual partners who do not inject drugs. Based on studies of more than 10,000 persons entering treatment for heroin abuse in New York City in 1983, the percentage of male IV drug users was estimated as 73%, giving a male-to-female ratio of 2.70:1. This high male-to-female ratio indicates that the majority of male IV drug users must be having sexual relationships with females who do not inject drugs. A survey of 50 male IV drug users admitted to detoxification treatment programs showed a total of 53 regular heterosexual partners, of whom 40 were reported not to inject drugs. This gave a ratio of 1:0.80 male IV drug users to female heterosexual partners, and a ratio of 1:2.16 female IV drug users to females who are heterosexual partners of IV drug users but do not inject drugs themselves.

The relative risk of AIDS among female IV drug users compared to female heterosexual partners was estimated as 11 to 1 in September 1984. Through September 1986, 472 cases of AIDS among female IV drug users and 134 cases among female heterosexual partners had been reported to the New York City Department of Health.[8] Using the same ratio of 1:2.16 of female IV drug users to female heterosexual partners gives a relative risk of 7.8:1 for female IV drug users compared to female heterosexual partners of male IV drug users. Both of these ratios suggest that, to date, the risk of developing AIDS from heterosexual contact is relatively small compared to the risk from IV drug use.

Both the data on sexual behavior as a predictor of seropositivity and the data on the relative risk of AIDS among female IV drug users compared to female heterosexual partners indicate that, to date, heterosexual transmission has been relatively infrequent compared to IV-drug-use transmission.

An examination of the available data on AIDS and prostitution in New York City also leads to the same conclusion.

As a first step in this argument, evidence suggests that many "street-worker" prostitutes are IV drug users. Interviews were conducted with 95 female prostitutes who had been arrested in New York City.[9] (These street-workers should not be considered representative of call girls or prostitutes working through escort services.) Intravenous drug use was common in this sample of arrested prostitutes; 40 (42%) reported histories of drug injection, and urinalysis revealed that 28% were positive for opiates and 69% for cocaine.

Second, many prostitutes who use IV drugs are HIV seropositive. Because the women referred to above were interviewed in a jail facility, it was not possible to obtain blood samples. Wallace and colleagues[10] have conducted a serosurvey of street-working prostitutes and found that half of those who injected drugs were HIV antibody positive. This is consistent with the above-noted findings of high seropositivity among female IV drug users who engaged in prostitution.[2,6]

In spite of this, however, there have been few AIDS cases in New York City among men who lack other risk factors. Given the length of time that HIV has been present among IV drug users in New York City, and the substantial proportion of street prostitutes who inject drugs and are seropositive, there has been very little apparent transmission from prostitutes to their customers. More than 8,000 cases of AIDS have been reported to the

New York City Department of Health; only 39 occurred in men in whom no risk factor is identified, and in fewer than half of these is contact with prostitutes claimed as a risk factor.[11]

Further evidence for the infrequency of prostitute-to-customer transmission comes from a study by Rabkin and colleagues.[12] They examined HIV seropositivity in a sample of men attending a clinic for sexually transmitted diseases in New York City. They specifically examined contact with prostitutes as a potential risk factor, but it was not a statistically significant predictor of HIV exposure.

One possible explanation for the apparently low rate of transmission of HIV from prostitutes to customers in New York is the use of "safe sex" procedures. The great majority—62/67 (92%)—of the female prostitutes interviewed in jail reported that they usually required their customers to use condoms. (Questions about safe sex procedures were added to the interview after the first 28 subjects were interviewed.) When the customer did not want to use a condom, the prostitute would charge extra and/or deceive him by substituting manual masturbation. These subjects also reported that oral sex was the predominant activity with their customers, and while HIV has been found in saliva, infection from saliva appears to be very rare if it occurs at all.[13]

Our interviews with key informant ex-prostitutes indicate that these safe-sex practices were adopted in the early 1970s, certainly before HIV was widely spread among IV drug users in New York City. General concern about sexually transmitted diseases, including herpes, was the main stated reason for the increase in the use of condoms at that time. It should also be noted that sexually transmitted diseases pose a significant economic threat to prostitutes, particularly those with regular customers.

DISCUSSION

It is likely that HIV was first introduced into the intravenous drug user group in New York City during the middle to late 1970s. Since then the virus has spread among this group through the sharing of equipment for injecting drugs and through homosexual and heterosexual contact. We have examined sets of data that compared drug-injection-related transmission to heterosexual transmission in seroprevalence studies of IV drug users and that compared the relative risk of developing AIDS among females who inject drugs and females who do not inject drugs but are sexual partners of men who do inject drugs. These comparisons suggest that, to date in New York City, transmission related to IV drug use has been approximately an order of magnitude more frequent than male-to-female heterosexual transmission. The available data on prostitution indicate that prostitutes who inject drugs are relatively likely to have been exposed to HIV, but to date there has been relatively little transmission to customers.

There are several possible reasons why there has been relatively little transmission through heterosexual activity compared to the sharing of drug injection equipment to date in New York City. First, the efficiency of transmission through sharing of drug equipment may simply be much greater than that for unprotected vaginal intercourse. Second, even if the relative efficiencies of transmission were approximately equal, the observed differences might also be explained in terms of two variables that are key to rapid transmission during the early stages of an epidemic: the frequency with which an individual performs acts likely to transmit the virus, and the number of different persons with whom the individual practices those acts. At least prior to the awareness of AIDS, many IV drug users injected several times per day at so-called "shooting galleries"—where rented equipment permitted sharing with large numbers of anonymous partners. In New York, these IV drug users engaging in high-frequency sharing of equipment were also likely to be sharing with each other, regardless of sex or sexual orientation. One would thus expect a relatively rapid transmission of HIV among those persons frequently engaging in high-risk behavior with multiple partners.

Regarding heterosexual transmission, there are certainly some persons who average several different partners per day, but most of these are likely to be prostitutes, who generally seem to have been using "safer sex" procedures since before the AIDS epidemic began. While there are few data available on the levels of male heterosexual activity in New York, it is doubtful that there are large numbers of men who average several different heterosexual partners per day over long periods of time. Thus, women with high numbers of heterosexual partners are relatively likely to be taking precautions against sexual transmission, and there are probably relatively few men with high numbers of heterosexual partners. Heterosexual transmission is therefore likely to occur at a much slower rate.

Given that an HIV-infected person may be infectious for an indefinite period of time, the slower initial spread of HIV through heterosexual transmission compared to drug equipment transmission tells us little about whether heterosexual transmission will eventually become more frequent. Most heterosexual transmission that is currently occurring in New York is probably between IV drug users and their partners. Efforts are urgently needed to prevent more IV drug users from being infected with HIV. Prevention of further HIV infection among IV drug users will also prevent heterosexual transmission. Indeed, prevention of HIV transmission from shared drug injection equipment may be critical for the eventual control of heterosexual transmission in New York City. Effective means of preventing transmission to persons who are current or potential sexual partners of IV drug users are also urgently needed.

A number of such projects for preventing drug injection transmission and heterosexual transmission from IV drug users are currently under way. These include expanded drug treatment programs to help drug users stop using drugs, and education within these programs so that clients who continue to use drugs will be able to reduce their risks. Street outreach education and antibody-testing and counseling projects are targeting IV drug users and their sex partners. There are also attempts to organize drug users to help each other make it less likely that they will share injection equipment.[14,15] The newness of these projects does not permit any systematic assessment of their relative or absolute effectiveness. Research will be needed not only to evaluate specific programs, but also to develop a conceptual understanding of the behavior changes needed to interrupt HIV transmission through both drug injection equipment and heterosexual activity.

In addition to these prevention projects oriented towards drug users and their sexual partners, education of the entire population with respect to preventing heterosexual transmission will also be needed on a long-term basis.

REFERENCES

1. New York City Department of Health AIDS Surveillance, September 1986.

2. Des Jarlais DC, Friedman SR, Hopkins W: Risk reduction for the acquired immunodeficiency syndrome among intravenous drug users. *Ann Intern Med* 1985; 103:755–759.

3. Novick DM, Kreek MJ, Des Jarlais DC, et al: Antibody to LAV among intravenous drug users: Historical, therapeutic and ethical aspects, in Harris L (ed): *Problems of Drug Dependence 1985: Proceedings of the 47th Annual Scientific Meeting.* NIDA Research Monograph 67. Washington, DC, The Committee on Problems of Drug Dependence, Inc, 1985, pp 318–320.

4. Cohen H, Marmor M, Des Jarlais DC, et al: Risk factors for HTLV-III/LAV seropositivity among intravenous drug users. Presented at the International Conference on the Acquired Immunodeficiency Syndrome (AIDS), Atlanta, Ga, April 14–17, 1985.

5. Weiss SH, Ginzburg HM, Goedert JJ, et al: Risk for HTLV-III exposure and AIDS among parenteral drug abusers in New Jersey. Presented at the International Conference on the Acquired Immunodeficiency Syndrome (AIDS), Atlanta, Ga, April 14-17, 1985.

6. Schoenbaum EE, Selwyn PA, Klein RS, et al: Prevalence of and risk factors associated with HTLV-III/LAV antibodies among intravenous drug abusers in a methadone program in New York City. Presented at the International Conference on AIDS, Paris, France, June 23-25, 1986.

7. Des Jarlais DC, Chamberland ME, Yancovitz SR, et al: Heterosexual partners: A large risk group for AIDS [letter]. *Lancet* 1984; 2:1346–1347.

8. New York City Department of Health Bureau of Health Statistics, 1986.

9. Wish ED, Johnson BD: The impact of substance abuse on criminal careers, in Blumstein A, Cohen J, Visher CA (eds): *Criminal Careers and Career Criminals.* Washington, DC, National Academy Press, 1986, vol 2, pp 52–58.

10. Wallace JI, Christonikos N, Merlink R, et al: HTLV-III/LAV exposure in New York City prostitutes. Presented at the International Conference on AIDS, Paris, France, June 23-25, 1986.

11. New York City Department of Health AIDS Surveillance, 1986.

12. Rabkin CS, Thomas PA, Jaffe H, et al: Prevalence of antibody to HTLV-III/LAV in a population attending a sexually transmitted disease clinic. *Sex Transm Dis* 1986; 13:80–83.

13. Friedland G, Saltzman B, Rogers M, et al: Lack of transmission of HTLV-III/LAV infection to household contacts of patients with AIDS or AIDS-related complex with oral candidiasis. *N Engl J Med* 1986; 314:344–349.

14. Friedman SR, Des Jarlais DC, Goldsmith DS: An overview of current AIDS prevention efforts aimed at intravenous drug users. *J Drug Issues* (in press).

15. Friedman SR, Des Jarlais DC, Sotheran JL, et al: AIDS and self-organization among intravenous drug users. *Int J Addict* (in press).

AIDS in Africa: Evidence for heterosexual transmission of the human immunodeficiency virus*

THOMAS C. QUINN, MD

Since its initial recognition in 1981, acquired immunodeficiency syndrome (AIDS) has become a global epidemic. Nearly 40,000 cases have been reported from over 85 countries on five different continents. Presently, an estimated 5–10 million people worldwide have been infected with the causative agent, referred to as the human immunodeficiency virus (HIV).[1,2] From natural history studies, it can be expected that at least 10–30% of these HIV-infected individuals may develop AIDS within the next three years,[3–6] resulting in a cumulative number of more than one million cases of AIDS worldwide by 1990. With the present lack of effective curative therapy or a vaccine, this disease now represents one of the most serious epidemics of the century.

SURVEILLANCE FOR AIDS IN AFRICA

Shortly after the recognition of AIDS in the United States, cases were identified among Africans residing in Europe.[7,8] In contrast to European cases, with a male-to-female ratio of 16:1, African cases had a male-to-female ratio of 1.7:1, and 90% of affected individuals had no identifiable risk factors.[9] This recognition of African AIDS cases with unusual epidemiologic features prompted a series of investigations in Africa.[10,11] Initially limited to urban areas of Central Africa, recent reports have documented the spread of AIDS to nearly all countries of Africa, with major foci primarily in Central and East African countries.[12–15] Unfortunately, accurate statistics regarding the number of AIDS cases are not available, since surveillance has only been instituted within the past six months in several African countries and remains nonexistent in other countries. Presently, 19 African countries have officially reported over 2,000 cases of AIDS to the World Health Organization. In 1985, in Kigali, Rwanda, and Kinshasa, Zaire, it was estimated that the annual incidence of AIDS for adult residents was approximately 500–1,000 cases/million adults.[11,14] Unlike in the United States, where the sex ratio is 13:1 men to women, the sex ratio of AIDS cases in Africa is now 1:1.[14] As in developed countries, AIDS in Africa primarily affects young and middle-aged persons. The mean age of AIDS patients in Kinshasa was 33.6 years (mean, 32 years; range, 1.5–64 years), and men were significantly older than women—mean ages, 37.4 years and 30.0 years, respectively. Peak age-specific incidence rates of 786/million and 601/million were present among 30–39-year-old men and women,

---

Dr Quinn is Senior Investigator, Laboratory of Immunoregulation, National Institutes of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, Md, and Associate Professor of Medicine, Johns Hopkins University School of Medicine, Baltimore, Md.

Address correspondence to Dr Quinn, Division of Infectious Diseases, Blalock 1111, The Johns Hopkins Hospital, Baltimore, MD 21205.

* This article is based on a presentation at the "Symposium on the Heterosexual Transmission of AIDS," sponsored by the Department of Epidemiology and Social Medicine, Montefiore Medical Center and Albert Einstein College of Medicine, and held October 21, 1986, at the New York Academy of Medicine.

respectively.[14] These sex and age distributions of AIDS cases reflect patterns seen with other sexually transmitted diseases, both in developed and developing countries, in which the incidence and morbidity rates are higher among younger women and older men.[12]

In addition to the equal sex distribution of cases, other evidence that suggested heterosexual transmission was the fact that women with AIDS were more likely than men to be unmarried (61% versus 36%), and nearly one third of the married AIDS patients had had at least one previous marriage or "union libre" (persistent cohabitation without formal marriage).[14] One third of AIDS patients reported having had at least one sexually transmitted disease during the three years preceding their illness. In many areas of Africa, a large proportion of the AIDS cases comprise female prostitutes, or men and women with a history of multiple sexual partners within the previous five years.[10,11,13,17-19] However, additional factors that may influence transmission of HIV include frequent exposure to unsterilized needles (80% of AIDS cases), and a history of blood transfusion within the previous three-year period prior to the onset of illness (9%).[14] More recent case-control studies have further demonstrated that AIDS is primarily transmitted in Africa by heterosexual activity, blood transfusions, exposure to blood-contaminated needles and syringes for medical or ritual purposes, and via perinatal transmission from mother to infant.[12,18-23]

Seroprevalence data on HIV infection suggest that of these different modes of transmission, heterosexual transmission appears to be the predominant mode of infection in Africa. In a recent study of HIV infection among 6,000 healthy persons residing in Kinshasa, Zaire, a bimodal distribution of infection was evident with a peak prevalence under one year of age and among young adults aged 16–29 years (Fig 1).[12] The first peak in seroprevalence among children under one year of age represented a combination of passive antibody transfer and transmission of virus from mother to infant. The low prevalence rate between ages 2 and 14 argues strongly against arthropod transmission, since arthropod-transmitted diseases usually have peak infection rates among these age groups.[24] As sexual activity increased during adolescence, the rate of HIV infection increased dramatically, particularly among women aged 15–29 years. This increase was followed by a rise in HIV infection among men between the ages of 20 and 39 years. This pattern, in conjunction with the distribution of AIDS cases, is consistent with that of a sexually transmitted disease with higher prevalence rates among younger, sexually active women having sexual relations with older men.[12,16]

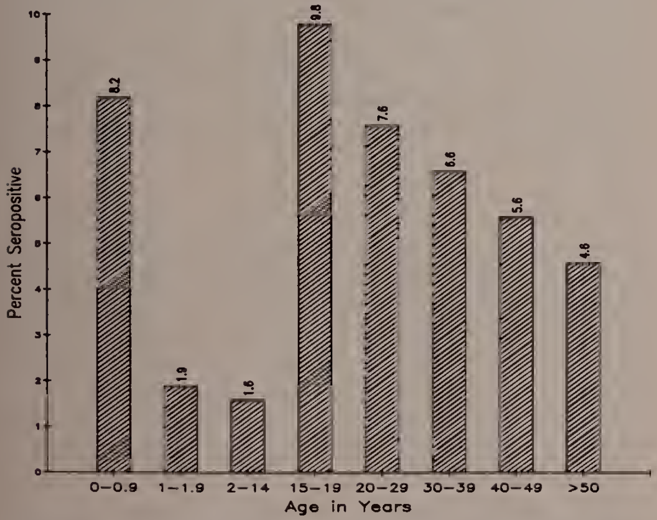


FIGURE 1. HIV seroprevalence rates among 6,000 healthy persons by age in Kinshasa, Zaire, 1984–1986.

RISK FACTORS FOR HETEROSEXUAL TRANSMISSION

Risk factors associated with HIV infection among heterosexuals residing in Africa include the number of sexual partners, having sex with prostitutes, being a prostitute, and being a sexual partner of an infected person.[12] In Africa, prostitutes show the highest rate of infection (27–88%),[13,18] and they may have played an important role in the dissemination of HIV in areas such as Nairobi, where HIV antibody prevalence was initially higher in prostitutes than in men with sexually transmitted diseases who frequented the prostitutes.[13] In a recent study by Piot et al,[26] HIV seroprevalence rates among prostitutes in Nairobi, Kenya, rose from 4% to 59% between 1980 and 1986. This rapid dissemination of HIV infection in a high-risk group in Africa was similar to that observed among homosexually active men in San Francisco during the same time period.[3,5] This rise in infection among the prostitutes was subsequently followed by a steady increase in seroprevalence rates, from 1% to 18%, among men attending clinics for treatment of sexually transmitted diseases (STD) in Nairobi during the same time period.[26]

HIV infection among female prostitutes correlated with socioeconomic status and mean number of sexual encounters per year. For example, a seroprevalence rate of 66% was present among prostitutes of low socioeconomic status who averaged 963 sexual encounters per year, compared to a prevalence rate of 31% among prostitutes of higher socioeconomic status who averaged 124 sexual encounters per year.[13] In this same study, sexual exposure to men from Central Africa was significantly associated with HIV infection among these female prostitutes, suggesting transcontinental spread of the epidemic into East Africa. Similarly, the history of sexual exposure to these female prostitutes correlated with infection in Kenyan men attending the STD clinics, suggesting initially male-to-female followed by female-to-male bidirectional transmission.[13]

In further studies of HIV infection among the Nairobi prostitutes and the men attending the STD clinics, HIV seropositivity was significantly associated with current infection with sexually transmitted diseases such as gonorrhea, genital ulcers, and syphilis.[13,26] In a study in Zambia, seropositivity in men also correlated with the presence of genital ulcers.[15] These observations suggest that the disruption of genital epithelial integrity caused by sexually transmitted diseases that are common in Africa may facilitate transmission of HIV during vaginal intercourse. Alternatively, the presence of these sexually transmitted diseases may be indicative of high-risk activities for HIV infection or of exposure to unsterilized needles for the treatment of sexually transmitted diseases, and may not be causally related to HIV transmissability. However, in two studies of female prostitutes and of men attending STD clinics in Africa, HIV seropositivity was directly associated with the number of sexual exposures independent of the frequency of needle exposures—which was not associated with HIV seropositivity.[13,25]

Further evidence for heterosexual transmission comes from a household study of AIDS patients in Zaire, in which HIV antibody was significantly higher among spouses than among other household members or controls. In this study by Mann et al,[27] 20 (9.8%) of 204 case household members and three (1.9%) of 155 control household members were HIV seropositive (relative risk = 5.1; 95% confidence interval, 1.7–15.2). Eleven (three men and eight women) (61%) of 18 spouses of three women and fifteen men with AIDS were HIV antibody positive compared to only one (3.7%) of 27 spouses of seronegative control patients (relative risk = 16.5; 95% confidence interval, 3.7–75.0). Except for spouses, the rate of HIV seropositivity did not differ significantly between case and control households. Furthermore, of adults in case households who were not spouses, the number who were seropositive for HIV was identical to that predicted from sex- and age-specific HIV seroprevalence rates. These data document the potential for heterosexual transmission among sexual partners of HIV-infected individuals, and that bidirectional transmission is possible. In addition to this study, several clusters of AIDS cases in Africa have been identified in the chronology of events suggesting both female-to-male and male-to-female transmission of HIV.[10,28]

Unlike HIV infection among homosexual men in the United States and Europe, specific sexual activities other than vaginal intercourse do not appear to play a significant role in heterosexual transmission. Anal intercourse, reported by only 4–8% of female AIDS patients, was not associated with HIV infection in surveys in Kenya, Zaire, and Rwanda.[13,17-19,25] Clearly, the most important variable for heterosexual transmission is the number of sexual exposures within a given time period. In a recent study of female prostitutes in Zaire by Mann et al,[25] HIV seroprevalence rates were 19% for those women with fewer than 200 sexual partners per lifetime, which increased steadily to a high of 34% for those women with a history of more than 1,000 lifetime sexual partners (Fig 2). The only variable that had a negative effect on seropositivity was a history of condom use. Although the numbers are small, none of eight female prostitutes who insisted their partners use condoms were seropositive, compared to an infection rate of 33.8% among female prostitutes whose partners used condoms less than half of the time. These data provide indirect evidence that even with promiscuity, barrier contraceptives such as the condom may effectively prevent transmission of HIV.

OTHER MODES OF HIV TRANSMISSION

Heterosexual transmission of HIV in Africa is amplified by further dissemination of the AIDS virus into the general heterosexual population via blood transfusions and injections with blood-contaminated needles for medicinal purposes. In some areas of Africa, such as Uganda, Rwanda, and Zaire, seroprevalence rates among blood donors range from 8% to 18%, suggesting that the risk to transfusion recipients in these areas is very high. In Kinshasa, 9% of 295 AIDS patients reported receiving at least one blood transfusion during the three to five years prior to onset of illness.[22] Among 2,384 hospital workers in Kinshasa, 9.3% of seropositive workers and 4.8% of seronegative workers reported transfusions within the past ten years ($p < 0.05$).[21] Even among children in Central Africa, 60% of 40 seropositive children compared to 32.7% of 328 seronegative children and 14.3% of 92 healthy outpatient control children had a history of prior blood transfusions ($p < 0.001$).[20]

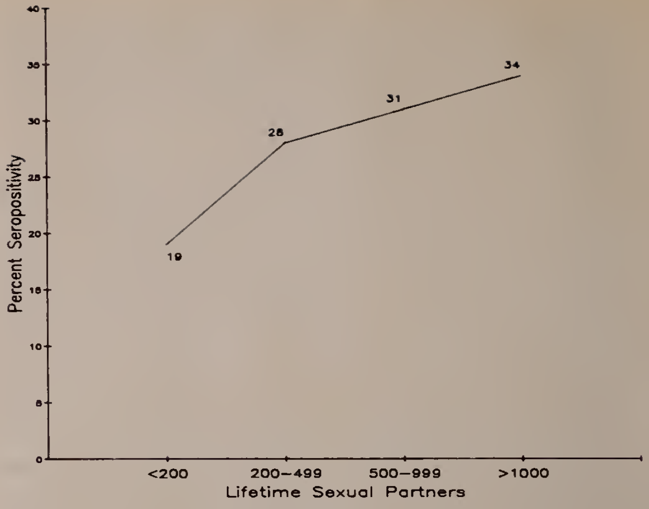


FIGURE 2. HIV seroprevalence rates in 377 female prostitutes according to mean number of lifetime sexual partners, Zaire, 1986.

Similarly, in the same seroprevalence study of hospital workers in Kinshasa,[21] significantly more HIV seropositive than seronegative workers reported receiving medical injections during the previous three years. Among those reporting injections, seroprevalence was nearly twice as high for those who received five or more injections as for those receiving fewer than five injections. Together, these two additional factors, blood transfusions and exposure to blood-contaminated needles and syringes, contribute significantly to the dissemination of HIV among the general population. With increasing prevalence of HIV infection in the general population, heterosexual transmission becomes more efficient, and promiscuity becomes a less important variable.

CONCLUSIONS

The present available data in Africa demonstrate that heterosexual transmission exists, and that it can, given the right circumstances, sustain an epidemic of HIV infection in the general population. The rate at which HIV infection will spread by heterosexual contact outside Africa depends on many factors, such as the prevalence of HIV in the heterosexual population, its introduction and amplification in a highly promiscuous subpopulation of heterosexuals, the degree of sexual promiscuity, the efficiency of heterosexual transmission, and the prevalence of genital infections and use of barrier contraceptives.[29] Most importantly, the long latency period between viral exposure and the development of clinical disease harbors the delayed recognition of the epidemic in a heterosexual population. Thus, it is appropriate to institute studies to determine the occurrence of HIV infection in heterosexual populations in the United States known to be at high risk, such as sexual partners of individuals with AIDS or at risk for AIDS, heterosexual prostitutes, and patients with sexually transmitted diseases. Educational programs on AIDS prevention should be instituted immediately and should be directed at heterosexuals as well as at other high-risk groups. The major emphasis of these programs should be on reducing risk factors and on safer sexual practices, including condom use. For Africa, the obstacles to AIDS control are even greater and include cultural, sociologic, and economic factors. To be successful, these programs will have to be integrated into the existing health and educational programs and will require full support by appropriate government agencies. Controlling the spread of HIV worldwide will require a major international commitment, not only in terms of providing financial help, but also in providing scientific, educational, and technical assistance.

References

1. Mahler H, Assad F: The World Health Organization's programme on AIDS. *Abstracts of the Second International Conference on AIDS*, Paris, France, June 23-25, 1986, p 5.
2. Coffin J, Haase A, Levy JA, et al: Human immunodeficiency viruses [letter]. *Science* 1986; 232:697.
3. Curran JW, Morgan WM, Hardy AM, et al: The epidemiology of AIDS: Current status and future prospects. *Science* 1985; 229:1352-1357.
4. Melbye M: The natural history of human T lymphotropic virus-III infection: The cause of AIDS. *Br Med J* 1986; 292:5-12.
5. Jaffe HW, Darrow WW, Echenberg DF, et al: The acquired immunodeficiency syndrome in a cohort of homosexual men: A six-year follow-up study. *Ann Intern Med* 1985; 103:210-214.
6. Goedert JJ, Biggar RJ, Weiss SH, et al: Three-year incidence of AIDS in five cohorts of HTLV-III infected risk group members. *Science* 1986; 231:922-995.
7. Clumeck N, Sonnet J, Taelman H, et al: Acquired immunodeficiency syndrome in African patients. *N Engl J Med* 1984; 210:492-497.
8. Brunet JB, Ancelle R: Update: Acquired immunodeficiency syndrome—Europe. *MMWR* 1985; 34:583-589.
9. World Health Organization: Acquired immunodeficiency syndrome (AIDS): Situation in Europe as of 31 December 1985. *Wkly Epidemiol Rec* 1986; 61:125-128.
10. Piot P, Quinn TC, Taelman H, et al: Acquired immunodeficiency syndrome in a heterosexual population in Zaire. *Lancet* 1984; 2:65-69.
11. Van de Perre P, Rouvroy D, Lepage P, et al: Acquired immunodeficiency syndrome in Rwanda. *Lancet* 1984; 2:62-65.
12. Quinn TC, Mann JM, Curran JW, et al: AIDS in Africa: An epidemiological paradigm. *Science* 1986; 234:955-963.
13. Kreiss JK, Koech D, Plummer FA, et al: AIDS virus infection in Nairobi prostitutes: Spread of the epidemic to East Africa. *N Engl J Med* 1986; 314:414-418.
14. Mann JM, Francis H, Quinn T, et al: Surveillance for AIDS in a Central African city: Kinshasa, Zaire. *JAMA* 1986; 255:3255-3259.
15. Melbye M, Njelesani EK, Bayley A, et al: Evidence for heterosexual transmission and clinical manifestations of human immunodeficiency virus infection and related conditions in Lusaka, Zambia. *Lancet* 1986; 2:1113-1115.
16. Aral SO, Holmes KK: Epidemiology of sexually transmitted diseases, in Holmes KK, Mardh PA, Sparling PF, et al (eds): *Sexually Transmitted Diseases*. New York, McGraw-Hill, pp 127-141.
17. Clumeck N, Robert-Guroff M, Van de Perre P, et al: Seroepidemiological studies of HTLV-III antibody prevalence among selected groups of heterosexual Africans. *JAMA* 1985; 254:2599-2602.
18. Clumeck N, Van de Perre P, Carael M, et al: Heterosexual promiscuity among African patients with AIDS. *N Engl J Med* 1985; 313:182.
19. Van de Perre P, Clumeck N, Carael M, et al: Female prostitutes: A risk group for infection with human T-cell lymphotropic virus type III. *Lancet* 1985; 2:524-527.
20. Mann JM, Francis H, Davachi F, et al: HTLV-III/LAV seroprevalence in pediatric in-patients 2-14 years old in Kinshasa, Zaire. *J Pediatrics* 1986; 78:673-677.
21. Mann JM, Francis H, Quinn TC, et al: HTLV-III/LAV seroprevalence among hospital workers in Kinshasa, Zaire: Lack of association with occupational exposure. *JAMA* 1986; 256:3099-3102.
22. Mann JM, Francis H, Quinn TC, et al: Role of blood transfusions in transmission of HTLV-III infection in Zaire. *Am J Pub Health* (in press).
23. Mann JM, Francis H, Davachi F, et al: Risk factors for human immunodeficiency virus seropositivity among children 1-24 months old in Kinshasa, Zaire. *Lancet* 1986; 2:654-657.
24. Zuckerman AJ: AIDS and insects. *Br Med J* 1986; 292:1094-1095.
25. Mann JM, Quinn TC, Francis H: Sexual practices associated with LAV/HTLV-III seropositivity among female prostitutes in Kinshasa, Zaire. Presented at the International Conference on AIDS, Paris, France, June 23-25, 1986.
26. Piot P, Plummer FA, Rey M, et al: Retrospective seroepidemiology of AIDS virus infection in Nairobi populations. *J Infect Dis* (in press).
27. Mann JM, Quinn TC, Francis H, et al: Prevalence of HTLV-III/LAV in household contacts of patients with confirmed AIDS and controls in Kinshasa, Zaire. *JAMA* 1986; 256:721-724.
28. Jonckheer T, Dab I, Van de Perre P, et al: Cluster of HTLV-III/LAV infection in an African family. *Lancet* 1985; 1:400-401.
29. Piot P, Mann JM: Bidirectional heterosexual transmission of human immunodeficiency virus (HIV). Presented at the International Conference on AIDS, Paris, France, June 23-25, 1986.

AIDS in adolescents: A rationale for concern*

KAREN HEIN, MD

Adolescents between the ages of 13 and 19 years comprise approximately 10% of the American population, accounting for approximately 25 million individuals.[1] Adolescents are not currently identified as a high-risk group for the acquisition or spread of the human immunodeficiency virus (HIV) infection, although the definition of risk has been changing to encompass the notion of risk behavior rather than risk groups. Based on current knowledge of adolescent behavior and development, this age group has been targeted as a "bridging" group to those currently infected.

Although only a few hundred adolescents were diagnosed as having acquired immunodeficiency syndrome (AIDS) in 1986 (Centers for Disease Control, AIDS Surveillance Unit, Atlanta, Ga, personal communication, December 1986), many aspects of the life styles of American adolescents, especially those in the inner cities, seem to place them directly in the path of this epidemic. The youngest members of each of the groups known to be at highest risk for contracting and transmitting AIDS are themselves adolescents (ie, young bisexual and homosexual men, and intravenous drug abusers). But, more critically, it is the chain of heterosexual partners of both of these groups who may form a bridge to a much larger adolescent population whose patterns of sexual behavior may expose them to the virus. As we come to understand more about the heterosexual transmission of AIDS, it appears that adolescents coming into sexual contact with those already infected may face a substantial risk.

CASE HISTORIES

The following two case histories of 17-year-olds illustrate some of the reasons why adolescents should be a focus of concern.

An adolescent female presented to a comprehensive adolescent health clinic after being rejected by the military. An HIV antibody screening test had been performed as part of a recruitment requirement and was found to be positive. The young woman had previously considered herself to be in good health. In the course of a general health assessment she was found to be in the first trimester of an unintended pregnancy. She elected to terminate the pregnancy, but within six months she became pregnant for a second time. She stated that in the past she had had intercourse with another adolescent whom she believes was an intravenous drug abuser.

A 17-year-old male presented to a general adolescent medicine clinic for health screening that was required for enrollment in a job training program. He complained of back pain and fatigue. He admitted to intravenous substance abuse ending five months prior to evaluation. On evaluation, several problems were identified, including glomerulonephritis, evidence of previous hepatitis B infection, and a positive tuberculin skin test. After eliciting the history of previous intravenous drug abuse, HIV status was assessed. Screening and confirmatory tests were positive for HIV infection. During counseling regarding HIV status, the patient revealed that he had had at least two sexual partners within the previous three months with whom he continued to have intercourse after he became aware of his HIV status. He informed only one of these partners and did not use condoms or other forms of contraception.

These case histories illustrate the basis for our current concern about adolescents. These two youngsters did not seek help because they suspected HIV infection or because they recognized themselves as being members of a known high-risk group. They were identified only because of their normal adolescent activities. In the first case, detection was made inadvertently as part of a career decision to join the military. In the second case, detection resulted from a comprehensive evaluation following a request for a routine physical examination that was required for entry into a job-related training program. Both adolescents sought medical care through the usual channels open to them, namely health services specially geared to their age group. Neither might have been appropriately cared for in health facilities for children or adults. The health services, type of counseling, and support that adolescents receive must be appropriate for their age group.

RATIONALE FOR CONCERN

Most adolescents do not seek health services on a regular basis. Thus risk status for HIV infection must be inferred from data collected for other purposes. This information can be summarized in three categories: sexual behavior and relevant consequences, physiologic considerations, and drug abuse.

Address correspondence to Dr Hein, Associate Professor of Pediatrics, Department of Pediatrics, Albert Einstein College of Medicine, Jacobi BE-21, Pelham Pkwy and Eastchester Rd, Bronx, NY 10461.

Partial funding for this research was provided by The Carnegie Corporation of New York.

* This article is based on a presentation at the "Symposium on the Heterosexual Transmission of AIDS," sponsored by the Department of Epidemiology and Social Medicine, Montefiore Medical Center and Albert Einstein College of Medicine, and held October 21, 1986, at the New York Academy of Medicine.

Sexual Behavior and Relevant Consequences

Adolescent Sexual Experience. In the United States, roughly half of all teenagers have had intercourse by the age of 19.[2] However, among the 25 million adolescents in the US, there are subgroups who either have intercourse at an early age or whose patterns of sexual behavior may put them at increased risk. Studies conducted by Zelnick and Kantner[3] in the 1970s (Fig 1) revealed changes during that decade that have profound implications for the possible spread of HIV if it were to be introduced into the adolescent population. In comparing data from 1971, 1976, and 1979, some important changes are evident. The latest survey[3] found that approximately 60% of white urban females and approximately 80% of black urban females have had intercourse by age 19. The greatest increase in the three survey years was among the adolescent white female population.

To complement the national data, regional data support the assertion that some adolescents are currently at greater risk than others for the acquisition and spread of AIDS (Fig 2). Three curves are presented that compare the age at first intercourse of young inner-city women.[4] The curve on the left represents data for young women housed in a detention center. The curve in the center is based on data for young women living in group homes in the Bronx. The curve on the right was derived from a national survey conducted by Sorenson[5] in the 1970s. The average age of first intercourse in the detention population was 12 as compared to an average age of first intercourse for the nation at that time of age 16. The age of sexual initiation for the group home residents was intermediate between that of the other two groups. Some of these young women were runaways. Most were having intercourse with multiple partners with whom they had no lasting relationships and little knowledge of their partners' prior sexual or drug-related behavior. These data begin to define a population of urban adolescents who may be at immediate and particular risk of HIV infection.

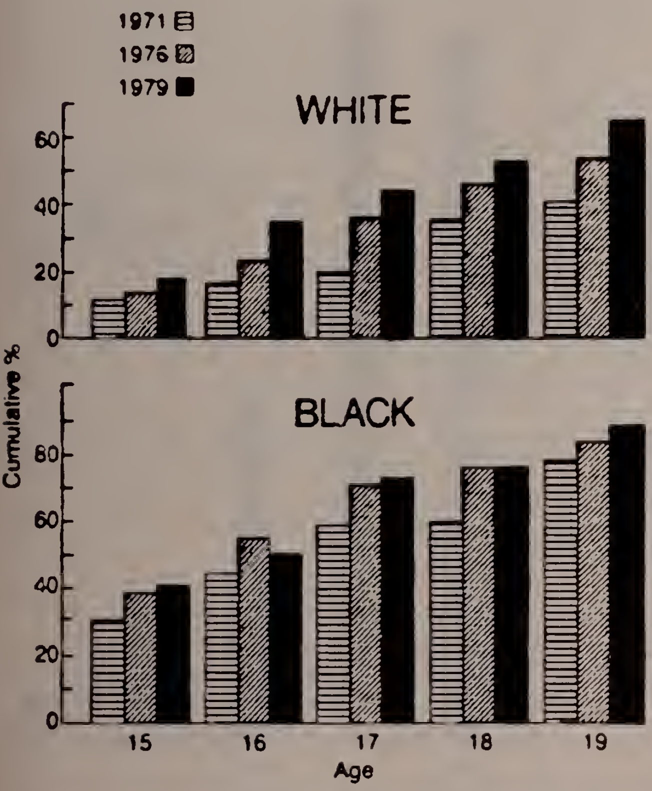


FIGURE 1. Proportion of black and white urban US females aged 15 to 19 years who were sexually experienced in 1971, 1976, and 1979 (from Bell and Hein,[9] adapted from Zelnick and Kantner[3]) (courtesy of Alan Guttmacher Institute).

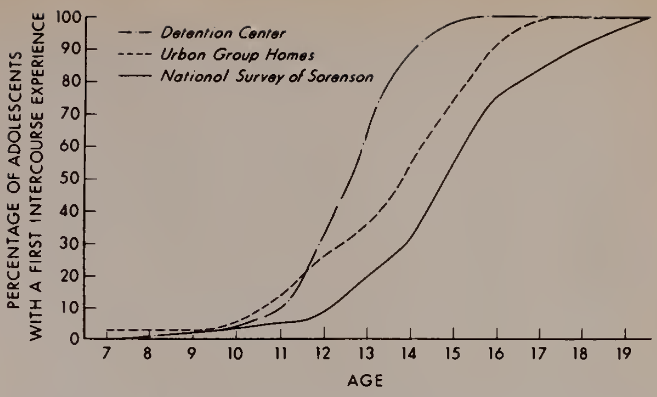


FIGURE 2. Percentage of sexually experienced adolescents aged seven to 19 years for three populations of teenaged females (from Hein, et al[4]) (courtesy of C.V. Mosby Company).

Knowledge of some of the patterns of sexual behavior among adolescents who have had intercourse may help identify teenagers who are at greater risk. Following the scheme of Sorenson,[5] two such patterns of sexual relationships are the "serial monogamous" pattern and that of the "sexual adventurer." According to Sorenson's[5] survey of American adolescents in the 1970s, roughly half of the nonvirginal females and only one quarter of the nonvirginal males could be described as being serially monogamous. Some serial monogamists also occasionally had intercourse with other partners besides the primary consort at that time. More than half of all sexually experienced adolescent females had intercourse with male partners who were 20 years of age or older.

The "sexual adventurer" was defined by Sorenson[5] as an adolescent who by the age of 19 had had a total of 17 sexual partners, and who, in the month preceding the interview, had had an average of 3.2 sexual partners. The sexual adventurer, therefore, becomes another subgroup of adolescents at risk for the acquisition or spread of AIDS to his/her partners, as compared to the serially monogamous adolescent. The sexual adventurer pattern describes approximately 41% of the sexually experienced adolescent males and about 13% of the sexually experienced females in the country.[5]

Contraception. Approximately half of sexually experienced adolescents use any form of contraception at first intercourse, whatever the age at initiation.[2] Oral contraceptives are used by approximately one third of teenaged females, and condoms, depending on the study and the population, are used by approximately 12%. One third to one fourth of adolescents have never used any form of contraception at all.[2]

Pregnancy. Pregnancy, another consequence of the early initiation of sexual activity among American teenagers, occurs in approximately one million teenagers every year, and roughly 85% of these pregnancies are unintended.[2] Since adolescent females tend to have intercourse with males who are two to three years older than they, these younger females may be exposed to HIV infection and thus unintentionally expose their offspring.[5] Pregnant young teenage girls are less likely to obtain prenatal care as compared to adults; thus they and their babies currently have little opportunity to benefit from counseling or services that might inform them about possible risk to themselves or their offspring.

Issues relevant to pregnant teenagers living in New York were recently reviewed. In New York State every day roughly 180 teenagers become pregnant.[6] Minority group status appears to be associated with increased risks to both mother and child.[7] In New York City and some other urban centers, HIV infection has been disproportionately identified in black and Hispanic groups. Cumulative incidence rates of AIDS among blacks and Hispanics are more than three times the rate among whites.[8]

Venereal Disease. National data regarding the incidence and prevalence of various venereal diseases can be very misleading when applied to the adolescent age range. If data are reported per 100,000 of population at a given age, it would appear that rates for the youngest adolescents (10–14-year-olds) are the lowest as compared to other age groups through young adulthood.[9] However, this is an artifact produced by using a denominator that is not relevant to the question.

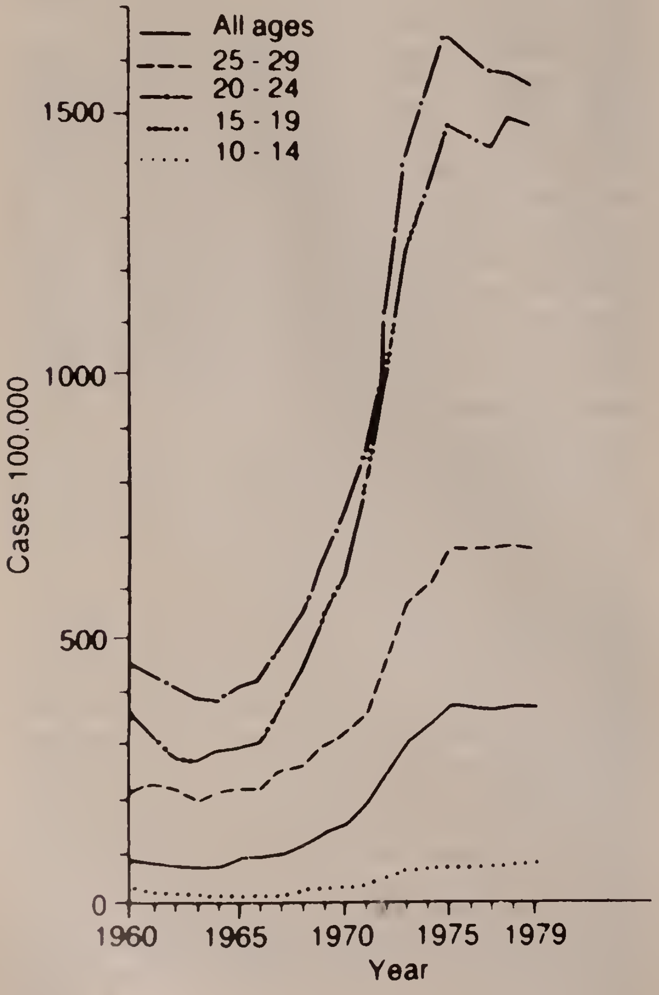


FIGURE 3. Apparent increase in the incidence of gonorrhea among females in the age groups 10 to 14, 15 to 19, 20 to 24, and 25 to 29, and in all females (from Bell and Hein[9]) (courtesy of McGraw-Hill Book Company).

In Figures 3–5, data are presented which give a more accurate portrayal of the rates of venereal disease in American youth.[9] Instead of reporting the number of cases per 100,000 of population (Fig 3), national data have been recalculated and graphed using the percent of sexually active adolescents and young adults in each age group as the denominator (Figs 4, 5). For 1971 and 1976, the 10–14-year-olds and 15–19-year-olds showed the highest rates of gonorrhea (Fig 4), not the lowest, accounting for roughly 3,500 cases per 100,000 sexually active teenagers. For syphilis, the rate was 100–200/100,000 for the youngest groups (Fig 5). Rates of hospitalization for pelvic inflammatory disease were similarly highest in the youngest age groups when the denominator was corrected for the percent of sexually active females.

Thus, the youngsters who are having intercourse at a very early age also have higher rates of venereal disease than their young adult counterparts. Similar analyses of data regarding syphilis and chlamydia reveal that the highest rates occur among the young adolescents, despite the impression that much of venereal disease resides within the young adult population.

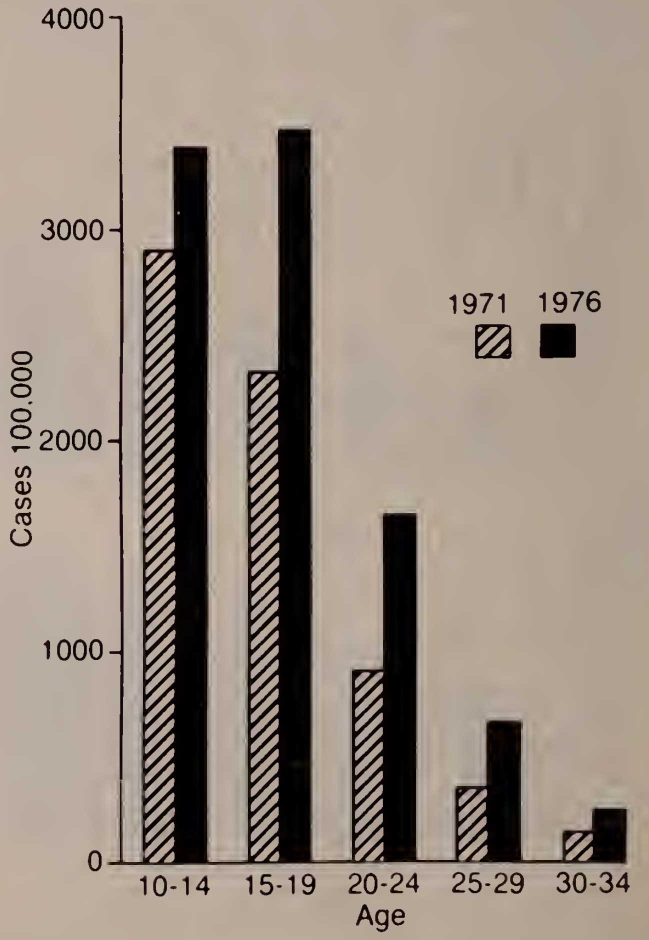


FIGURE 4. Rates of gonorrhea expressed as number of cases per 100,000 of sexually experienced females aged 10 to 14, 15 to 19, 20 to 24, 25 to 29, and 30 to 34[9] (courtesy of McGraw-Hill Book Company).

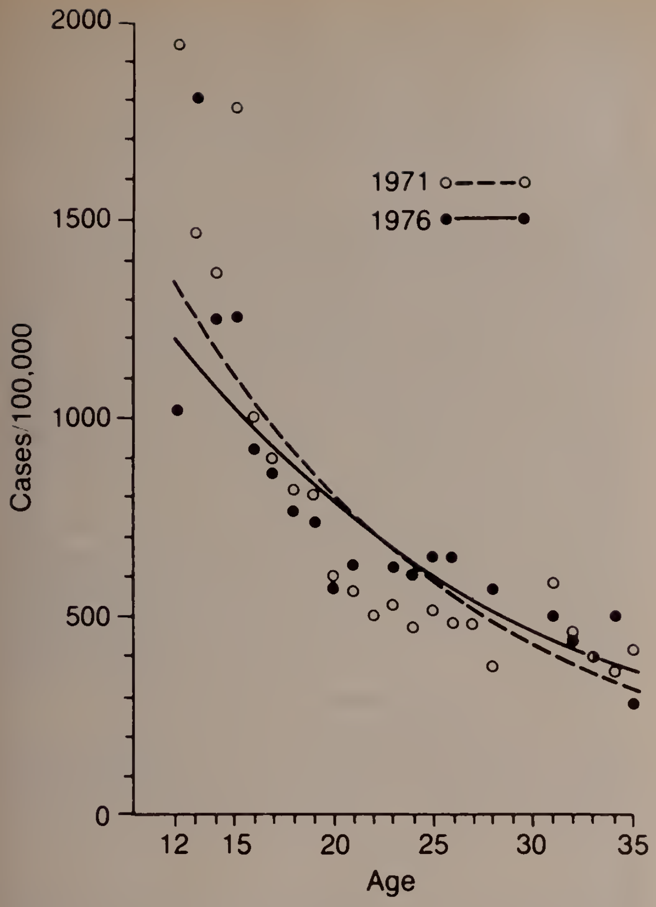


FIGURE 5. Incidence of pelvic inflammatory disease in hospitalized adolescents[9] (courtesy of McGraw-Hill Book Company).

Asymptomatic venereal disease continues to be a problem in some segments of the adolescent population. In the 1970s in the Bronx, youngsters arrested as "persons in need of supervision" or for committing acts of delinquency were screened for the presence of gonorrhea.[10] In a study in which 600 females under the age of 17 years were tested for the presence of cervical infection with gonorrhea, after eliminating all who were symptomatic for any genitourinary symptoms (including any history or any evidence of vaginal discharge or the presence of white blood cells in urine), asymptomatic gonorrheal cervical infection was detected in 7% of these young women.[10]

A similar study conducted during the 1970s involving more than 2,000 males, all under the age of 17, documented a reservoir of 1.9% who had positive urethral cultures but no symptoms of gonorrhea.[10]

Since these young people harbor venereal diseases that were prevalent prior to the introduction of HIV, such as gonorrhea, syphilis, and/or chlamydia, we might therefore expect that these youngsters may be a new reservoir of undetected AIDS virus.

PHYSIOLOGIC REASONS WHY ADOLESCENTS MAY BE AT SPECIAL RISK

There are anatomic and physiologic differences between adolescents and young adults that indicate that teenage females may be at even greater risk than young adult females for the complications of some venereal diseases, and, by analogy, possibly AIDS.

Differences in ovarian function and differences in the cervical milieu in young adolescents may facilitate entry and spread of certain organisms.[9] There are differences in properties of cervical mucous that affect the penetrability of sperm and also the ability of the motile organisms to penetrate through the cervix. As a female progresses through puberty there are changes in vaginal pH. The adult pH is more protective against the invasion of some organisms associated with venereal diseases.

The location of the squamo-columnar junction changes with age. The junction at which squamous epithelial cells that line the vagina and external portion of the cervix that protrudes into the vagina (portio vaginalis) overlaps with endometrial cells differs in the adolescent as compared to the adult woman. The location of this "transition zone" in adolescents may expose young women to organisms or other factors that may predispose them to a greater risk of developing cervical intraepithelial neoplasia.[9]

There is a high prevalence of anovulatory cycles within two years after menarche. It has been observed that the use of oral contraceptives may confer some protective effects against some venereal diseases.[9] The hypothesis is that the presence of progesterone (or the progestins present in oral contraceptives) alters characteristics of cervical mucous and the length of menses. These two factors plus other physiologic effects help prevent or alter the establishment and spread of some venereal diseases. By analogy, the adolescent who is not ovulating is also not producing progesterone. Therefore, the young adolescent female would not benefit from whatever protective effects may come from the presence of this hormone. These factors may contribute to the documented high rates of venereal disease in this population. Whether or not these factors will also be relevant to HIV infection is not known.

The association between cervical infection with human papilloma viruses and cervical cancer has been documented in recent years.[11] Factors that have been identified as being useful in predicting increased risk of developing cervical carcinoma include early age at first intercourse and a history of multiple sexual partners. Once again the adolescent female may be at particular risk. The rates of abnormal cervical cytology seen in a study of young women under the age of 17 years housed at the Spofford Juvenile Detention Center were very high.[12] Pap smear screening revealed rates of dysplasia and early forms of cervical intraepithelial neoplasia (CIN) of 35 per 1,000. This rate is comparable to that of the highest risk groups in the adult population. Whether or not young adolescent females with early age at first intercourse are also at increased risk for HIV infection and consequent alterations in immune status is unknown at this time.

DRUG ABUSE

Past experience in the 1960s and 1970s regarding drug abuse patterns may have relevance for the current AIDS epidemic. We have much to learn from the past regarding adolescents. Figure 6 shows data collected at the Spofford Juvenile Detention Center from the late 1960s through 1977 (just prior to the documentation of AIDS).[13] Usage patterns for various drugs are shown for the adolescent

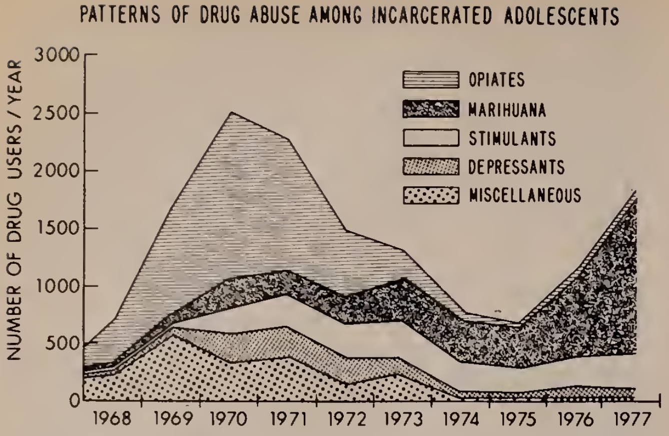


FIGURE 6. Analysis of types of drugs used by 14,810 of 66,000 adolescents admitted to a youth detention center between 1968 and 1977[13] (courtesy of the American Medical Association).

population arrested for status offenses or acts of delinquency. All youngsters were 17 years of age or younger. During the late 1960s and early 1970s, considerable publicity was given to the young adult drug abuser. Heroin was not thought to be a problem among adolescents. The medical consequences of heroin use in adolescents were not documented in the medical literature until the epidemic was long established in the adult population. Medical correlates of heroin use were noted and published in 1979.[13,14] Medical sequelae that followed increased use of heroin were then reported; these included increased admissions to in-patient units and increased cases of hepatitis and drug-related deaths. The pattern of these sequelae followed the heroin usage pattern, as shown in Figures 7 and 8.

The problem in the teenage population was not appreciated because adolescents are traditionally grouped statistically with children or adults, not studied as a unique population. Services were organized for young adults long before an appropriate response was mounted by public health and educational agencies serving youth. We should learn from previous errors. In this case, we have an opportunity to intervene before large numbers of adolescents are infected and before the obvious medical sequelae of HIV infection have become apparent.

The opportunity now presents itself to focus attention on the adolescent population as distinct from young adults or children. They are not big children and they are not small adults. They have a unique set of sociologic, epidemiologic, and physiologic circumstances that require special attention now, before wider spread of HIV infection is documented in this segment of the population.

In the Bronx, drug addiction is currently the major risk factor for AIDS. Two thirds of all AIDS cases are associated with intravenous (IV) drug abuse and about 50% of

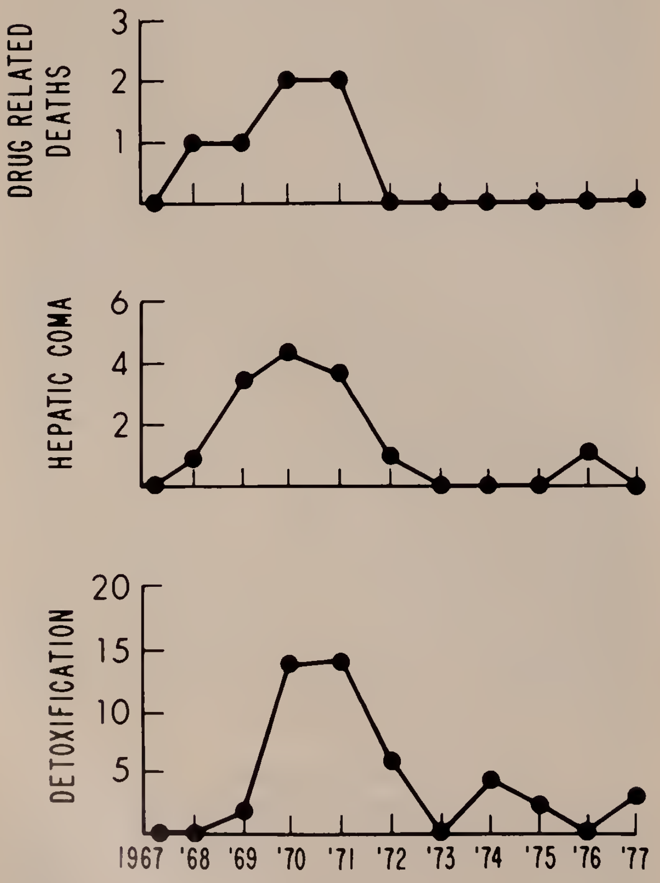


FIGURE 7. Ten-year experience from an in-patient unit for adolescents describing the number of teenagers admitted who died of drug-related causes or hepatic coma or who were admitted for drug detoxification[13] (courtesy of the American Medical Association).

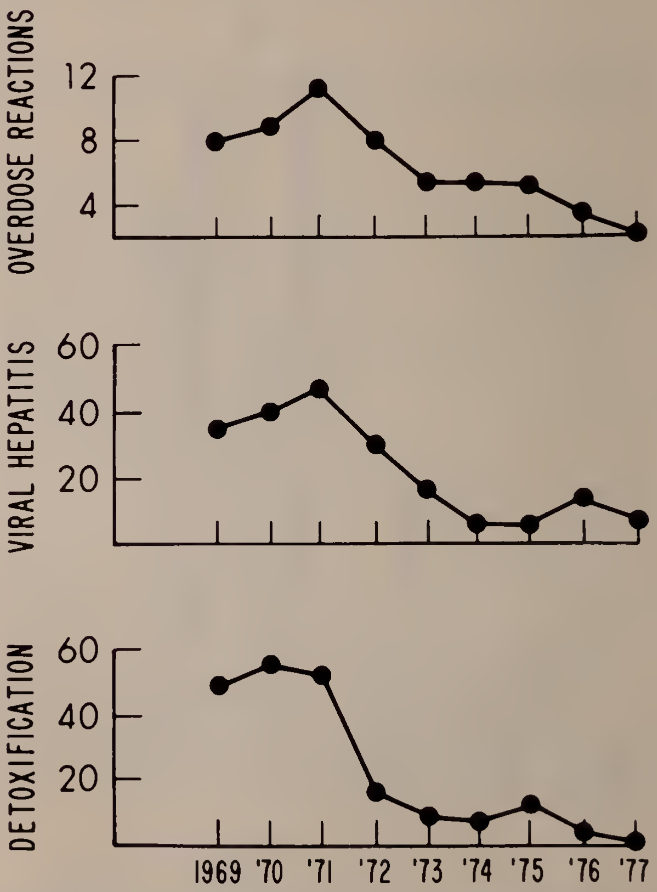


FIGURE 8. Nine-year experience with juvenile detention facility describing number of cases of overdose reactions, hepatitis, and heroin detoxification[13] (courtesy of the American Medical Association).

the borough's 60,000–70,000 IV drug users are believed to be positive for HIV antibodies.[15] This group is mostly composed of young adults (aged 20–40 years) who are black and Hispanic and who are sexually active with multiple partners, making large scale heterosexual transmission a genuine possibility.[16]

One indication of the emerging problem of HIV infection in a young heterosexually active population can be found in the results of the blood testing program for new military recruits. While the national rate of HIV infection is still low (1.6/1,000), the figure for the Bronx (and for Manhattan; Newark; Washington, DC; and Puerto Rico) appears to be about ten times this rate, or 1.6%.[17,18] The ratio of males to females in this sample is 3:1 as compared to 13:1 nationally for AIDS cases. This pattern is more characteristic of that observed among IV drug abusers and may also implicate heterosexual transmission. Further, these rates must represent an underestimation since the military does not attract those adolescents who are the most marginal, and who would be expected to have the highest rates of sexually transmitted diseases, drug abuse, promiscuity, and pregnancy.

IMPLICATIONS FOR EDUCATIONAL INTERVENTIONS

In the absence of an effective treatment or vaccine against HIV infection, risk reduction programs must be developed to restrict the introduction and spread of AIDS to the adolescent population.[19] Sampling adolescents themselves to learn about their specific concerns, attitudes, and beliefs regarding AIDS should be the basis for designing interventions for teenagers. Educational materials prepared for teenagers should be based on their own questions, perceptions, fears, and convictions.

It appears that there are two groups within the adolescent population, though we do not currently know the dimensions of each group. They include those young people who may be currently infected or at risk, and those whom we might call the "worried well" (adolescents who are very concerned or anxious, but who do not have reason to be concerned at present). Neither group has adequate age-appropriate services or support. We should begin to separate the "worried well" from the smaller group of teenagers for whom concern is warranted. A major goal of risk reduction programs for the latter must be to try to alter current patterns of sexual behavior, including attitudes toward use of contraceptives.

CONCLUSION

Because of contemporary American adolescent life styles, teenagers are an important group for targeted interventions to prevent further spread of HIV infection. The factors that support our current concern are prevailing patterns of adolescent sexual activity, the age of initiation of first intercourse, current contraceptive practices, the choice of sexual partners, rates of venereal diseases, and anatomic and physiologic considerations.

It is hypothesized that the "sexual adventurers," in particular, form a subpopulation of adolescents who are most likely to be the entree of HIV infection into the adolescent population, just as this group has been a reservoir for other sexually transmitted diseases. While more effective treatments and possibly primary preventive methods are being developed for all age groups, special attention should be paid to adolescents. They are a proximal group that may become rapidly infected in the near future. Therefore, they require the immediate attention of health policy and health care agencies.

Acknowledgments. I wish to acknowledge a group of individuals with whom I have been working over the past year. Together we have considered the implications of AIDS in adolescents and are jointly planning new programs to address the issues raised in this paper. Key members include Ernest Drucker, PhD, Nancy Reuben, MD, Sten Vermund, MD, and Sandra Shephard, MD. Special acknowledgment is made to Sharon Lockett and Carol Ruffo for their assistance in preparing this manuscript.

REFERENCES

1. Projections of the population of the United States. *Current Population Reports*. Series 25, No. 952. Bureau of the Census, 1986.
2. *Teenage Pregnancy: The Problem That Hasn't Gone Away*. New York, The Alan Guttmacher Institute, 1981.
3. Zelnick M, Kantner JF: Sexual activity, contraceptive use and pregnancy among metropolitan-area teenagers: 1971–1979. *Fam Plann Perspect* 1980, 12:230–231, 233–237.
4. Hein K, Cohen MI, Marks A, et al: Age at first intercourse among homeless adolescent females. *J Pediatr* 1978; 93:147–148.
5. Sorensen RE: *Adolescent Sexuality in Contemporary America*. New York, World Publishing Co, 1973, pp 180–280.
6. Randolph LA, Gesche M: Black adolescent pregnancy: Prevention and management. *J Community Health* 1986; 11:10–18.
7. Clarke MI: Black teenage pregnancy: An obstetrician's viewpoint. *J Community Health* 1986; 11:23–30.
8. AIDS among Blacks and Hispanics—United States. *MMWR* 1986; 35:655–666.
9. Bell T, Hein K: The adolescent and sexually transmitted disease, in Holmes K, et al (eds): *Sexually Transmitted Diseases*. New York, McGraw-Hill, 1984, pp 73–84.
10. Hein K, Marks A, Cohen MI: Asymptomatic gonorrhea: Prevalence in a population of urban adolescents. *J Pediatr* 1977; 90:634–635.
11. Howley P: On human papillomaviruses. *N Engl J Med* 1986; 315:1089–1090.
12. Hein K, Schreiber K, Cohen MI, et al: Cervical cytology. The need for routine screening in the sexually active adolescent. *J Pediatr* 1977; 91:123–126.
13. Hein K, Cohen MI, Litt IF: Illicit drug use among urban adolescents. A decade of retrospect. *Am J Dis Child* 1979; 133:38–40.
14. Hein K, Cohen M, Litt I, et al: Juvenile detention. Another boundary issue for physicians. *Pediatrics* 1980; 66:239–245.
15. Drucker E: AIDS and addiction in New York City. *Am J Drug Alcohol Abuse* 1986; 12:165–181.
16. Schoenbaum EE, Selwyn PA, Klein RS, et al: Prevalence of and risk factors associated with HTLV-III/LAV antibodies among intravenous drug abusers in a methadone program in New York City. Presented at the International Conference on AIDS, Paris, France, June 23–25, 1986.
17. *Human T-lymphotropic Virus Type III/Lymphadenopathy-Associated Virus Antibody Prevalence in U.S. Military Recruit Applicants*. US Dept of Health and Human Services. Atlanta, Centers for Disease Control, 1986.
18. Burke D, Brundage JF, Bernier W, et al: Infections among prospective U.S. military recruits. Presented at the International Conference on AIDS, Paris, France (Poster 676), June 23–25, 1986.
19. Koop E: *Surgeon General's Report on Acquired Immune Deficiency Syndrome*. US Dept of Health and Human Services, Public Health Service, October 1986.

Education and contact notification for AIDS prevention*

DEAN F. ECHENBERG, MD, PHD

Epidemics are dynamic phenomena. The strategies used to deal with epidemics generally evolve as knowledge increases. These strategies must be evaluated continually to insure success. This paper discusses how disease control strategies evolved in San Francisco as the prevalence of acquired immunodeficiency syndrome (AIDS) in different groups changed.

The goal of an AIDS prevention program is to prevent transmission of the human immunodeficiency virus (HIV). A broad range of intervention programs is required to attain this goal. These programs must be appropriate to the particular circumstances of each community where they are to be implemented, and, even though no one approach will be successful everywhere, education must be the cornerstone of every effort. This education must focus on the appropriate groups of individuals—that is, those who are either infected or at risk of becoming infected.

PREVALENCE OF DISEASE

One factor that determines how this education will be applied in any population group is the prevalence of the disease in that population. When many people are infected, mass education directed to the entire population is one of the most important methods. When very few people are infected, then attempts must be made to locate these individuals and individually educate them, because of the potential health consequences of a sexually active and uninformed infected individual.

If one looks at the epidemiology of AIDS in San Francisco one can understand how the city's different strategies evolved. A cohort of homosexual men in San Francisco, recruited from the Department of Public Health's sexually transmitted disease clinics, has been followed since 1978.[1,2] Originally, these men volunteered for hepatitis B studies. In 1984 it was realized that many of these men were infected with the AIDS virus. Consequently, they were asked to participate in AIDS studies, and blood specimens which had been drawn from them between 1979 and 1980 and saved were examined.

One of the saddest statistics in the AIDS tragedy is that by the time the first publication on AIDS appeared in the medical literature in July 1981, 30–40% of the men in this cohort were already infected.[3] It was another six months to a year before the mode of transmission was understood, at which time 40–50% of the cohort was infected.

The strategies that evolved at that time to deal with this situation in San Francisco are obviously different from those that are applied where the prevalence of the disease is much lower. In addition, today there is a blood test for potential infectivity which was not available then.

In the initial stages of the epidemic, a strategy was evolved that said in effect that all people in the classic high-risk group should consider themselves to be infectious through sexual contact. This included not only gay men but also individuals with hemophilia and intravenous (IV) drug users. This strategy of mass education has been relatively effective. It has had a great impact in decreasing the level of unsafe sexual activities that facilitate transmission of the virus.[4] In 1982, an average of 382 cases of rectal gonorrhea were seen each month in San Francisco at a sexually transmitted disease clinic. This number has now decreased to fewer than 30 per month.[5] However, it subsequently became apparent that cases could occur outside of the classic high-risk groups. This then called for the use of additional strategies.

Fifty percent of the gay men in San Francisco are infected with the AIDS virus.[6] In the general heterosexual population, the prevalence of HIV infection is less than a fraction of one percent. In this latter group, the progress of the epidemic might be even more insidious because heterosexual individuals often may not know they are infected. The incubation period for AIDS can be up to seven years and possibly longer.[2] An infected individual might be able to carry the virus and unknowingly infect others during this time. Even with mass education campaigns applied to the general population, one cannot assume an overall decrease in unsafe sexual activity. The immediacy of the epidemic is very different among heterosexuals than it has been among homosexual men.

The approach in San Francisco has been to find individuals in the heterosexual community who have been exposed and do not know it and to offer them risk-reduction education and serological testing.[7] If this is not done one will see rivulets of AIDS extending unchecked into the general heterosexual population, resulting in a much larger problem in the future.

From the San Francisco Department of Public Health.

Address correspondence to Dr Echenberg, Director, Bureau of Communicable Disease Control, San Francisco Department of Public Health, 101 Grove St, Room 402, San Francisco, CA 94102.

* This article is based on a presentation at the "Symposium on the Heterosexual Transmission of AIDS," sponsored by the Department of Epidemiology and Social Medicine, Montefiore Medical Center and Albert Einstein College of Medicine, and held October 21, 1986, at the New York Academy of Medicine.

INTERVENTION STRATEGY

Two strategies have been developed in San Francisco to deal with both high- and low-prevalence groups. In both groups a mass education campaign is being used and in the latter, a contact notification program has been started in addition. An active surveillance program seeks out all AIDS cases in San Francisco and each case is investigated. Anyone who has been diagnosed as having AIDS is asked for the names of their heterosexual partners so that they can be contacted and the situation explained to them. Once the program is explained (we use very sensitive investigators who have had long experience in dealing with sexually transmitted diseases in the community in San Francisco), a very high degree of cooperation is obtained. The identity of the index case is never revealed and the program is completely voluntary. It is permissible for individuals to tell their partners themselves. If in some cases they would rather not participate, that is their prerogative, although that has been rare.

The same techniques have been used in San Francisco during the past few years for over 6,000 cases of syphilis and gonorrhea. Although there is no treatment for AIDS as there is for syphilis and gonorrhea, an intervention is still available. It is possible to tell people who would not otherwise know it that they might be infected. A major assumption of the program is that an individual would not want to infect others unknowingly. The program has located quite a number of women who were infectious and did not know it. In fact most of the individuals named as heterosexual partners are women of childbearing age, so a contact tracing program is especially important in preventing perinatal transmission of HIV.

RESULTS

Twenty-seven heterosexual contacts have been investigated since the beginning of the program. Seven of them were HIV antibody positive. Almost all of them were women of childbearing age. It is interesting that about half of the women had concerns about being infected even before they were contacted by our investigators. They thought they were at risk because of their sexual activities and were greatly relieved when the blood tests proved to be negative. Those who were positive did not know they were infected.

It is extremely important for anyone who is contemplating these kinds of programs to understand that confidentiality is essential. There must be very strong legal protections to safeguard this information from all subpoenas. In addition, there is a need for a precise technical approach based on current knowledge of HIV transmission, as well as a need for very sensitive and informed investigators.

In addition to contact tracing of the heterosexual partners of AIDS patients, we have begun a similar program of contact tracing among individuals who have become infected after having received contaminated blood, and among mothers of pediatric AIDS cases. Partner referral is also encouraged among all HIV-infected cases.

CONCLUSIONS

Each geographic region will have to examine its own AIDS epidemiology. In some situations where the prevalence is extremely high, contact tracing is inefficient and inappropriate. On the other hand, where the number of cases is still small, where there are confidentiality safeguards, and where there are sensitive case counselors, contact tracing and partner referral can play an important role in dealing with the AIDS epidemic by identifying individuals who are unknowingly infected with the AIDS virus.

This technique will be especially useful in dealing with what may be the next phase of the AIDS epidemic, the spread via heterosexual contact into those groups where one now sees the highest rates of syphilis and gonorrhea. These are the same groups among which there is a high level of intravenous drug use. They include adolescents and young adults, especially in minority groups in low-income socioeconomic areas.

In addition, it will become extremely important that all health care providers understand the consequences of their being the first to diagnose HIV infection in a particular individual from a low-prevalence group. These individuals must be advised to refer other sexual partners who might have become unknowingly infected. If this is not done the result will be an unending chain of infections extending further into the community.

REFERENCES

1. Jaffe HW, Feorino PM, Darrow WW, et al: Persistent infection with human T-lymphotropic virus type III/lymphadenopathy-associated virus in apparently healthy homosexual men. *Ann Intern Med* 1985; 102:627–628.
2. Jaffee HW, Darrow WW, Echenberg DF, et al: The acquired immunodeficiency syndrome in a cohort of homosexual men. A six-year follow-up study. *Ann Intern Med* 1985; 103:210–214.
3. Echenberg DF, Rutherford GW, O'Malley P, et al: Update—Acquired immunodeficiency syndrome in the San Francisco cohort study 1978–1985. *MMWR* 1985; 34:573–575.
4. McKusick L, Wiley JA, Coates TJ, et al: Reported changes in the sexual behavior of men at risk for AIDS, San Francisco 1982–1984—the AIDS behavioral research project. *Public Health Rep* 1985; 100:622–629.
5. Rectal gonorrhea in San Francisco, October 1984–September 1986. *San Francisco Epidemiologic Bulletin*, vol 2, no. 12, December 1986.
6. Winkelstein W Jr, Lyman DM, Padian N, et al: Sexual practices and risk of infection by the human immunodeficiency virus. The San Francisco Men's Health Study. *JAMA* 1987; 257:321–325.
7. Echenberg DF: A new strategy to prevent the spread of AIDS among heterosexuals [editorial]. *JAMA* 1985; 254:2129–2130.

SPECIAL ARTICLE

AIDS care in New York City: The comprehensive care alternative

Jack A. DeHovitz, MD, MPH, Virginia Pellegrino, MSc, MPH

In this issue of the *Journal*, Arno and Hughes[1] critically examine the differences in the policy responses of San Francisco and New York City to the epidemic of acquired immunodeficiency syndrome (AIDS). They note that while both cities have devoted an enormous amount of financial support to fighting the epidemic, the timing of the development of specific services varied. For example, the first outpatient program for AIDS patients in San Francisco was opened in September 1981, with the first inpatient unit being established there in July 1983. In contrast, a dedicated AIDS outpatient clinic did not begin in New York until 1984, with the first discrete inpatient unit following in November 1985.[1] Although the San Francisco experience provides an important foundation on which to build, the unique risk-factor and case mix of New York's population of AIDS patients requires the development of innovative programs to deal with these diverse populations.

New York State has embarked on a program to designate comprehensive AIDS centers at various hospitals both in New York City and upstate. The first center was designated at St Clare's Hospital and Health Center in May 1986. From our experience in developing this program, we have developed a framework that addresses the needs of patients with AIDS and other manifestations of HIV infection.

The initial impetus for the development of comprehensive AIDS programs stemmed from the unique medical problems of AIDS patients. While some of the problems resemble those of patients with other catastrophic diseases such as cancer, there are some differences that require unique medical interventions. Perhaps the most obvious is the fact that the clinical spectrum of AIDS and severe human immunodeficiency virus (HIV) infection is extremely variable. Although some patients with Kaposi's sarcoma (KS) have an excellent prognosis for survival for three to five years, many patients present with an opportunistic infection which leads to rapid deterioration and death. Other patients present with severe dementia without any of the opportunistic infections or cancer that fulfill the current diagnostic criteria for AIDS. In addition to the various acuity levels, the conditions of those who have symptomatic HIV infection are subject to rapid fluctuation. These individuals may be fully ambulatory and feeling relatively well one day, and be acutely ill the next.

Although the opportunistic infections that appear with this disease are not new, the conventional therapies often cause unexpectedly frequent or severe toxicities. Treatment of the various malignancies that accompany HIV-induced immunosuppression is also difficult. Often the use of conventional chemotherapeutic regimens results in severe myelosuppression, or increased risk of opportunistic infection. Finally, the introduction of new antiviral regimens such as azidothymidine (AZT) may be accompanied by toxicities not revealed in comparatively brief initial clinical trials.

As health care professionals have become more experienced in caring for those with HIV infection, several other unique characteristics have emerged which support a comprehensive approach to care. In addition to their complex medical problems, persons with symptomatic HIV infection or asymptomatic seropositivity face intense psychosocial problems. These range from the isolation and stigmatization they encounter from friends, family, and society, to learning to cope with having a terminal illness at an early age.[2]

A majority of AIDS patients develop neurological and psychiatric complications as a result of HIV infection.[3] In addition to recurrent episodes of acute opportunistic infections, many patients will have some clinical manifestation of neurologic disease which can leave them permanently debilitated and unable to live outside of an institutional setting.

The supervision of the care of AIDS patients requires a dedicated team consisting of, at a minimum, infectious disease specialists, oncologists, pulmonologists, and neurologists. As the population of patients with AIDS invariably expands, more broadly trained practitioners such as internists assisted by physician assistants and/or nurse practitioners can extend the expertise of these subspecialists.[4] The availability of a dedicated medical staff will

---

Dr DeHovitz is Assistant Professor, Department of Preventive Medicine and Community Health, SUNY Health Science Center at Brooklyn, and Assistant Medical Director, Spellman AIDS Program, St Clare's Hospital and Health Center, New York, NY. Ms Pellegrino is Vice-President for Planning at St Clare's Hospital and Health Center.

Address correspondence to Dr DeHovitz, Department of Preventive Medicine and Community Health, Box 43, SUNY Health Science Center at Brooklyn, 450 Clarkson Ave, Brooklyn, NY 11203.

allow rapid intervention in acute medical problems, improved communication, and increased patient compliance. In addition, the presence of dedicated AIDS practitioners will facilitate the process of clinical investigation.

DESIGNING AN INSTITUTIONAL RESPONSE

In view of the unique dimensions of HIV-related disease described above, the acute care hospital is the only resource that can deliver the range of services this population requires. A comprehensive approach to care may prevent the inappropriate utilization of resources by AIDS patients and ensures that all of the needs of patients with AIDS are addressed.

A hospital-based program for persons with AIDS should endeavor to provide a comprehensive range of services that should include at least the following: acute inpatient services, alternate level of care services, outpatient services, home care services, and access to dental services. In addition, the large number of homeless people with AIDS presents a significant problem in New York City. While housing does not fall within the normal range of services provided directly by hospitals, linkages to community-based organizations involved in this service should be established.

Acute Inpatient Care. A number of acute care hospitals have developed discrete inpatient units as the core service of their AIDS program.[5] While controlled studies have not been done, a discrete unit may be more effective in caring for AIDS patients than scattered placement from both a cost and a quality of care standpoint. In a discrete unit the staff members have chosen to care for AIDS patients, which prevents the isolation and resistance to caring for patients that exists in many hospitals. The availability of extensive psychosocial support services and other specific programs for AIDS patients, which can be readily accomplished in a discrete unit, ensures that patients receive the level and range of services they require. For example, the formation of patient support groups is facilitated in the environment of a discrete unit. A discrete unit also helps to reduce the length of acute hospitalization through focused, early interventions by discharge planning and financial resource staff.

The existence of a discrete inpatient unit alone, however, will not solve the problems of lengthy acute hospital stays. The development of alternate levels of care within institutions and community-based home care and housing services are, therefore, key components of the comprehensive care model.

Alternate Levels of Care. A significant number of AIDS patients being treated in acute care hospitals in New York City are there only because there are no alternatives for them in either an institutional or community-based setting. At St Clare's Hospital, for example, as many as 15% of the patients on any given day do not require acute care services. Within this group of hospitalized AIDS patients are a substantial number who suffer from neurologic or psychiatric conditions associated with HIV infection. These conditions, which include neuropathies and severe dementias, frequently require long-term institutional care, but not at an acute care level. The level and type of services that these patients require do not currently exist in the institutional sector and will have to be developed.

The solution to the alternate level of care needs of many of these patients cannot be achieved simply by increasing access to traditional skilled nursing facilities. The services they need may be more comparable to a general acute care service than a long term care service as traditionally defined. Hospitals should consider developing extended care units for persons with neurologic or psychiatric conditions related to HIV infection as part of the development of the comprehensive care model.

Outpatient Services. The development of a multi-faceted outpatient program is an essential component of the comprehensive care model. A full range of services should be available, from HIV screening through diagnosis and treatment.

The outpatient program should be designed with two goals in mind. First, it should result in decreased use of the emergency room by AIDS patients. Second, it should facilitate the referral of patients to the appropriate level of outpatient care. To accomplish these goals, a triage system should be implemented to assess patient needs and rapidly integrate individuals ranging from the worried well to those who are acutely ill. Using a triage system, for example, new patients are evaluated by an adult nurse practitioner and referred to the appropriate service within the outpatient program. The triage system can also facilitate the response to current outpatients who need to be seen by someone immediately rather than waiting for their scheduled appointment.

In the outpatient setting a primary care physician and/or physician extender, ideally one who is also involved in the inpatient unit, should be assigned to each patient.[6] A full range of multispecialty services must also be available, such as neurology, gastroenterology, hematology/oncology, and dermatology.

An important service of the outpatient program is ambulatory infusion therapy. Through this program patients can receive intravenous antibiotics and blood transfusions on an outpatient basis. This program has significant cost-saving potential since the alternative is short-term admission to the hospital. It also provides a psychological benefit for AIDS patients by allowing them to remain at home.

A comprehensive outpatient program for people with HIV infection must also include a mental health component. Persons with HIV infection—ranging from those who are seropositive to those with confirmed AIDS—face multiple psychosocial issues which are appropriately addressed on an outpatient basis. As in the inpatient program, the outpatient service should use various types of support groups as well as individual counseling.

The high number of intravenous (IV) drug abusers among the AIDS patients in New York City necessitates that linkages be established to drug treatment programs. In some instances hospitals should consider developing a methadone maintenance treatment program or other drug treatment program as a component of the outpatient service. The integration of drug treatment services with other services for persons with AIDS is essential to address the increasing problem of IV drug abusers with AIDS being admitted to the hospital for therapy for an opportunistic infection, responding appropriately to therapy, and then returning to actively using drugs after discharge.[7]

Dental Services. The provision of dental services is another important component of the comprehensive care program for persons with HIV infection. Dental professionals should be an integral part of the medical and support team caring for persons with HIV infection.

There are several diseases associated with HIV infection which manifest themselves in the head and neck region. These diseases include discrete oral lesions which can be detected and treated early by trained dental professionals. Also, many patients with AIDS experience excessive weight loss, a condition which is often exacerbated by dental problems that can be effectively treated.

In addition to treating dental problems, a considerable amount of clinical research can be done by the dentists involved in a comprehensive care setting. These dentists can also design and conduct educational programs for the dental community at large regarding the treatment of patients with AIDS.[8]

Home Care. Out-of-hospital management of AIDS patients requires rigorous planning and close integration with inpatient services. In addition to simple assistance with activities of daily living, AIDS patients at home often require sophisticated and specialized nursing intervention. For example, they may require IV hydration because of persistent diarrhea, IV antibiotics because of an opportunistic infection, or even simple intramuscular injection of narcotics for pain control.

Other service components of community-based care, such as home attendant care, residential facility support, and home hospice care, overlap with the needs for specialized nursing intervention. The hospital provides the ideal site for the coordination of such services. In this way management plans can be designed in consultation with both hospital-based and community-based nursing personnel prior to discharge. It will also encourage careful monitoring of the patient after discharge. This coordination between inpatient, outpatient, and community-based staff will prevent frequent and avoidable readmissions as well as provide a means of communication for the various staff members.

The Visiting Nurse Service (VNS) of New York currently provides home care for patients in New York City who are Medicaid eligible and have AIDS. In 1986 they provided care to over 1,000 patients in the five boroughs.[9] Although both the quality and breadth of services offered is exceptional, delays in instituting services, as well as communication problems, have resulted in both increased length of stay as well as avoidable readmissions. To address this problem plans are underway to staff comprehensive AIDS programs with VNS personnel to facilitate the transition from inpatient care to home care.

CONCLUSION

The scope of the AIDS epidemic in New York and the unique characteristics of the disease demand that hospitals, as the focal point of the health care system, develop an immediate response to addressing the needs of persons with HIV infection. The most appropriate and meaningful approach is one which is comprehensive and incorporates diverse levels of services.

The comprehensive care model as described represents an appropriate hospital-based response to this epidemic for the immediate future. However, in the absence of an effective vaccine or antiviral therapy to alter the course of the epidemic, we will be faced with over 10,000 severely ill individuals with HIV infection in New York City by 1991. As an outgrowth of the implementation of the comprehensive care model, presumably at multiple hospitals throughout New York City, it may become necessary, as has been suggested, to designate specialized regional hospital centers for the care of persons with HIV infection.[10] This option, however, requires careful study because of the implications it may have for further isolation of this population.

REFERENCES

1. Arno PS, Hughes RG: Local policy responses to the AIDS epidemic: New York and San Francisco. *NY State J Med* 1987; 87:264–272.
2. Morin SF, Charles KA, Malyon AK: The psychological impact of AIDS on gay men. *Am Psychol* 1984; 39:1288–1293.
3. Navia BA, Jordon BD, Price RW: The AIDS dementia complex: I. Clinical features. *Ann Neurol* 1986; 19:517–524.
4. Institute of Medicine, National Academy of Sciences: *Confronting AIDS: Directions for Public Health, Health Care and Research.* Washington, DC, National Academy Press, 1986, p 140.
5. Volberding PA: The clinical spectrum of the acquired immunodeficiency syndrome: Implications for comprehensive patient care. *Ann Intern Med* 1985; 103:729–733.
6. LaCamera DJ, Masur H, Henderson DK: Symposium on infections in the immunocompromised host. The acquired immunodeficiency syndrome. *Nurs Clin North Am* 1985; 20:241–256.
7. DeHovitz J: Planning for the AIDS epidemic: Public health control measures and the provision of patient care. *J Community Health* 1986; 11:215–218.
8. Andriolo M, Rosenberg J, Wolf J: AIDS and AIDS-related complex: Oral manifestations and treatment. *J Am Dental Assoc* 1986; 113:586–589.
9. *AIDS Program Summary.* New York, Visiting Nurse Service, 1986.
10. Barnes D: AIDS stresses health care in San Francisco. *Science* 1987; 253:964.

Coagulase-negative staphylococcal sepsis in drug-dependent newborns who may be HIV-positive

FRANCISCO MEDINA MEJIA, MD, STEPHEN R. KANDALL, MD

Neonatal sepsis due to coagulase-negative staphlylococci has been reported with increasing frequency in very low birth weight infants or infants subjected to invasive procedures or treatment.[1-3] We report two cases of coagulase-negative staphylococcal sepsis in passively addicted, but otherwise healthy, term infants who were not subjected to invasive procedures. One mother-infant pair was found to be serologically positive for human immunodeficiency virus (HIV); HIV status in the other pair is not known. Vertical perinatal transmission of acquired immunodeficiency syndrome (AIDS) from drug-dependent mothers to their infants[4-6] and newly described immunologic alterations in these infants[4] may render these neonates susceptible to infections with ubiquitous and indolent organisms.

CASE REPORTS

Case 1. A 2.72-kg female infant was born after a term gestation complicated by the mother's intravenous heroin abuse during the first six months of pregnancy. At 24 hours of age, the infant exhibited temperature instability, irritability, and tremors. A diagnostic workup for sepsis was performed, and therapy with ampicillin and gentamycin was started. The infant was also treated with camphorated tincture of opium (paregoric) for suspected opiate withdrawal.

Antibiotic therapy was discontinued on the third day of life, after negative blood, urine, and spinal fluid cultures were reported; the following day, a coagulase-negative staphylococcus susceptible to novobiocin was reported growing from the original blood culture. The isolated organism was sensitive to ampicillin, penicillin, oxacillin, erythromycin, cephalothin, gentamycin, and chloramphenicol. On day six, a second blood culture was drawn which, four days later, grew a coagulase-negative staphylococcus with the same sensitivities as those of the first organism. On day ten, a third blood culture was drawn, and intravenous vancomycin, 45 mg/kg/day, was started; this blood culture also grew a coagulase-negative staphylococcus with the same antibiotic sensitivities. Vancomycin treatment was continued for ten days; blood cultures drawn during the treatment and following cessation of treatment were both negative. Drug withdrawal was managed with gradual reduction of the dosage of paregoric. The infant was discharged on the 28th day of life. No serious infections or hospital visits were noted at a nine-month follow-up evaluation.

Case 2. A 2.73-kg male infant was born after a 38-week gestation to a 32-year-old woman on maintenance methadone therapy who had abused injected cocaine during the pregnancy. On the second day of life, the infant had an elevated temperature, hypertonia, hyperreflexia, and tremulousness. Treatment with camphorated tincture of opium (paregoric) was started; on day 13, phenobarbital was added to control withdrawal. A diagnostic workup for sepsis was also performed at that time, and treatment with oxacillin and gentamycin was started. On day 16, the blood culture grew a coagulase-negative staphylococcus sensitive to vancomycin, tetracycline, and nitrofurantoin, and resistant to ampicillin, penicillin, oxacillin, erythromycin, clindamycin, cephalothin, and trimethoprim-sulfamethoxazole. A culture of cerebrospinal fluid (CSF) showed no growth. Blood cultures were repeated on day 16 and grew a coagulase-negative staphylococcus with the same antibiotic sensitivities. Blood and CSF cultures repeated on day 19 showed no growth.

Vancomycin therapy, 45 mg/kg/day, was started on day 19 and was continued for seven days. Blood cultures drawn during treatment and following cessation of therapy were negative. On day 33, HIV antibody studies were performed on both the mother and the infant; both were positive as determined by Abbott EIA (enzyme immunoassay method). Neonatal withdrawal was managed with gradual reductions in paregoric and phenobarbital dosages, and the infant was discharged from the hospital on the 48th day of life. No serious infections or hospital visits were noted at a nine-month follow-up evaluation.

DISCUSSION

Despite a wide spectrum of morbidity,[7] an increased incidence of neonatal sepsis has not been reported in drug-dependent newborns. Recently, however, abnormal lymphocyte proliferation in response to mitogens and abnormal T4/T8 ratios have been documented in these infants.[4,8] These immunologic alterations may relate to subsequent development of AIDS, since most pediatric HIV infections and AIDS cases are acquired perinatally from infected mothers who used intravenous drugs during the pregnancy.[6] This same immunopathy could render such neonates susceptible to infections caused by bacteria of normally low virulence, such as coagulase-negative staphylococci.

Since sepsis can mimic neonatal drug withdrawal, the former should be considered when symptoms of withdrawal are not readily managed with appropriate medications. Despite low virulence, coagulase-negative staphylococcal infections can occasionally cause neonatal death.[2,9] The combination of maternal intravenous drug abuse, neonatal withdrawal, and isolation of coagulase-negative staphylococcus from neonatal cultures, even in a term infant, should prompt immediate institution of vancomycin therapy.

Acknowledgments. The authors thank Stacy B. Danheiser for her secretarial services.

From the Department of Pediatrics, Beth Israel Medical Center, Mount Sinai School of Medicine, New York.

Address correspondence to Dr Kandall, Professor, Department of Pediatrics, Beth Israel Medical Center, First Ave at 16th St, New York, NY 10003.

References

1. Munson DP, Thompson TR, Johnson DE, et al: Coagulase-negative staphylococcal septicemia: Experience in a newborn intensive care unit. *J Pediatr* 1982; 101:602–605.
2. Baumgart S, Hall SE, Campos JM, et al: Sepsis with coagulase-negative staphylococci in critically ill newborns. *Am J Dis Child* 1983; 137:461–463.
3. Stabile A, Assunta Pesaresi M, Currò V: Coagulase-negative staphylococcal septicemia in newborn babies [letter]. *Am J Dis Child* 1985; 139:541–542.
4. Rubinstein A, Sicklick M, Gupta A, et al: Acquired immunodeficiency with reversed T4/T8 ratios in infants born to promiscuous and drug-addicted mothers. *JAMA* 1983; 249:2350–2356.
5. Cowan MJ, Hellmann D, Chudwin D, et al: Maternal transmission of acquired immune deficiency syndrome. *Pediatrics* 1984; 73:382–386.
6. Recommendations for assisting in the prevention of perinatal transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus and acquired immunodeficiency syndrome. *MMWR* 1984; 73:382-386.
7. Kandall SR, Albin S, Gartner LM, et al: The narcotic-dependent mother. Fetal and neonatal consequences. *Early Hum Dev* 1977; 1:159–169.
8. Culver KW, Ammann AJ, Partridge JC, et al: Lymphocyte abnormalities in infants born to IV drug abusers. *Pediatr Res* 1986; 20:389A.
9. Kumar ML, Jenson HB, Dahms BB: Fatal staphylococcal epidermidis infections in very low-birth-weight infants with cytomegalovirus infection. *Pediatrics* 1985; 75:110–112.

An unusual presentation of lymphoma in a homosexual man

SANFORD DUBNER, MD, KEITH S. HELLER, MD

Reported cases of lymphoma in homosexual men have increased since the acquired immunodeficiency syndrome (AIDS) epidemic was first detected in the early 1980s. Lymphoma in the general population presents more commonly in the head and neck area than in any other anatomical region. Most patients are seen with disease arising in lymph nodes, but many exhibit disease originating in extranodal areas. Parotid lymphomas may arise either in the parotid parenchyma or in the lymph nodes within the parotid gland.[1] We discuss a case of lymphoma presenting as a parotid mass in a homosexual man with AIDS.

CASE REPORT

AIDS was diagnosed in a 40-year-old homosexual man when he presented with pneumocystis pneumonia six months prior to his current admission for a right-sided preauricular mass which had increased in size over the previous three months. The patient denied weakness, weight loss, night sweats, or any history of intravenous drug abuse. His history was significant for a splenectomy 20 years earlier for traumatic injury. On admission, the preauricular mass measured 5 cm × 7 cm, and there was no evidence of facial nerve paralysis. Laboratory studies revealed a white blood cell count of 6,700/mm^3, with 56% lymphocytes, 33% segmented neutrophils, 10% monocytes, and 1% eosinophils.

The patient underwent surgical exploration of the parotid region. The mass was found to be within the substance of the parotid gland. Because of the suspicion of lymphoma, an incisional biopsy of the mass was performed. On gross examination the mass was firm and pale gray. Histologic examination indicated that the mass was a malignant lymphoma, diffuse type, with large cleaved lymphocytic cells. A postoperative workup included a computed tomographic scan of the abdomen and pelvis, which showed small inguinal lymph nodes but was otherwise unremarkable. The bone marrow was normal. Tissue and serum specimens studied at the National Cancer Institute revealed the patient to be antibody positive for the human immunodeficiency virus (HIV). The viral genome was not found in B cell tumor cells.

The patient received four courses of cytoxan, adriamycin, vincristine, and prednisone in one-month intervals. Radiation therapy was not administered. A follow-up examination after five months revealed complete remission, with total shrinkage and disappearance of the parotid mass.

DISCUSSION

The incidence of non-Hodgkin's lymphoma in patients with AIDS or AIDS-related complex (ARC) has been increasing since the AIDS epidemic began in 1980.[2] These patients frequently present with extranodal sites of tumor, particularly in the central nervous system, bone marrow, or abdomen. The frequency of extranodal tumor sites is greater than in patients without AIDS and more closely approximates the occurrence rates seen in patients with congenital or iatrogenic immunodeficiency.[3] In the non-AIDS population, approximately 5% of extranodal lymphomas are located in salivary glands, and as many as 90% of salivary gland lymphomas are parotid in origin.[4] A recent review noted that the frequency with which lymphoma occurred as an isolated parotid mass was between 0.6% and 2.4% of parotidectomy specimens.[1] Patients undergoing immunosuppressive therapy for transplantation surgery are likewise at increased risk for developing lymphomas; their risk is approximately 40 times that of the general population.

A four-year survival rate of 33% in the generalized population with non-Hodgkin's lymphoma has been reported.[5] To date, the majority of men with both AIDS and non-Hodgkin's lymphoma have died.[2] Non-Hodgkin's lymphoma and AIDS occurring concurrently in a given patient confers a poor prognosis, not only because of the sequelae of progressive lymphoma, but also because of an increased incidence of opportunistic infection.

Because of the increased risk of lymphoma, the management of AIDS patients should differ from the management of other patients with parotid tumors. In the non-AIDS patient, the presence of a parotid mass is an indication for parotidectomy, generally without a preliminary biopsy. In a patient with AIDS, incisional biopsy rather than parotidectomy may be the preferred initial procedure, particularly if a needle aspiration suggests a nonepithelial tumor. If frozen section reveals an epithelial tumor, parotidectomy may be performed. Otherwise, the procedure is terminated.

From the Head and Neck Service, Department of Surgery, Booth Memorial Medical Center affiliate of New York University Medical Center, Flushing, NY.

Address correspondence to Dr Heller, Department of Surgery, Booth Memorial Medical Center, Main St at Booth Memorial Ave, Flushing, NY 11355.

The surgeon who is aware of the possibility of lymphoma in the differential diagnosis of parotid gland enlargement in the patient with acquired immunodeficiency syndrome or immunosuppression may spare his patient an unnecessary parotidectomy and all of its concomitant risks.

REFERENCES

1. Nichols RD, Rebuck JW, Sullivan JC: Lymphoma and the parotid gland. *Laryngoscope* 1982; 92:365-369.
2. Ziegler JL, Beckstead JA, Volberding PA, et al: Non-Hodgkin's lymphoma in 90 homosexual men. Relation to generalized lymphadenopathy and the acquired immunodeficiency syndrome. *N Engl J Med* 1984; 311:565-570.
3. Frizzera G, Rosai J, Dehner LP, et al: Lymphoreticular disorders in primary immunodeficiencies: New findings based on an up-to-date histologic classification of 35 cases. *Cancer* 1980; 46:692-699.
4. Hyman GA, Wolff M: Malignant lymphomas of the salivary glands. Review of the literature and report of 33 new cases, including four cases associated with the lymphoepithelial lesion. *Am J Clin Pathol* 1976; 65:421-438.
5. Freedman SI: Malignant lymphomas of the major salivary glands. *Arch Otolaryngol* 1971; 93:123-127.

Oral candidiasis and AIDS

JEFFREY S. GELWAN, MD; BURTON M. GOLD, MD; HENRY J. SHIH, MD;
CRESCENS PELLECCHIA, MD

Candidal infections of the gastrointestinal tract are common in patients with acquired immunodeficiency syndrome (AIDS). Oral candidiasis, though not diagnostic of the syndrome, has been shown to carry a poor prognosis in patients in the prodromal stage, called AIDS-related complex (ARC).[1-3] In a prospective study of high-risk patients with unexplained oral candidiasis, Klein et al[1] found that 59% of these patients acquired a major opportunistic infection or secondary cancer at a median interval of three months. The authors concluded that in high-risk patients, oral candidiasis may represent an intermediate stage in the spectrum of disease that progresses from ARC to AIDS.

The association between oral and esophageal candidiasis in patients with AIDS and ARC has recently been investigated.[4-6] Since the presence of esophageal candidiasis fulfills the definition of AIDS as determined by the Centers for Disease Control (CDC), endoscopic evaluation of high-risk patients with oral candidiasis has been undertaken. Tavitian et al,[4] in a prospective study, evaluated 25 patients with oral candidiasis (eight with AIDS and 17 with ARC) to determine the presence of esophageal candidiasis. Esophageal candidiasis was found in all 25 patients, as determined by endoscopy and cytologic analysis, though six patients with moderate esophagitis were asymptomatic. The investigators concluded that in patients with AIDS or ARC, oral candidiasis indicates the presence of esophageal candidiasis. In a separate report, Tavitian et al[5] studied ten patients with AIDS and oral candidiasis and again found that all ten had evidence of esophageal candidiasis. Although our experience in general supports the relationship between oral and esophageal candidiasis, we have found three exceptions.

CASE REPORTS

Case 1. A 36-year-old man was referred to the gastroenterology service at Nassau County Medical Center for evaluation of odynophagia of two weeks' duration. He gave a history of intravenous drug abuse and exposure to a friend with AIDS. For the prior five months, he had noted generalized lymphadenopathy and whitish patches in the oral cavity unresponsive to oral mycostatin. Laboratory tests disclosed the following: culture of the buccal mucosa positive for *Candida albicans*; elevated globulin levels in the blood; the presence of antibodies to human immunodeficiency virus (HIV); and a normal esophagram. Esophagogastroduodenoscopy was performed to evaluate odynophagia and was entirely normal.

Case 2. A 26-year-old homosexual man underwent esophagogastroduodenoscopy for evaluation of pain on swallowing. His medical history was significant for gonococcal proctitis, *Campylobacter* enterocolitis, and exposure to AIDS. The patient has been followed in both infectious disease and gastroenterology clinics for oral thrush that was minimally responsive to oral nystatin and for chronic intermittent diarrhea. Recently, he was noted to have generalized lymphadenopathy and neutropenia. Both the esophagram and esophagogastroduodenoscopy were within normal limits. Ten months later, dysphagia developed, and endoscopy performed at that time confirmed the presence of candidal esophagitis.

Case 3. A 41-year-old man was admitted to Nassau County Medical Center for evaluation of progressive weakness, fever, night sweats, dysphagia, and weight loss. His medical history was significant for intravenous drug abuse, alcoholism, alcoholic liver disease, anemia, and sickle cell trait. One year prior to admission, the patient noted the onset of oral thrush, followed by the development of odynophagia and dysphagia to both solids and liquids. On admission, his physical examination was remarkable for a temperature of 102.3°F, oral thrush, generalized lymphadenopathy, and hepatomegaly. Laboratory data revealed the following: a throat culture positive for *Candida albicans*; anemia; neutropenia; hypergammaglobulinemia; elevated erythrocytic sedimentation rate; positive anergy panel; and a bone marrow biopsy demonstrating hyperplasia of erythrocyte, granulocyte, and megakaryocyte series, with a culture positive for *Mycobacterium avium intracellulare* complex.

An esophagram (Fig 1) done to evaluate odynophagia/dysphagia revealed a long midesophageal region showing irregular thickened folds suggestive of esophagitis. Subsequently, esophagogastroduodenoscopy was performed and revealed multiple areas of erythema and friability throughout the mid and distal esophagus. No exudate or discrete ulcerations were seen. Cytologic studies were interpreted as epithelia atypism, Pap class II-III, with probable viral inclusions suggestive of herpes. No fungal structures were seen. Pathologic examination (Fig 2)

From the Division of Gastroenterology (Drs Gelwan and Pellecchia) and the Departments of Radiology (Dr Gold) and Pathology (Dr Shih), Nassau County Medical Center, East Meadow, NY.

Address correspondence to Dr Pellecchia, Division of Gastroenterology, Nassau County Medical Center, 2201 Hempstead Tpke, East Meadow, NY 11554.

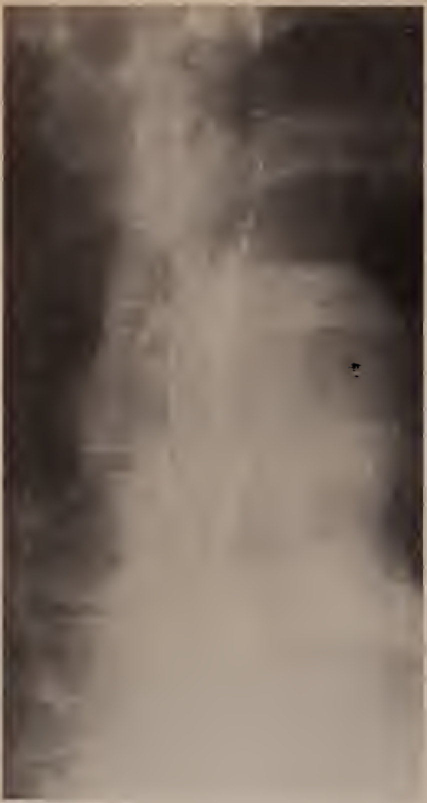


FIGURE 1. Fluoroscopic spot film of esophagram showing a long area of mid-esophagus with irregular thickened folds (Case 3).

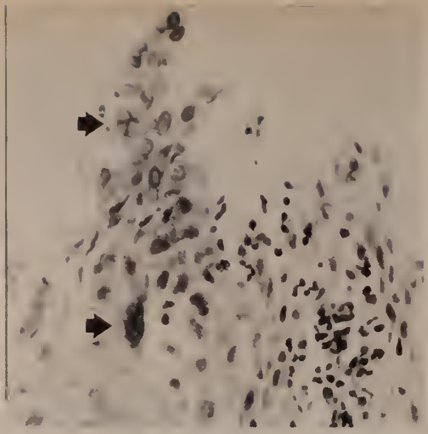


FIGURE 2. Esophageal biopsy demonstrating acute necrotizing inflammation with "ground-glass" and molded nuclei (upper arrow) forming a multinucleated giant cell (lower arrow) (hematoxylin-eosin stain, original magnification × 250) (Case 3).

demonstrated acute necrotizing inflammation with "ground-glass" and molded nuclei forming a multinucleated giant cell suggestive of herpes.

DISCUSSION

Oral candidiasis can exist without esophageal candidiasis in patients with either AIDS or ARC, even in the presence of esophageal symptoms. In patients 1 and 3 presented in this report, odynophagia was present. Yet esophageal candidiasis was ruled out during esophagogastroduodenoscopy. Case 2 illustrates that oral candidiasis can exist in patients with AIDS or ARC for some time before esophageal candidiasis develops. These cases demonstrate the need for further investigation into the association between oral and esophageal candidiasis in patients with either AIDS or ARC. Meanwhile, early upper endoscopy should be considered in evaluating patients with either oral candidiasis and/or esophageal symptoms.

REFERENCES

1. Klein RS, Harris CA, Small CS, et al: Oral candidiasis in high-risk patients as the initial manifestation of the acquired immunodeficiency syndrome. *N Engl J Med* 1984; 311:354-358.
2. Romanowski B, Weber J: Oral candidiasis and the acquired immunodeficiency syndrome [letter]. *Ann Intern Med* 1984; 101:400-401.
3. Chandrasekar PH, Molinari JA: Oral candidiasis: Forerunner of acquired immunodeficiency syndrome (AIDS)? *Oral Surg Oral Med Oral Pathol* 1985; 60:532-534.
4. Tavitian A, Raufman J-P, Rosenthal LE, et al: Oral candidiasis indicates esophageal candidiasis in patients with AIDS and AIDS-related complex, in Abstracts submitted for 50th annual scientific meeting, American College of Gastroenterology. *Am J Gastroenterology* 1985; 80:867.
5. Tavitian A, Raufman J-P, Rosenthal LE: Oral candidiasis as a marker for esophageal candidiasis in the acquired immunodeficiency syndrome. *Ann Intern Med* 1986; 104:54-55.
6. Gelb A, Miller S: AIDS and gastroenterology. *Am J Gastroenterology* 1986; 81:619-622.
7. Kodsi BE, Wickremesinghe C, Kozinn PJ, et al: Candida esophagitis. A progressive study of 27 cases. *Gastroenterology* 1976; 71:715-719.
8. Epstein JB, Truelove EL, Izutzu KT: Oral candidiasis: Pathogenesis and host defense. *Rev Infect Dis* 1984; 6:96-106.

Herpes simplex pericarditis in AIDS

ROBIN S. FREEDBERG, MD; AARON J. GINDEA, MD; DOUGLAS T. DIETERICH, MD;
JEFFREY B. GREENE, MD

Viral infection is the most common cause of pericarditis in healthy young adults and is frequently associated with clinical or occult myocarditis. Although numerous viruses can infect the heart in immunologically competent individuals,[1] herpes simplex virus has not been definitively identified as a cardiac pathogen.

Pericardial effusions are common among hospitalized patients with acquired immunodeficiency syndrome (AIDS).[2] We describe a patient with AIDS in whom a pericardial effusion developed; herpes simplex virus was cultured from the effusion.

CASE REPORT

A 44-year-old homosexual man, with no known heart disease or risk factors for coronary artery disease and no history of oral or genital herpes, was admitted to New York University Medical Center with severe weight loss, and a one-week history of sharp epigastric and substernal pain associated with several episodes of vomiting.

The physical examination was remarkable for cachexia, mild orthostasis, fever, and oral thrush. The hematocrit was 27%, the white blood cell count was 6,100/mm^3 with a relative lymphopenia, and the erythrocyte sedimentation rate was 100 mm/h. The electrocardiogram (including the QT interval) was within normal limits, although the QTc was mildly prolonged (450 msec). A chest film showed a normal cardiac silhouette and clear lungs. Lymphocyte subset studies revealed a marked depression of the helper population, with an OKT4/OKT8 ratio of 0.2, consistent with AIDS.

From the Department of Medicine, New York University Medical Center, New York.

Address correspondence to Dr Freedberg, Department of Medicine, New York University Medical Center, 530 First Ave, Suite 4F, New York, NY 10016.

The patient received antacids and ketoconazole for oral candidiasis, and hydration was achieved with intravenous crystalloids and blood. Endoscopy of the upper gastrointestinal tract revealed esophageal ulcers. A biopsy showed multinucleated giant cells, some with intranuclear inclusions suggesting cytomegalovirus or herpes infection. Treatment with intravenous acyclovir (15 mg/kg/day) was begun, and the patient continued to receive this therapy throughout his hospital stay.

One month later, acute respiratory distress developed. A chest film showed diffuse interstitial infiltrates, which prompted empiric therapy with intravenous trimethoprim-sulfamethoxazole and erythromycin. Bronchoscopic biopsy was unrevealing, but because the patient failed to respond to the treatment regimen, trimethoprim-sulfamethoxazole was replaced with intravenous pentamidine for presumed refractory *Pneumocystis carinii* infection. Progressive pulmonary failure necessitated intubation and mechanical ventilation for one week.

Three days after extubation, the patient had an episode of ventricular tachycardia, which was terminated with a precordial thump. Electrolyte levels, including magnesium and calculated ionized calcium, were within normal limits. Arterial blood gas studies showed a PO_2 of 47 mm Hg and an A-a gradient of 63 mm Hg, and the patient agreed to reinsertion of the endotracheal tube. The electrocardiogram showed nonspecific T-wave flattening and a somewhat more prolonged QTc (480 msec).

Three days later, the patient had an episode of polymorphic ventricular tachycardia with loss of consciousness, which required electrical cardioversion. An electrocardiogram revealed a markedly prolonged QT interval (QTc 570 msec) and more dramatic changes in the precordial T-wave morphology (Fig 1). A chest film showed a globular cardiac silhouette with a small increase in the cardiothoracic ratio, and a bedside two-dimensional echocardiogram confirmed a small pericardial effusion with normal valves and wall motion (Fig 2). Serial cardiac enzyme determinations were normal. Doxepin was discontinued because of the possibility of tricyclic antidepressant-related repolarization abnormalities and ventricular irritability.

Initially, there were no recurrences of ventricular ectopy on a continuous infusion of bretylium. On the following day, however, the patient had multiple episodes of torsades de pointes (Fig 3) despite therapy with this agent and intravenous administration of magnesium sulfate. An isoproterenol infusion was begun, and further episodes of ventricular tachycardia were ultimately aborted by ventricular pacing at 120 beats/min.

The patient underwent open chest biopsy of the lung and pericardium under general anesthesia without complication. Two hundred cc of serous fluid was drained from the pericardial space. Pathologic examination of the lung showed chronic inflammation, and the pericardium showed fibrosis and congestion. Stains of both tissues for bacteria, mycobacteria, fungi, *P carinii* and *Legionella* were negative. Viral culture of the lung tissue grew cytomegalovirus, and viral culture of the pericardial fluid grew herpes simplex type I. The QTc remained prolonged, and attempts to discontinue overdrive pacing led to recurrent episodes of torsades de pointes. The patient experienced progressive pulmonary failure and, two months after admission, he died.

Discussion

Many patients with AIDS suffer cardiac disorders. In an autopsy study of 41 patients who died of AIDS, ten were found to have cardiac involvement: four with Kaposi's sarcoma involving coronary arteries, myocardium and/or pericardium; three with culture-negative thrombotic endocarditis; two with nonspecific pericarditis; and one with cryptococcal myocarditis.[3] Noninvasive studies have demonstrated occult cardiac abnormalities to be common, with the echocardiographic incidence of pericardial effusion in hospitalized patients with AIDS reported to be 30%.[2]

In this patient, herpes simplex was recovered from the pericardial fluid, despite prolonged antiviral therapy with acyclovir. While a large number of prevalent viral pathogens are commonly implicated in pericarditis in otherwise healthy individuals, we are unaware of any cases of herpes pericarditis confirmed by culture findings. In one report of a healthy 22-year-old man whose pericarditis was associated with a concomitant rise in complement-fixing antibodies to both herpes zoster and herpes simplex, the authors presumed the latter to represent an anamnestic response to infection with the zoster virus.[4] Not surprisingly, in view of the fact that the viruses that commonly infect the pericardium are the same as those that typically cause myocarditis, herpes viruses have also rarely been invoked as etiologic agents in myocarditis. However, in one recent report of a patient with a cardiomyopathy that developed shortly after a varicella rash, herpes-like particles were found in a right ventricular biopsy specimen.[5]

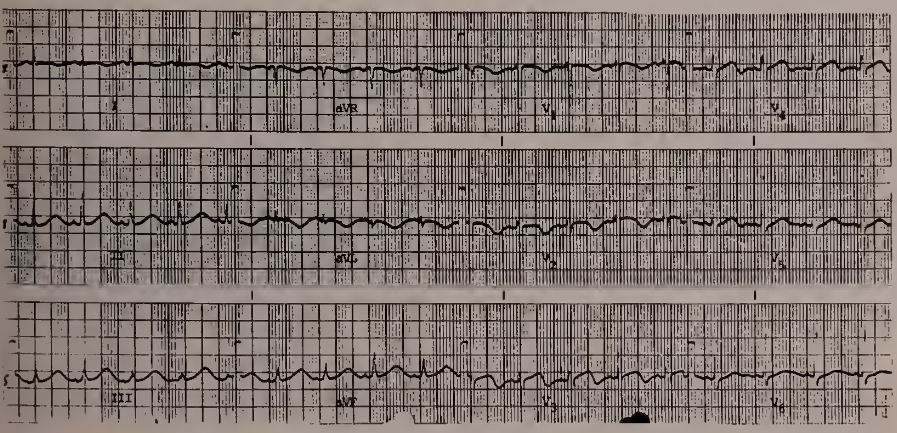


FIGURE 1. 12-lead electrocardiogram obtained after episode of symptomatic ventricular tachycardia demonstrated marked QT prolongation and repolarization abnormalities in the precordial leads.

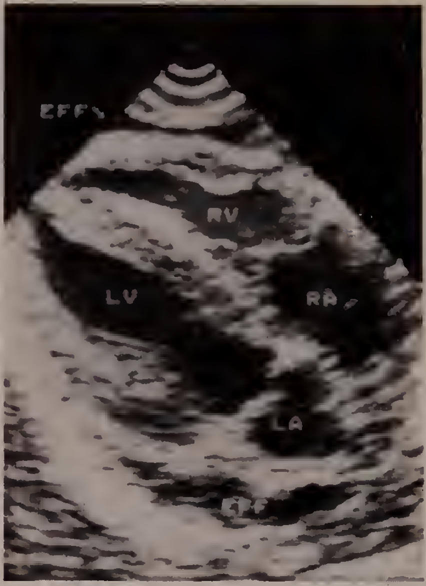


FIGURE 2. Bedside two-dimensional echocardiogram using subxiphoid approach shows small pericardial effusion (EFF) anteriorly and in the posterior atrioventricular sulcus (LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle).

Although arrhythmias are common in patients with pericarditis, those of ventricular origin are rare in the absence of underlying parenchymal heart disease.[6] The patient reported here, in whom no metabolic cause for repolarization abnormalities and torsades de pointes could be identified, may have had an associated myocarditis, which was solely manifested by these abnor-

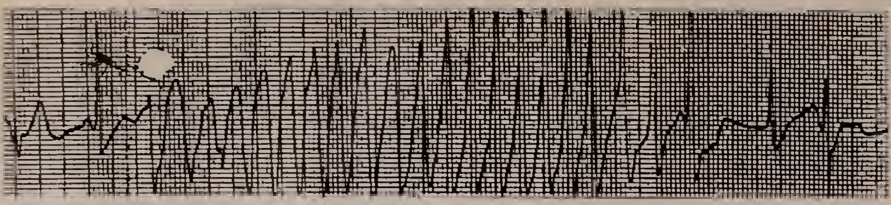


FIGURE 3. One of multiple episodes of torsades de pointes.

malities.[7,8] Although drug-related conduction abnormalities are the most frequent cause of torsades de pointes, the discontinuation of the only medication with a known association failed to correct the QT interval in this case. Given the limited experience with pentamidine, it is conceivable that this drug may have produced the detrimental QT prolongation. However, there is no report in the literature of any high-grade ventricular arrhythmia, such as torsades de pointes, associated with pentamidine.

Small pericardial effusions that lack hemodynamic significance are rarely cultured in critically ill patients. This case, however, suggests the possibility that immunocompromised patients with pericardial effusions may have a serositis caused by herpes simplex. We speculate that viruses causing self-limited illness in immunologically competent individuals may be the cause of some of the pericardial effusions found in patients with AIDS.

REFERENCES

1. Wenger NK: Infectious myocarditis. *Cardiovasc Clin* 1972; 4:167-185.
2. Reitano J, King M, Cohen H, et al: Cardiac function in patients with acquired immunodeficiency syndrome or AIDS prodrome [abstract]. *J Am Coll Cardiol* 1984; 3:525.
3. Cammarosano C, Lewis W: Cardiac lesions in the acquired immunodeficiency syndrome. *J Am Coll Cardiol* 1985; 5:703-706.
4. Winfield CR, Joseph SP: Herpes zoster pericarditis. *Br Heart J* 1980; 43:597-599.
5. Lowry PJ, Edwards CW, Nagle RE: Herpes-like virus particles in myocardium of patient progressing to congestive cardiomyopathy. *Br Heart J* 1982; 48:501-503.
6. Spodick DH: Arrhythmias during acute pericarditis: A prospective study of 100 consecutive cases. *JAMA* 1976; 235:39-41.
7. Gittleman IW, Thorner MC, Griffith GC: Q-T interval of electrocardiogram in acute myocarditis in adults with autopsy correlation. *Am Heart J* 1951; 41:78-90.
8. Karajalainen J, Viitasalo M, Kala R, et al: 24-hour electrocardiographic recordings in mid acute infectious myocarditis. *Ann Clin Res* 1984; 16:34-39.

Fatal hemoptysis in a patient with AIDS-related complex and pulmonary aspergilloma

GERARD T. LOMBARDO, MD; NANARAO ANANDARAO, MD; CHING-SHEN LIN, MD;
ANDRE ABBATE, MD; WILLIAM H. BECKER, MD

Hemoptysis is common in patients with pulmonary aspergilloma. Opinions vary as to its management. The underlying disease, the severity and frequency of hemoptysis, and the immune status of the patient have been proposed as factors to influence therapy. Unlike invasive aspergillosis, the behavior of cavitary disease in the immunocompromised host has not been fully described. This report describes a pulmonary aspergilloma in a patient with AIDS-related complex (ARC).

CASE REPORT

A 28-year-old man with a ten-year history of intravenous drug use was admitted to the Methodist Hospital several times during a 16-month period. The first admission was in March 1984 for right upper lobe pneumococcal pneumonia and was complicated by septicemia and a bronchopleural fistula. Closed tube thoracotomy resulted in complete reexpansion of the lung, although a thin-walled cavity remained in the right upper lobe.

Six months later, the patient presented with fever and cough. Sputum cultures showed no fungal or significant bacterial growth, and blood cultures were negative. The patient was anergic to skin testing with purified protein derivative and mumps antigen. His white blood cell count ranged from 4,000 to 5,000 cells/mm^3, with a normal differential. The physical examination was normal, as were the blood chemistry studies and liver function tests. The patient's fever and cough improved with ampicillin therapy. A chest radiograph showed the cavity in the right upper lobe with apical capping and elevation of the right hilum. A computed tomographic (CT) scan showed that the cavity was empty and had a thick wall (Fig 1). The patient signed himself out of the hospital against medical advice.

Six weeks later he was readmitted because of hemoptysis of approximately 200 cc over a 24-hour period. He had lost ten pounds since the previous admission and was still using intravenous drugs. The patient was afebrile and appeared thin and unkempt. His respiratory rate was 14/min, and his blood pressure was 110/70 mm Hg. There were no skin lesions or adenopathy.

Examination of his ears, nose, and throat did not reveal a source of bleeding, but extensive oral candidiasis was observed. Coarse bronchial breath sounds were heard in the right upper chest posteriorly. There were no cardiac murmurs. The abdomen

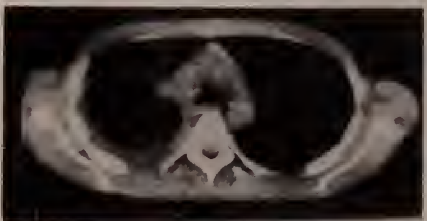


FIGURE 1. Computed tomographic (CT) scan showing an empty cavity in the right upper lobe.

From the Departments of Pulmonary Medicine (Drs Lombardo, Anandarao, and Abbate), Pathology (Dr Lin), and Medicine (Dr Becker), the Methodist Hospital, Brooklyn, NY.

Address correspondence to Dr Lombardo, Department of Medicine, Methodist Hospital, 506 6th St, Brooklyn, NY 11215.

and extremities were normal. The white blood cell count and differential had remained unchanged. There was a polyclonal hypergammaglobulinemia, and serum titers for cytomegalovirus antibody were positive. Sputum cultures subsequently grew *Aspergillus fumigatus*, *Candida albicans*, and *Mycobacterium avium intracellulare.* Blood and bone marrow cultures showed no bacterial growth. A chest radiograph and CT scan of the chest showed an irregular homogenous mass within the cavity (Figs 2, 3) suggestive of aspergilloma. Pulmonary function tests showed mild airflow restriction.

Because of continuous hemoptysis, bronchoscopy and surgical resection were advised, but the patient refused and signed himself out of the hospital. One month later, he was admitted to another hospital because of blood-streaked sputum. He was described there as a chronically ill cachectic man with oral candidiasis and diffuse lymphadenopathy. He again refused surgery. He returned to the emergency room at Methodist Hospital two months later complaining of blood-streaked sputum, had massive hemoptysis, and died one hour later. There was no clinical or laboratory evidence of coagulopathy.

An autopsy confirmed a large cavity measuring 10 cm in diameter within the right upper lobe, with fibrous adhesions to the aperture of the thoracic cage. The cavity contained an irregular, soft white and brown fungal mass mixed with clotted and fluid blood. The lining was uneven and gray-white or dark blue in color, with many small bronchi directly opening into the cavity. A 0.2-cm wide branch of the pulmonary artery had eroded into the wall of the cavity. The trachea and main bronchi contained large amounts of blood, which had been extensively aspirated in both lungs. The stomach contained 300 mL of blood. A smear and histologic sections of the cavity content showed septate and branched hyphae with budding spores and conidiophores (fruit heads). The culture yielded *Aspergillus fumigatus.* The histologic sections of the cavity wall showed diffuse lymphocytic and plasma cell infiltration and a necrotizing artery with fibrin thrombus at the rupture site. There was no evidence of *Aspergillus* invasion into the blood vessels. The rest of the lung parenchyma showed severe, acute, and chronic bronchitis but no evidence of fungal, viral, bacterial, or protozoal infection. There was marked generalized lymphadenopathy.

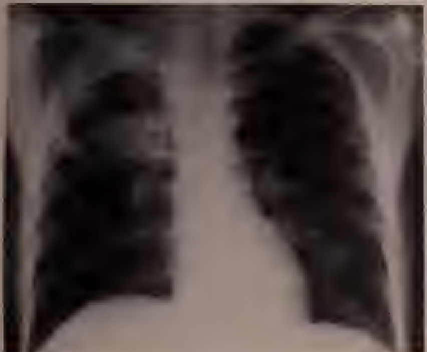


FIGURE 2. Posteroanterior roentgenogram of the chest showing an intracavitary mass.

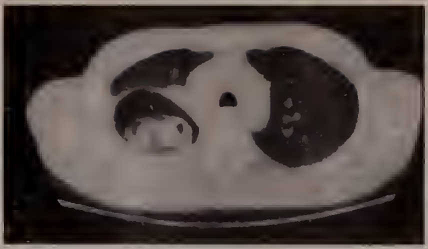


FIGURE 3. Repeat computed tomographic (CT) scan of the chest showing an intracavitary mass, six weeks after the first CT scan (Fig 1).

DISCUSSION

Aspergillus fumigatus is a saprophytic fungus which can colonize pulmonary cavities and result in mycetoma. The pathogenesis and natural course of aspergilloma have not been well defined because of the broad spectrum of underlying diseases and conditions associated with the organism. Features of aspergilloma include a solid, rounded mass on chest roentgenogram, positive sputum culture, and the presence of precipitating antibody against *Aspergillus* antigens. In immunocompetent patients, it is considered to be a slow-growing organism, although mycetomas measuring up to 3 cm have developed over several weeks.[1] Patients may be asymptomatic or have cough, dyspnea, malaise, or hemoptysis.

Hemoptysis is the most common symptom, occurring in 60–80% of cases.[1] The pathogenesis of hemoptysis may involve frictional forces within the cavity wall, hemolytic endotoxins, or proteolytic enzymes from granulocytes. The latter were implicated in the mechanism of cavitation and hemoptysis in a recent study of immunocompromised patients with acute leukemia and invasive aspergillosis.[2] As granulocyte numbers increase, there is a greater tendency for pulmonary cavitation and life-threatening hemoptysis; therefore, early operative intervention was suggested for this group. The influence of local factors and, in particular, granulocyte numbers in patients with AIDS-related complex or acquired immunodeficiency syndrome and aspergilloma has not been studied.

In cavitary aspergillomas, the incidence of fatal hemoptysis varies between 0% and 25%, depending on the population and the study design. This creates most of the controversy regarding management and especially indications for elective surgery. One study suggested a nonoperative approach for patients with aspergilloma and sarcoidosis based on a low incidence of fatal hemoptysis,[3] while another study concluded that in the same population surgery was inescapable in 30% of cases.[4] For patients with chronic lung disease[5,6] and tuberculous cavities,[7,8] similar controversies exist. Table I summarizes the outcome of patients who were treated without surgery. The underlying diseases varied, and in some studies the immune status of the subject was not always clear. Additionally, the pathogenesis of hemoptysis remains obscure since details regarding ultimate size, rate of growth, and the local environment of the mycetoma were not discussed in the context of frequency or se-

TABLE I. Clinical Course of Reported Patients with Mycetoma and Fatal Hemoptysis

Author	Patient Number	Follow-up After Diagnosis of Mycetoma (yr)	Number With Fatal Hemoptysis (%)
Kaplan & Johns	12	7.1	1*(8)
Israel et al	24	2	4 (17)
Karas et al	20	—	4†(20)
Varkey & Rose	9	4.2	0
Faulkner et al	31	2.7	1 (3)
Butz et al	14	2.5‡	0
Reddy et al	10	3.9	0
Solit et al	19	6	5 (26)
Hammerman et al	33	1	5 (15)
Rafferty et al	18	4.5	3§(17)
Total	190		23 (12)
Mean		3.9	

* Patient had hepatic failure and a coagulopathy.
† All had complications of invasive aspergillosis; one had leukemia.
‡ Data given in only eight of 14.
§ Two patients had complications of invasive aspergillosis.

verity of hemoptysis. This makes the overall figure of 10% fatal hemoptysis difficult to interpret and to apply to indications for surgery.

In patients with AIDS and ARC, fungal disease is well recognized,[9] although to our knowledge mycetoma has not been described. A 6-cm mycetoma developed in the patient described here during a six-month period. Hemoptysis was variable over a five-month period and ultimately proved fatal. The cause of death was erosion of a pulmonary artery within the cavity wall as shown by autopsy.

It is apparent that treatment of pulmonary aspergillomas must be individualized based on the progression of disease and other host factors. Considering the degree of immune dysfunction and the severity of disease, rapid growth of a mycetoma and hemoptysis in a patient with AIDS-related complex may require surgery early in the course of treatment.

Acknowledgment. The authors thank Ms Cathy Monahan for her secretarial assistance.

REFERENCES

1. Bardana EJ: The clinical spectrum of aspergillosis—part 2: Classification and description of saprophytic, allergic, and invasive variants of human disease. *CRC Crit Rev Clin Lab Sci* 1981; 13:85-159.
2. Albelda SM, Talbot GH, Gerson SL, et al: Pulmonary cavitation and massive hemoptysis in invasive pulmonary aspergillosis. *Am Rev Respir Dis* 1985; 131:115-120.
3. Kaplan J, Johns JC: Mycetomas in pulmonary sarcoidosis: Non-surgical management. *Johns Hopkins Med J* 1979; 145:157-161.
4. Israel HL, Lenchner GS, Atkinson GW: Sarcoidosis and aspergilloma: The role of surgery. *Chest* 1982; 82:430-432.
5. Karas A, Hankins JR, Attar S, et al: Pulmonary aspergillosis: An analysis of 41 patients. *Ann Thorac Surg* 1976; 22:1-7.
6. Varkey B, Rose HD: Pulmonary aspergilloma. A rational approach to treatment. *Am J Med* 1976; 61:626-631.
7. Faulkner SL, Vernon R, Brown PP, et al: Hemoptysis and pulmonary aspergilloma: Operative versus nonoperative treatment. *Ann Thorac Surg* 1978; 25:389-392.
8. Butz RO, Zvetina JR, Leininger BJ: Ten-year experience with mycetomas in patients with pulmonary tuberculosis. *Chest* 1985; 87:356-358.
9. Armstrong D, Gold JWM, Dryjanski J, et al: Treatment of infections in patients with the acquired immunodeficiency syndrome. *Ann Intern Med* 1985; 103:738-743.
10. Reddy PA, Christianson CS, Brasher CA, et al: Comparison of treated and untreated pulmonary aspergilloma. *Am Rev Respir Dis* 1970; 101:928-934.
11. Solit RW, McKeown JJ Jr, Smullens S, et al: The surgical implications of intracavitary mycetomas (fungus balls). *J Thorac Cardiovasc Surg* 1971; 62:411-422.
12. Hammerman KJ, Sarosi GA, Tosh FE: Amphotericin B in the treatment of saprophytic forms of pulmonary aspergillosis. *Am Rev Respir Dis* 1974; 109:57-62.
13. Rafferty P, Biggs BA, Crompton GK, et al: What happens to patients with pulmonary aspergilloma? Analysis of 23 cases. *Thorax* 1983; 38:579-583.

Peace Corps becomes fourth federal agency to begin screening for AIDS

As of March of this year, the Peace Corps began testing new volunteers for antibodies to the AIDS virus. This federal agency joins the military, the State Department (for employees in the Foreign Service and their dependents), and the Labor Department's Job Corps program in their requirement of an AIDS screening test.

This action on the part of the Peace Corps was prompted by "growing foreign pressure on the United States to prevent infected Americans from spreading or introducing the deadly virus within their countries," according to an article in *The Philadelphia Inquirer* (March 1, 1987, p 11-A). However, Peace Corps officials have denied any foreign influence, stating instead that AIDS screening was instituted to protect the health and safety of their volunteers.

As yet, there have been no reported cases of AIDS among the Peace Corps' 6,000 volunteers. But antibodies to the AIDS virus were detected among several volunteers who underwent the testing procedure on their own.

Number of AIDS cases expected to rise into the next century

According to figures released in March, approximately 31,000 Americans had been diagnosed as having AIDS. By 1991, that number will have increased to 270,000 (*The Philadelphia Inquirer*, March 1, 1987)—with 179,000 deaths attributable to AIDS; by the year 2000, the number of AIDS cases is expected to reach 1 million (*The New York Times*, March 4, 1987). And the Centers for Disease Control estimates that there are 50 undiagnosed carriers of AIDS for every diagnosed case.

These projections were made based on current methods of prevention and treatment. According to a survey of specialists in the fields of biotechnology, cancer, and infectious disease, 46% of those polled anticipated the availability of a safe and effective vaccine against AIDS in the 1990s, while 28% were optimistic that an effective cure would be developed before the year 2000.

Supreme Court ruling could benefit victims of AIDS

A recent Supreme Court ruling protects people who might otherwise face discrimination due to physical or mental disabilities caused by a contagious disease. The Court's seven-to-two decision was in support of a teacher with active, infectious tuberculosis who had lost her job in a Florida elementary school. The ruling applies only to employees who receive federal money and does not affect private businesses.

The teacher based her case on a 1973 federal law that prevents discrimination against handicapped people who receive financial assistance from the federal government and are qualified and able to maintain a job. "Although the decision . . . did not directly involve acquired immune deficiency syndrome, it was hailed as a major victory, if less than a total one, by homosexual rights advocates and others concerned about discrimination against victims of AIDS," notes an article in *The New York Times* (March 4, 1987, pp A1, A21). The majority opinion, written by Justice William J. Brennan, Jr, does not block the dismissal of an employee "who poses a significant risk of communicating an infectious disease to others in the workplace." Rather, it states that "employers have an affirmative obligation to make a reasonable accommodation for a handicapped employee" who is capable of carrying out his or her activities.

As AIDS is not believed to be spread through casual contact, such as in school or at the workplace, but rather through contact with contaminated blood or intimate sexual activity, it appears that persons with AIDS could claim protection under the federal Rehabilitation Act of 1973. The current ruling did not discuss the situation in which a person carries the AIDS virus and can transmit the infection but does not exhibit clinical disease.

AZT approved for sale in Britain

Azidothymidine (AZT), a drug that has shown promise as a treatment for AIDS, has been approved for distribution and sale in Britain. Although AZT is not a cure for AIDS, it seems to be effective in controlling replication of the virus in some patients with the disease.

Subsequent to the British approval, the Food and Drug Administration (FDA) approved the sale of AZT as a prescription drug in the US. In January of this year, the FDA's Anti-Infective Drugs Advisory Committee recommended that the FDA approve AZT for treatment of patients with AIDS who have advanced opportunistic infections—commonly characterized by *Pneumocystis carinii* pneumonia—and for certain patients with advanced AIDS-related complex (ARC).

In September 1986, an early stage clinical trial of AZT was stopped ahead of schedule when evaluation of the data revealed that AZT seemed to enhance short-term survival for a specific subpopulation of patients with AIDS. The FDA then granted AZT a "treatment investigational new drug application," which enabled expanded distribution of the drug to more than 3,000 eligible patients. Ultimately, on March 20, 1987, the FDA granted its approval. The drug, manufactured by Burroughs Wellcome Co, will be sold under the trade name Retrovir. Initial availability of the drug will be limited due to small supplies, but the company anticipates a supply sufficient to treat all patients with AIDS in the US by the end of the year. The long-term effects of AZT therapy are not yet known.

CDC to expand testing for AIDS

The Centers for Disease Control (CDC) is planning to test blood samples obtained anonymously from methadone and venereal disease clinics throughout the country for antibodies to the AIDS virus. CDC officials hope that this testing program will provide a more accurate measure of the incidence of AIDS infection among drug abusers and sexually active people. The program would supple-

---

NEWS BRIEFS are compiled and written by Vicki Glaser.

ment information currently obtained through a surveillance system encompassing ten hospitals nationwide.

HIV screening proposed for international travelers

A panel of 25 experts representing 12 nations evaluated the potential advantages and disadvantages of screening international travelers for antibodies to the AIDS virus. The panel, under the sponsorship of the World Health Organization, determined that such an initiative would be a waste of money and would not substantially reduce the transmission of the AIDS virus. Casual contact with a person carrying the AIDS virus does not appear to pose a risk of infection. The panel did recommend educational programs that would provide travelers with a better understanding of the disease and of the transmission of the AIDS virus.

Condom advertising gains acceptance

On Long Island, NY, an informal survey of broadcast stations and newspapers revealed that many station managers and editors would be willing to accept condom advertising under specified conditions (*The New York Times*, March 8, 1987). As yet, however, most organizations questioned had not been approached by any advertising agencies or condom manufacturers.

The stipulations for accepting condom advertising were similar among the various company spokesmen questioned: the specific advertisement would have to be "in good taste"; the material would have to be "presented in a responsible and mature manner"; and "the health issue [should be] the priority in the copy." In addition, those surveyed agreed that such advertisements should be aired at night, when children would be less likely to be watching television or listening to the radio. One publisher's position is to accept only public service advertisements prepared by government agencies.

In general, most of the media representatives have maintained an open mind toward condom advertising and stated that they would address the issue in greater detail when approached by a prospective client. According to the article, although "some religious groups have condemned the ads, saying they promote promiscuity, most local station managements felt that the commercials promote condoms as a health device, not as contraception."

Heterosexuals slow to face the reality of AIDS

Studies of the transmission of human immunodeficiency virus (HIV)—the virus that causes AIDS—have shown that HIV can be transmitted from women to men as well as from men to women, despite the preponderance of AIDS cases among men, and particularly homosexual men. In some regions of the world, AIDS occurs with similar frequencies in both men and women. It is a disease of heterosexuals and homosexuals. Although heterosexual cases of AIDS accounted for only 4% of total reported cases in 1986, the number of cases of heterosexually transmitted AIDS increased by more than 200% in 1986, compared to an 80% growth reported for cases among homosexual men and intravenous drug users (*The Atlantic Monthly*, February 1987; 249(2):39).

But while the male homosexual population has been forced to face the devastating spread of AIDS, the heterosexual community in general has distanced itself from the epidemic. "To travel around this country talking to people about AIDS is to learn quickly that most Americans still regard the emergence of the disease, which struck gays first, as an act of God," Katie Leishman writes in *The Atlantic Monthly*.

The author delves into the field of behavioral epidemiology—the study of the relationship between behavior and disease—as it relates to AIDS, as she examines the responses of people who are at risk of exposure to AIDS. She notes "a strange phenomenon: many people who know they are at extraordinarily high risk nevertheless dispense with even minimal precautions . . . . People perceive themselves to be immune, and, moreover, possessed of an intuitive power that enables them to choose safe partners."

Helping people to reduce their risk often means altering their behavior, which requires educational programs, which requires funding. However, diverting funds from research efforts to educational efforts is a controversial issue, as the long-term effectiveness of behavior modification programs has yet to be proven. The author cites a theory developed in 1956 of "four critical health beliefs that individually or in combination may lead someone to alter his behavior: the belief in susceptibility, . . . the belief in the severity of the outcome, the belief that one can have something and not know it, and the belief that the action that has been recommended will achieve the desired end."

There are several characteristics of AIDS that would weaken a behavioral modification effort, according to this theory. First, one can become infected and carry the AIDS virus for years without having any symptoms of the disease. Furthermore, before being able to persuade a person to be tested for the AIDS virus, one must be prepared to offer them an effective treatment or cure, which is not yet possible for AIDS. In addition, the risks of being exposed to AIDS and of contracting the disease once infected are not yet thoroughly understood. With respect to risk of exposure, one method recommended to ensure "safe sex" has been the use of a condom; however, condoms are associated with a 10% annual failure rate for preventing pregnancy. "In general, the higher the degree of risk that can be projected for a particular individual, the easier it is to affect his behavior . . . . When the perceived risk involved is as low as it is for most heterosexuals thinking about AIDS, the perceived severity of the outcome may not matter."

The author concludes that a continuing increase in the numbers of AIDS cases will eventually lead to panic and more dramatic behavioral changes among persons at risk.

AIDS testing proposed for transfusion recipients

The United States Public Health Service (USPHS) has recommended that people who received blood transfusions and other blood-based products in the US between 1978 and April 1985 undergo testing for antibodies to the AIDS virus (*The New York Times*, March 17, 1987). Routine screening of blood products for the AIDS virus went into effect in the spring of 1985.

Because of the low risk of acquiring AIDS from blood transfusions, government officials refrained from promoting testing for all transfusion recipients. Rather, the USPHS is leaving the decision to offer the test to the discretion of physicians. Physicians are advised to consider the following factors in determining the risk facing each patient: the number and volume of transfusions, whether the blood had been collected in areas with many AIDS cases, and the likelihood that a person, if infected with the virus, might pass it on to others.

Approximately 12,000 transfusion recipients might have been exposed to the AIDS virus during this period, according to USPHS estimates. In the region served by the Greater New York Blood Center—encompassing 18 million people in New York City, Long Island, north to Albany, and in northern New Jersey—2.1 million people received transfusions during this time. Estimates place 418 of this group at high risk of having been infected with the AIDS virus.

Health care costs of AIDS projected into next decade

Using incidence rates of AIDS determined by the Centers for Disease Control, Scitovsky and Rice (*Public Health Rep* 1987; 102(1):5–17) estimated the direct and indirect costs of AIDS in the United States for the years 1985, 1986, and 1991. Personal medical care costs are expected to increase from $630 million in 1985, to $1.1 billion in 1986, to $8.5 billion in 1991. Such expenses represent 0.2% of the estimated total personal health care costs in the US in 1985 and 0.3% in 1986; the projected figure for 1991 is 1.4%.

Other direct, nonpersonal costs of AIDS—including funds for research, screening programs, educational efforts, and general support services—have been estimated at $319 million in 1985, $542 million in 1986, and $2.3 billion in 1991. Indirect costs, attributed to time lost at the workplace, were estimated to be $3.9 billion in 1985, $7 billion in 1986, and $55 billion in 1991.

The authors recommend three approaches to reducing the health care costs associated with the AIDS epidemic: establishing alternatives to hospital care; disseminating information about AIDS to prevent further spread of the disease; and supporting research efforts aimed at developing a vaccine and a cure for AIDS.

New AIDS drug to be tested in humans

Plans to test peptide T, a new drug that appears to prevent the AIDS virus from entering cells, are underway in Sweden and the US (*Science* 1987; 235:1138–1139). Swedish investigators used the drug on an experimental basis last year to treat four patients with AIDS. While receiving peptide T for one month, the patients exhibited no adverse effects, and their conditions improved—marked by increased lymphocyte counts and reduced viral effects on their brains, as shown with nuclear magnetic resonance.

The Swedish group will now begin a randomized, controlled clinical trial with the drug, and, in the US, researchers at the National Institute of Mental Health have asked the FDA for permission to institute clinical trials with peptide T. The first stage of trials in the US would evaluate the drug's toxicity.

Although it is not known how the drug blocks passage of the AIDS virus into cells, one theory holds that peptide T interferes with binding of the virus. Peptide T does not, however, destroy the AIDS virus, and patients being treated with this drug could still have active viruses in their bodies. Therefore, researchers have suggested that, if effective, peptide T would be administered with an antiviral compound, such as azidothymidine (AZT).

Information on medical specialties

The American Board of Medical Specialties (Evanston, Ill) has prepared a general information brochure for the lay public that explains the various medical specialty areas. The pamphlet, *Which Medical Specialist for You*, outlines the training and credentials associated with each specialty and subspecialty, describes each field, and explains how to determine if a physician is a certified specialist or subspecialist.

This informative brochure educates the layman as follows:

> Specialists are doctors who concentrate on certain body systems, specific age groups, or on complex scientific techniques developed to diagnose or treat certain types of disorders. . . . Today, no one doctor can hope to master the total field of medical knowledge or maintain skills in all diagnostic tests, treatments, and procedures.

AMA gains judicial support for peer review activities

The American Medical Association (AMA) is concerned that good faith peer review activities—which are "crucial to maintaining and improving quality of care"—will cease due to physician fear of antitrust liability. The AMA has been pursuing an active role in protecting peer review immunity. Recent efforts have included intervening in court proceedings, drafting model state legislation, and drafting and supporting federal legislation to protect peer review activities. Passage of the Health Care Quality Improvement Act of 1986 ensured immunity from antitrust liability for good faith peer review activities aimed at identifying incompetent physicians.

Earlier this year, the AMA received official endorsement of good faith peer review activities from the US Department of Justice. In a letter to the AMA, Acting Assistant Attorney General Charles F. Rule stated the "the greatest potential of peer review is its ability to foster the basic goals of the antitrust laws in the health care industry—the efficient delivery of quality services in a competitive marketplace." Rule described the "harmony" between antitrust laws and legitimate peer review as follows:

> Correctly interpreted, the antitrust laws reflect the fact that the protection of competition in the health care market does not dictate that a hospital accord privileges to every practitioner who desires to use its facilities—the antitrust laws protect competition, *not competitors*. . . . Because the denial [of an incompetent practitioner, based on good faith peer review conducted by the medical staff] will enhance the quality and efficiency of health care and thereby strengthen the hospital's competitive position, peer review serves the underlying goals of the antitrust laws.

Recombinant human growth hormone receives FDA approval

On March 8, the US Food and Drug Administration (FDA) approved the sale and distribution of Humatrope, a genetically engineered form of human growth hormone manufactured by Eli Lilly Co (Indianapolis, Ind). This is the second recombinant human growth hormone product to receive FDA approval—Genentech Inc's (San Francisco, Calif) Protropin was approved in October 1985. The FDA considered Humatrope and Protropin as distinct products based on their slightly different structures, which may affect their safety and efficacy profiles. Both drugs have been approved for long-term therapy in children who fail to grow due to inadequate production of endogenous growth hormone.

Genetic defect in Down's syndrome linked to Alzheimer's disease

The same region of chromosome 21 that is duplicated in Down's syndrome (trisomy 21) may also be the genetic defect responsible for Alzheimer's disease (*Science* 1987; 235:1390). The DNA segment in question contains the gene for amyloid polypeptide, which is found in the brains of patients with Alzheimer's disease and in adults with Down's syndrome, who often exhibit lesions similar to those associated with Alzheimer's disease. Researchers have now demonstrated the presence of three copies of the gene in cells of patients with Alzheimer's disease as well as in cells of patients with Down's syndrome.

This finding "substantiates the existence of a genetic defect in Alzheimer's disease," stated authors Delabar et al. "Because with increasing age, the majority of people will develop neuropathological changes similar to those seen in Alzheimer's disease, it is reasonable to propose that an excess gene dosage of certain genes . . . could cause these changes to occur prematurely." The authors note, however, that the presence of this extra genetic material is not necessarily the sole determinant for the development of Alzheimer's disease. Environmental factors, such as viral infection, and gene regulatory systems can affect the activity of any given gene.

Baldness remedy takes a step forward

The dermatologic-drug advisory committee of the Food and Drug Administration has recommended that the FDA approve the sale and distribution of Rogaine as a prescription drug for the treatment of baldness (*The New York Times*, March 16, 1987). FDA approval is anticipated, and the drug could be on the market within a year.

The advisory committee found Rogaine to be safe, but did not support all of the manufacturer's claims of the drug's effectiveness. Rogaine's active ingredient is minoxidil, a drug used to treat high blood pressure. Rogaine, a liquid preparation, is applied twice a day to the affected region; once hair growth is achieved, application of the drug must continue or the new hair will fall out. Factors affecting the success of the drug include the age of the user, the duration of baldness, and the size and location of the bald area. The mechanism by which Rogaine stimulates hair growth is not known.

First human cancer gene sequenced

Researchers recently isolated a human cancer gene—the gene for retinoblastoma, a rare eye tumor that afflicts children—and now, another team of investigators has determined the gene code and the specific flaw that results in tumor formation in affected patients (*Science* 1987; 235: 1323, 1394–1399). This achievement could lead to a better understanding of the initiation and progression of cancer.

Unlike many other forms of cancer, in which a gene's presence or activity causes a cell to become malignant, in retinoblastoma, the absence or dysfunction of the gene is detrimental. Wen-Hwa Lee and colleagues from the University of California at San Diego are currently testing the retinoblastoma gene to determine its function. It is believed to carry instructions for the production of a normal cellular protein that may act to control cell growth.

Clinical clerkships approved for Caribbean medical school

St George's University School of Medicine, located on Grenada, has received approval from the New York State Education Department to place about 50 third- and fourth-year medical students in two-year clinical clerkships at two Brooklyn hospitals—Brooklyn Hospital and Methodist Hospital. Other New York area hospitals may join that list if they sign affiliation agreements with the university. This policy will be in effect through June 30, 1989, at which time it will be reevaluated by the New York State Education Department.

BOOKS RECEIVED

AIDS

AIDS: Facts and Issues. Edited by Victor Gong, MD, and Norman Rudnick. 388 pp. New Brunswick, NJ, Rutgers University Press, 1986. $25.00 (hardcover)

AIDS: A Manager's Guide. By Victor Schachter and Susan von Seeburg. 71 pp. New York, Executive Enterprises Publications Co, Inc, 1986. $9.95 (softcover)

What to Do About AIDS: Physicians and Mental Health Professionals Discuss the Issues. Edited by Leon McKusick, MD. 224 pp. Berkeley, University of California Press, 1986. $20.00 (hardcover)

Confronting AIDS. Directions for Public Health, Health Care, and Research. By the Institute of Medicine, National Academy of Sciences. 374 pp, illustrated. Washington, DC, National Academy Press, 1986. $24.95 (softcover)

CARDIOLOGY

Interpreting Cardiac Dysrhythmias. By J. Marcus Wharton, MD, and Nora Goldschlager, MD. 241 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $19.95 (softcover)

Clinical Electro-cardiography. A Primary Care Approach. By Ken Grauer, MD, and R. Whitney Curry Jr, MD. 544 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $24.95 (paperback)

Drugs for the Heart. Second Expanded Edition. Edited by Lionel H. Opie, MD. 238 pp, illustrated. Orlando, Fla, Grune & Stratton, Inc, 1987. $18.95 (softcover)

GENETICS

Clinical Genetics Handbook. By Ruth Y. Berini and Eva Kahn. 386 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $24.95 (softcover)

GERIATRICS

The Rejuvenation Strategy. A Medically Approved Fitness Program to Reverse the Effects of Aging. By Rene Cailliet, MD, and Leonard Gross. 287 pp, illustrated. Garden City, NY, Doubleday & Co, Inc, 1987. $16.95 (hardcover)

FAMILY PRACTICE

1986 Year Book of Family Practice. Edited by Robert E. Rakel, MD. 602 pp, illustrated. Chicago, Ill, Year Book Medical Publishers, 1986. $42.95 (hardcover)

MEDICAL BIOSTATISTICS

Biostatistics in Clinical Medicine. Second Edition. By Joseph A. Ingelfinger, MD, Frederick Mosteller, PhD, Lawrence A. Thibodeau, PhD, and James H. Ware, PhD. 339 pp, illustrated. New York, Macmillan Publishing Co, Inc, 1987. $29.95 (softcover)

MEDICAL EDUCATION

Medical School: Getting in, Staying In, Staying Human. By Keith R. Ablow. 144 pp. Baltimore, Md, Williams & Wilkins, 1987. $10.95 (paperback)

MISCELLANEOUS

A Diabetic Doctor Looks at Diabetes. His and Yours. By Peter Lodewick, MD. 232 pp, illustrated. New York, Bantam Books, 1984. $3.95 (paperback)

The Miracle Nutrient: Coenzyme Q10. The Revolutionary Scientific Breakthrough That Can Strengthen the Heart, Boost the Immune System and Extend Life Naturally. By Emile G. Bliznakov, MD, and Gerald L. Hunt. 240 pp, illustrated. New York, Bantam Books, 1986. $4.50 (paperback)

Beat the Odds. By Harold S. Solomon, MD. 311 pp, illustrated. New York, Villard Books, 1986. $19.95 (hardcover)

A Practical Guide to Financial Management of the Clinical Laboratory. Second Edition. By Janiece Sattler and Alice Smith, MT (ASCP). 159 pp, illustrated. Oradell, NJ, Medical Economics Books, 1986. $24.95 (softcover)

Dr. Nagler's Body Maintenance and Repair Book. By Willibald Nagler, MD, and Irene von Estorff, MD. 285 pp, illustrated. New York, Simon & Schuster, 1987. $18.95 (hardcover)

Managing Chronic Disease. Edited by C. Stewart Roger, MD, and Jack D. McCue, MD. 439 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $29.95 (softcover)

Jane Brody's Nutrition Book. A Lifetime Guide to Good Eating for Better Health and Weight Control. By Jane E. Brody. 552 pp, illustrated. New York, Bantam Books, 1987. $12.95 (softcover)

Human Embryo Research. Yes or No? By the Ciba Foundation. 232 pp, illustrated. New York, Methuen, Inc, 1986. $39.95 (cloth)

The All-in-One Calorie Counter. By Jean Carper. 384 pp. New York, Bantam Books, 1987. $4.50 (paperback)

Your Diabetic Child. A Comprehensive Compassionate Guide Written Especially for Parents. By Felicia M. Saunders. 192 pp, illustrated. New York, Bantam Books, 1987. $3.95 (paperback)

OBSTETRICS AND GYNECOLOGY

The Human Placenta. Clinical Perspectives. Edited by J. Patrick Lavery, MD. 288 pp, illustrated. Rockville, Md, Aspen Publishers, Inc, 1987. $59.00 (hardcover)

The Poisoned Womb. By John Elkington. 255 pp, illustrated. New York, Penguin Books, 1985. $6.95 (paperback)

OPHTHALMOLOGY

Laboratory Diagnosis in Ophthalmology. Edited by Zeynel A. Karcioglu, MD. 268 pp, illustrated. New York, Macmillan Publishing Co, 1987. $65.00 (cloth)

PEDIATRICS

Trauma in Children. Edited by Randall E. Marcus, MD. 288 pp, illustrated. Rockville, Md, Aspen Publishers, Inc, 1986. $54.00 (hardcover)

Handbook of Pediatric Cardiology. By Allan H. Rees, MD, Robert E. Solinger, MD, and Francisco Elbl, MD. 179 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1987. $23.75 (softcover)

PREVENTIVE MEDICINE

Black Adolescent Pregnancy. Prevention and Management. Edited by Norma J. Goodwin, MD. 74 pp, illustrated. New York, Human Sciences Press, Inc, 1986. $9.95 (softcover)

RADIOLOGY

Computed Tomography of the Spine. Diagnostic Exercises. By Robert Kricun, MD, and Morrie E. Kricun, MD. 290 pp, illustrated. Rockville, Md, Aspen Publishers, Inc, 1987. $56.00 (hardcover)

Radiation Therapy Simulation Workbook. By Sue Mizer, RT(T), Rhonda Rodgers Scheller, RT(T), and James A. Deye, PhD. 171 pp, illustrated. Elmsford, NY, Pergamon Press, Inc, 1986. $50.00 (hardcover)

SPORTS MEDICINE

Common Sports Injuries in Youngsters. By Richard B. Birrer, MD, and David B. Brecher, MD. 148 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $19.95 (softcover)

SURGERY

Color Atlas of Vascular Surgery. By John S. P. Lumley, MS, MBBS. 350 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1986. $119.00 (hardcover)

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Louis Abel, MD, Brooklyn. Died June 4, 1986; age 75. Dr Abel was a 1939 licentiate of the Royal College of Physicians of Edinburgh. He was a Fellow of the American College of Anesthesiologists and a member of the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Roy Chapman Ainsworth, MD, Utica. Died February 17, 1987; age 81. Dr Ainsworth was a 1934 graduate of the University of Rochester School of Medicine-Dentistry, Rochester. He was a member of the Academy of Medicine, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

John Lamont Alley, MD, Toronto, Ontario, Canada. Died November 23, 1986; age 79. He was a 1933 graduate of Cornell University Medical College, New York. Dr Alley was a member of the Medical Society of the County of Ulster and the Medical Society of the State of New York.

Adolph A. Apton, MD, New York. Died February 17, 1987; age 94. He was a 1920 graduate of Medizinische Fakultaet der Johann Wolfgang Goethe Universitaet, Frankfurt-Am-Main, Hessen. Dr Apton was a Qualified Fellow of the International College of Surgeons and a member of the Pan American Medical Association, the New York County Medical Society, and the Medical Society of the State of New York.

Armand U. Aquilino, MD, Syracuse. Died January 28, 1987; age 87. Dr Aquilino was a 1925 graduate of State University of New York Health Science Center at Syracuse. He was a member of the Academy of Medicine, the New York State Dermatological Society, the American Academy of Dermatology, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Benjamin John Barbanti, MD, Brooklyn. Died February 13, 1987; age 73. Dr Barbanti was a 1939 graduate of Facolta di Medicina e Chirurgia dell'Universita di Bologna. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Morris Bien, MD, Scarsdale. Died February 3, 1987; age 75. He was a 1938 graduate of Friedrich-Wilhelms-Universitaet Medizinische Fakultaet, Berlin. Dr Bien was a Fellow of the American College of Anesthesiologists and a Diplomate of the American Board of Anesthesiology. His memberships included the Academy of Medicine, the American Society of Anesthesiologists, Inc, the New York County Medical Society, and the Medical Society of the State of New York.

Saul Blau, MD, New York. Died January 31, 1987; age 74. Dr Blau was a 1941 licentiate of the Royal College of Physicians of Edinburgh. He was a Diplomate of the American Board of Dermatology, Inc, and a member of the Academy of Medicine, the New York State Dermatological Society, the American Academy of Dermatology, the Society for Investigative Dermatology, the New York County Medical Society, and the Medical Society of the State of New York.

Peter Brikates, MD, Carefree, Arizona. Died February 21, 1987; age 82. He was a 1932 graduate of University of Vermont College of Medicine, Burlington. Dr Brikates was a Fellow of the American College of Surgeons, a Diplomate of the American Board of Ophthalmology, and a member of the American Academy of Ophthalmology and Otolaryngology, the New York County Medical Society, and the Medical Society of the State of New York.

Salvatore J. Callerame, MD, Bronx. Died November 30, 1986; age 64. Dr Callerame was a 1947 graduate of State University of New York Health Science Center at Brooklyn. He was a member of the American Occupational Medical Association, the Bronx County Medical Society, and the Medical Society of the State of New York.

Jerome S. Coles, MD, New York. Died February 4, 1987; age 74. He was a 1936 graduate of New York University School of Medicine, New York. Dr Coles was a Diplomate of the American Board of Surgery, and a member of the New York County Medical Society and the Medical Society of the State of New York.

Louis Marius De Cicco, MD, New Rochelle. Died January 25, 1987; age 90. Dr De Cicco was a 1919 graduate of the University of Vermont College of Medicine, Burlington. He was a member of the Academy of Medicine, the American Geriatrics Society, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Fred Ennis Dexter, MD, Albany. Died January 17, 1987; age 73. He was a 1941 graduate of Albany Medical College of Union University, Albany. Dr Dexter was a Qualified Fellow of the International College of Surgeons, a Diplomate of the American Board of Orthopedic Surgery, and a member of the American Academy of Orthopaedic Surgeons, the Medical Society of the County of Albany, and the Medical Society of the State of New York.

Milton Eller, MD, North Woodmere. Died January 8, 1987; age 71. Dr Eller was a 1941 graduate of the University of Louisville School of Medicine, Louisville. He was a Fellow of the American College of Physicians and the American College of Chest Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the New York State Society of Internal Medicine, the International Society of Internal Medicine, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

George C. Escher, MD, Scarsdale. Died December 30, 1986; age 74. He was a 1937 graduate of State University of

New York Health Science Center at Brooklyn. Dr Escher was a member of the Endocrine Society, the Society of Surgical Oncology, the American Association for Cancer Research, the New York County Medical Society, and the Medical Society of the State of New York.

Goffredo G. Gensini, MD, Syracuse. Died October 20, 1986; age 58. Dr Gensini was a 1952 graduate of Facolta di Medicina e Chirurgia dell'Universita di Firenze. He was a Fellow of the American College of Cardiology and the American College of Chest Physicians. Dr Gensini was a member of the Onondaga County Medical Society and the Medical Society of the State of New York.

Louis Goldstein, MD, Teaneck, New Jersey. Died November 3, 1986; age 75. Dr Goldstein was a 1937 graduate of Medizinische Fakultaet der Universitaet, Bern. He was a member of the American Psychiatric Association, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Joseph Giuffre, MD, Bronx. Died February 13, 1987; age 81. Dr Giuffre was a 1932 graduate of Indiana University School of Medicine, Indianapolis. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Herbert Corle Hageman, MD, Ridgefield, Connecticut. Died September 29, 1986; age 88. Dr Hageman was a 1925 graduate of Albany Medical College of Union University, Albany. He was a member of the Medical Society of the County of Fulton and the Medical Society of the State of New York.

Merton Charles Hatch, MD, Syracuse. Died February 6, 1987; age 82. He was a 1929 graduate of State University of New York Health Science Center at Syracuse. Dr Hatch was a Fellow of the American College of Obstetricians and Gynecologists, a Diplomate of the American Board of Obstetrics and Gynecology, and a member of the Academy of Medicine, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Emanuel Hellreich, MD, Brooklyn. Died January 29, 1987; age 81. Dr Hellreich was a 1933 graduate of Universitaet Wien, Medizinische Fakultaet, Wien. He was a Fellow of the American College of Allergists. His memberships included the American Academy of Allergy, the American Geriatrics Society, the American Association for Cancer Research, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Morris Herman, MD, Bayside. Died February 5, 1987; age 81. Dr Herman was a 1930 graduate of New York University of Medicine, New York. He was a Diplomate of the American Board of Psychiatry and Neurology and a member of the Academy of Medicine, the New York Neurological Society, the American Neurological Association, the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Emery Sylvester Hetrick, MD, New York. Died February 3, 1987; age 57. Dr Hetrick was a 1957 graduate of Cornell University Medical College, New York. He was a Diplomate of the American Board of Psychiatry and Neurology and a member of the American Psychiatric Association, the American Medical Society for Alcoholism, the New York County Medical Society, and the Medical Society of the State of New York.

Frank Frederick Holmberg, MD, Sag Harbor. Died January 29, 1987; age 84. Dr Holmberg was a 1927 graduate of Cornell University Medical College, New York. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Sidney Immergut, MD, New York. Died February 17, 1987; age 84. He was a 1926 graduate of New York University School of Medicine, New York. Dr Immergut was a Qualified Fellow of the International College of Surgeons, a Fellow of the American College of Surgeons, and a Diplomate of the American Board of Urology. He was a member of the American Urological Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Martin Israel, MD, Margate, Florida. Died February 21, 1987; age 87. Dr Israel was a 1926 graduate of Columbia University College of Physicians and Surgeons, New York. He was a member of the American Academy of Family Practice, the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Martin L. Kaye, MD, New York. Died February 3, 1987; age 81. Dr Kaye was a 1929 graduate of State University of New York Health Science Center at Brooklyn. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Isidore Koulack, MD, Bronx. Died February 17, 1987; age 93. He was a 1927 graduate of Tufts University School of Medicine, Boston. Dr Koulack was a member of the American Academy of Family Practice, the Bronx County Medical Society, and the Medical Society of the State of New York.

John A. Lawler, MD, Bronx. Died February 24, 1987; age 86. He was a 1924 graduate of Columbia University College of Physicians and Surgeons, New York. Dr Lawler was a Fellow of the American College of Surgeons, a Diplomate of the American Board of Surgery, and a member of the Academy of Medicine, the New York State Surgical Society, the New York County Medical Society, and the Medical Society of the State of New York.

Samuel Benjamin Levy, MD, Camarillo, California. Died December 25, 1986; age 82. Dr Levy was a 1928 graduate of New York University School of Medicine, New York. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Jesse Waring Mahoney, MD, East Setauket. Died February 24, 1987; age 81. Dr Mahoney was a 1940 graduate of New York University School of Medicine, New York. Dr Mahoney was a Fellow of the American College of Surgeons, a Diplomate of the American Board of Surgery, and a member of the Academy of Medicine, the American Association for the Surgery of Trauma, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Michael Gregory Malko, MD, Brooklyn. Died February 2, 1987; age 74. Dr Malko was a 1939 graduate of State University of New York Health Science Center at Brooklyn. He was a member of the American Geriatrics Society, the American Occupational Medical Association, the Kings County Medical Society, and the Medical Society of the State of New York.

Benjamin L. Matthews, MD, Vestal. Died February 3, 1987, age 81. He was a 1929 graduate of State University of New York Health Science Center at Brooklyn. Dr Matthews was a Fellow of the American College of Surgeons and a member of the Broome County Medical Society and the Medical Society of the State of New York.

Bernard Mattikow, MD, Brooklyn. Died February 8, 1987; age 88. Dr Mattikow was a 1927 graduate of University of Maryland School of Medicine, Baltimore. He was a member of the Kings County Medical Society and the Medical Society of the State of New York.

Malcolm M. Merrill, MD, Canandaigua. Died February 1987; age 71. Dr Merrill was a 1946 graduate of Queens University Faculty of Medicine, Kingston. He was a member of the Ontario County Medical Society and the Medical Society of the State of New York.

Joseph Michaels, MD, Bayside. Died February 8, 1987; age 76. He was a 1938 graduate of Medizinische Fakultaet der Ludwig Maximilians Universitaet, Muenchen, Bayern. Dr Michaels was a Fellow of the American Psychiatric Association, a Diplomate of the American Board of Psychiatry and Neurology, and a member of the New York County Medical Society and the Medical Society of the State of New York.

Josef Munzesheimer, MD, Deerfield Beach, Florida. Died February 1, 1987; age 85. Dr Munzesheimer was a 1927 graduate of Medizinische Fakultaet der Ludwig Maximilians Universitaet, Muenchen, Bayern. He was a member of the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Medical Society of the County of Chautauqua, and the Medical Society of the State of New York.

Albert E. Nesbitt, MD, Bronx. Died January 14, 1987; age 79. He was a 1938 graduate of Howard University College of Medicine, Washington, DC. Dr Nesbitt was a member of the New York Cardiological Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

Bartholomew A. Nigro, MD, Kenmore. Died February 2, 1987; age 97. Dr Nigro was a 1920 graduate of State University of New York at Buffalo School of Medicine. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Robert Andrew Oliveri, MD, New York. Died January 28, 1987; age 47. He was a 1965 graduate of the University of Ottawa Faculty of Medicine, Ottawa, Canada. Dr Oliveri was a member of the New York County Medical Society and the Medical Society of the State of New York.

Charles Joseph Popovits, MD, Riverdale. Died December 14, 1986; age 78. He was a 1933 graduate of New York Medical College, Flower and Fifth Avenue Hospitals, New York. Dr Popovits was a Fellow of the American College of Allergists and a Diplomate of the American Board of Internal Medicine and the American Board of Allergy and Immunology. His memberships included the American Academy of Allergy, the American Society of Clinical Hypnosis, the New York County Medical Society, and the Medical Society of the State of New York.

Robert Reuben Princer, MD, Brooklyn. Died February 7, 1986; age 82. Dr Princer was a 1929 graduate of New York Medical College, Flower and Fifth Avenue Hospitals, New York. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

George J. Righter, MD, Rensselaerville, NY. Died January 26, 1987; age 81. He was a 1929 graduate of the State University of New York Health Science Center at Syracuse. Dr Righter was a Fellow of the American College of Surgeons, a Diplomate of the American Board of Otolaryngology, and a member of the Medical Society of the County of Albany and the Medical Society of the State of New York.

Eugene B. F. Riley, MD, Nokomis, Florida. Died January 18, 1987; age 64. Dr Riley was a 1945 graduate of New York University School of Medicine, New York. He was a member of the American Psychiatric Association, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Peter-Cyrus Rizzo III, MD, New York. Died February 5, 1987; age 56. He was a 1957 graduate of Cornell University Medical College, New York. Dr Rizzo was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Orthopedic Surgery. His memberships included the American Academy of Orthopaedic Surgeons, the New York County Medical Society, and the Medical Society of the State of New York.

Harry Rosenwasser, MD, Holmes. Died January 31, 1987; age 85. He was a 1927 graduate of State University of New York Health Science Center at Brooklyn. Dr Rosenwasser was a Diplomate of the American Board of Otolaryngology and a member of the Academy of Medicine; the New York Otological Society; the American Academy of Otolaryngology; the American Laryngological, Rhinological and Otological Society; the American Otological Society, Inc; the New York County Medical Society; and the Medical Society of the State of New York.

Joseph Edmund Rutecki, MD, Buffalo. Died January 28, 1987; age 66. Dr Rutecki was a 1945 graduate of the State University of New York at Buffalo School of Medicine. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. Dr Rutecki was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

A. Allen Samwick, MD, Brooklyn. Died February 19, 1987; age 79. He was a 1935 graduate of Universitaet Wien, Medizinische Fakultaet, Wien. Dr Samwick was a member of the International Society of Hematology, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Joseph H. Siris, MD, Floral Park. Died January 22, 1987; age 79. Dr Siris was a 1932 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Neurological Surgery and the American Board of Psychiatry and Neurology. His memberships included the New York Society of Neurosurgery, the American Academy of Neurology, the American Association of Neurological Surgeons, the Association for Research in Nervous and Mental Diseases, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Jacob Smith, MD, Bourne, Massachusetts. Died January 23, 1987; age 81. He was a 1933 graduate of Rush Medical College, Chicago. Dr Smith was a Fellow of the American College of Surgeons and the American College of Chest Physicians, and a member of the New York County Medical Society and the Medical Society of the State of New York.

Francis Davis Speer, MD, New York. Died January 3, 1987; age 78. He was a 1933 graduate of Hahnemann Medical College of Philadelphia. Dr Speer was a Fellow of the American College of Pathologists, a Diplomate of the American Board of Pathology, and a member of the New York Pathological Society, the American Society of Clinical Pathologists, the New York County Medical Society, and the Medical Society of the State of New York.

Anna R. Speigelman, MD, New York. Died January 20, 1987; age 84. She was a 1927 graduate of State University of New York Health Science Center at Brooklyn. Dr Spiegelman was a member of the Clinical Society of the New York Diabetes Association, the New York Cardiological Society, the American Public Health Association, the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Harry Sporn, MD, Old Westbury. Died February 19, 1987; age 78. Dr Sporn was a 1937 graduate of Universitaet Wien, Medizinische Fakultaet, Wien. He was a member of the American Geriatrics Society, the Nassau County Medical Society, and the Medical Society of the State of New York.

Leon David Star, MD, Lawrence. Died February 3, 1987; age 74. He was a 1941 graduate of State University of New York Health Science Center at Brooklyn. Dr Star was a Fellow of the American College of Preventive Medicine and the American College of Surgeons, a Qualified Fellow of the International College of Surgeons, and a Diplomate of the American Board of Surgery and the American Board of Preventive Medicine, Inc. His memberships included the Academy of Medicine, the New York Society for Thoracic Surgery, the Aerospace Medical Association, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Otto Steinbrocker, MD, Lake Worth, Florida. Died January 2, 1987; age 89. Dr Steinbrocker was a 1927 graduate of the New York University School of Medicine, New York. He was a Fellow of the American College of Physicians and a member of the Academy of Medicine, the American Rheumatism Association, the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Sonia Sara Stirt, MD, Forest Hills. Died February 7, 1987; age 85. Dr Stirt was a 1927 graduate of Friedrich-Wilhelms-Universitaet Medizinische Fakkultaet, Berlin. She was a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

T. Campbell Thompson, MD, Baltimore, Maryland. Died November 21, 1986; age 84. Dr Thompson was a 1928 graduate of Johns Hopkins University School of Medicine, Baltimore, Md. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Roland J. Walker, MD, Panama City, Florida. Died October 5, 1986; age 81. He was a 1931 graduate of Georgetown University School of Medicine, Washington, DC. Dr Walker was a Fellow of the American College of Cardiology and a member of the Aerospace Medical Association, the American Occupational Medical Association, the American Geriatrics Society, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Steven H. Weiss, MD, Syracuse. Died January 20, 1987; age 31. Dr Weiss was a 1980 graduate of Albany Medical College of Union University, Albany. He was a member of the Onondaga County Medical Society and the Medical Society of the State of New York.

Louis Wexler, MD, New York. Died February 7, 1987; age 70. He was a 1942 graduate of Faculte de Medecine de l'Universite de Lausanne, Lausanne. Dr Wexler was a Diplomate of the American Board of Dermatology, Inc, and a member of the American Academy of Dermatology, the New York County Medical Society, and the Medical Society of the State of New York.

Duncan L. Wormer, MD, Portville. Died January 2, 1986; age 91. Dr Wormer was a 1920 graduate of the University of Buffalo School of Medicine. He was a member of the Medical Society of the County of Cattaraugus and the Medical Society of the State of New York.

Rebecca Zeiger-Prozan, MD, Hempstead. Died January 31, 1987; age 94. Dr Zeiger-Prozan was a 1924 graduate of Woman's Medical College of Pennsylvania, Philadelphia. She was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1983; 83:1089-1094). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith^2 reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83: 925-927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337-338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, P.O. Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

Cover to cover . . . . always good reading.

NEW YORK STATE
JOURNAL OF MEDICINE

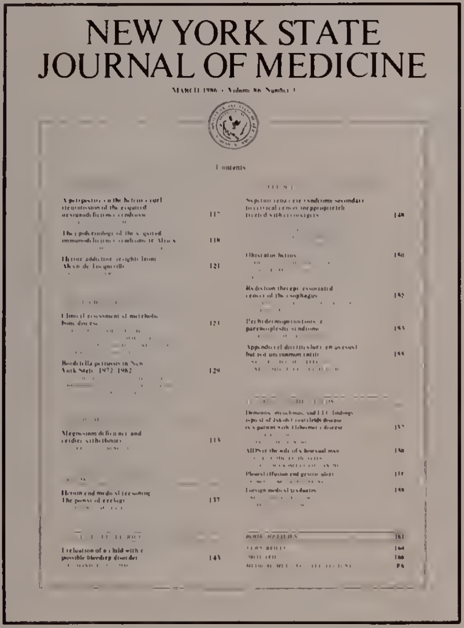
MEDICAL MALPRACTICE
AN OVERVIEW

Contents

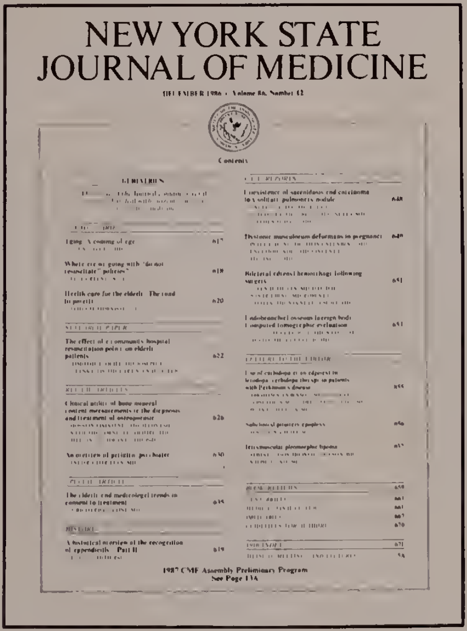
NEW YORK STATE
JOURNAL OF MEDICINE

CIGARETTE SMOKING,
TOBACCO, AND HEALTH
INTERNATIONAL PERSPECTIVES

Contents


NEW YORK STATE
JOURNAL OF MEDICINE

Contents


NEW YORK STATE
JOURNAL OF MEDICINE

Contents

1987 CME Assembly Preliminary Program
See Page 13A

In 1987, the *New York State Journal of Medicine* promises to provide timely and stimulating commentary, challenging research papers, analysis of clinical trends, debate on topical issues, and in-depth coverage of medical news. The journal will make a welcome gift to a colleague or student in the United States or abroad.

A free copy of the July 1985 issue, a special issue on smoking, will be included with each subscription while the supply lasts.

SUBSCRIPTION ORDER FORM Clip and mail

Circulation Department
New York State Journal of Medicine
420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042

Name____________________________________

Address____________________________________

____

	1 Year	2 Years	3 Years
U.S. & Canada	☐ $25	☐ $45	☐ $60
Foreign	☐ $30	☐ $55	☐ $75

Payment required with order.

Medical Management Consultant on Call

After 16 years as an office manager for a busy medical practice, Mary Ann Hamburger of Maplewood (763-7394) discovered "a tremendous need for a complete service for the business side of a medical office set-up." For the past 17 years Mary Ann has channeled her widespread contacts with "hospital administrators, medical societies, insurance brokers, physicians, attorneys, accountants and medical and furniture suppliers" and her knowledge of all aspects of the business side of medical offices to become a successful Medical Management Consultant.

Mary Ann's years of experience working on the managerial level in a medical office has resulted in her ability to recognize where the problems are in a medical office and to properly evaluate her clients so these problems will not arise. "Part of finding a location includes choosing a hospital where a doctor's area of specialization is needed," she points out. After location is determined, Mary Ann will find the right office space, hire effective and efficient staff, and also include staff training. In addition, Mary Ann focuses on two very critical areas in setting up a medical office—she will assist in obtaining medical equipment, furnishings and supplies as well as establishing fee schedules and the necessary CPT codes.

She assures that while the medical office is in the process of being set-up she will offer "personalized attention seven days a week."

Mary Ann will provide the same individual attention when a physician seeks her consultation to change a location and/or hire or join an associate or medical group. After finding a new location, the multi-faceted set-up procedure can begin. And it should be noted that Mary Ann can reorganize an existing practice as well according to the doctor's needs.

She also serves as an adviser for area hospitals and will run ads, screen and interview physicians to fill staff positions for the hospital. She has taught management seminars in hospitals and has worked with area institutions' medical education departments. She has also given seminars on "The Business Side of a Medical Practice." Mary Ann is currently studying the use of computers in medical field and will provide up-to-date information on billing and check-in procedures.

The Medical Management Consultant service eliminates a busy doctor's added responsibility of establishing and overseeing a state-of-the-art office. "I make sure that when the doctor opens his or her door for the very first patient all he or she has to do is concentrate on the medical side of the practice," Mary Ann explained.

Her special services can be utilized by calling, 763-7394.

Reprinted with Permission from the Jewish News Who's Who, Page 11, November 6, 1986.

MARY ANN HAMBURGER
MARY ANN HAMBURGER ASSOCIATES

74 Hudson Avenue
Maplewood, New Jersey 07040

201-763-7394

Freedom from pain

Just one part of pain relief therapy.

Vicodin® provides greater patient acceptance

COMPARATIVE PHARMACOLOGY OF THREE ANALGESICS

	CONSTIPATION	RESPIRATORY DEPRESSION	SEDATION	EMESIS	PHYSICAL DEPENDENCE
HYDROCODONE		X			X
CODEINE	X	X	X	X	X
OXYCODONE	XX	XX	XX	XX	XX

Blank space indicates that no such activity has been reported.

Table adapted from Facts and Comparisons (Nov.) 1984 and Catalano RB. The medical approach to management of pain caused by cancer. "Semin Oncol" 1975, 2; 379-92 and Reuler JB, et. al. The chronic pain syndrome: misconceptions and management. "Ann Intern Med" 1980; 93; 588-96.

- Vicodin offers: less nausea, less sedation, less constipation.

...and longer lasting pain relief– up to 6 hours.

- Vicodin contains hydrocodone not codeine. In one study, 10 mg. of hydrocodone alone was shown to be as effective as 60 mg. of codeine.[1]
- In a double-blind study, Vicodin (2 tablets), provided longer lasting pain relief than 60 mg. of codeine.[2]

Plus...

- Vicodin offers the convenience of CIII prescribing.
- Dosage flexibility–1 tablet every 6 hours or 2 tablets every 6 hours (up to 8 tablets in 24 hours).

1. Hopkinson JH III: *Curr Ther Res* 24: 503-516, 1978
2. Beaver, WT *Arch Intern Med*, 141:293-300, 1981.

vicodin®

hydrocodone bitartrate 5 mg. (Warning: May be habit forming) with acetaminophen 500 mg.

The original hydrocodone analge

Please see adjacent page for brief summary of prescribing information.

vicodin® CIII

hydrocodone bitartrate 5mg (Warning. May be habit forming). and acetaminophen 500mg.

INDICATIONS AND USAGE: For the relief of moderate to moderately severe pain.
CONTRAINDICATIONS: Hypersensitivity to acetaminophen or hydrocodone.
WARNINGS
Drug Abuse and Dependence: VICODIN is subject to the Federal Controlled Substances Act (Schedule III). Psychic dependence, physical dependence and tolerance may develop upon repeated administration of narcotics; therefore, VICODIN should be prescribed and administered with the same caution appropriate to the use of other oral-narcotic-containing medications.
Respiratory Depression: At high doses or in sensitive patients, hydrocodone may produce dose-related respiratory depression by acting directly on brain stem respiratory centers. Hydrocodone also affects centers that control respiratory rhythm, and may produce irregular and periodic breathing.
Head Injury and Increased Intracranial Pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a preexisting increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.
Acute Abdominal Conditions: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions.
PRECAUTIONS
Special Risk Patients: VICODIN should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture.
Information for Patients: VICODIN, like all narcotics, may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery; patients should be cautioned accordingly.
Cough Reflex: Hydrocodone suppresses the cough reflex; caution should be exercised when VICODIN is used postoperatively and in patients with pulmonary disease.
Drug Interactions: The CNS-depressant effects of VICODIN may be additive with that of other CNS depressants. When combined therapy is contemplated, the dose of one or both agents should be reduced. The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone. The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus.
Usage in Pregnancy: Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose. There are no adequate and well-controlled studies in pregnant women. VICODIN should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.
Nonteratogenic Effects: Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent. The intensity of the syndrome does not always correlate with the duration of maternal opioid use or dose.
Labor and Delivery: Administration of VICODIN to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used.
Nursing Mothers: It is not known whether this drug is excreted in human milk; therefore, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.
Pediatric Use: Safety and effectiveness in children have not been established.
ADVERSE REACTIONS
Central Nervous System: Sedation, drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, dizziness, psychic dependence, mood changes.
Gastrointestinal System: Nausea and vomiting may occur, they are more frequent in ambulatory than in recumbent patients. Prolonged administration of VICODIN may produce constipation.
Genitourinary System: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported
Respiratory Depression: (See WARNINGS.)
DOSAGE AND ADMINISTRATION: Dosage should be adjusted according to the severity of the pain and the response of the patient. However, tolerance to hydrocodone can develop with continued use, and the incidence of untoward effects is dose related. The usual dose is one tablet every six hours as needed for pain (If necessary, this dose may be repeated at four-hour intervals.) In cases of more severe pain, two tablets every six hours (up to eight tablets in 24 hours) may be required.
Revised January, 1987 MR 1987 5786

Knoll Pharmaceuticals
A Unit of BASF K&F Corporation
Whippany, New Jersey 07981

BASF Group

knoll®

REAL ESTATE FOR SALE OR RENT—CONT'D

FULL OR PART-TIME consultation and waiting room for psychiatrist, psychologist, nutritionist or surgeon in newly renovated internist's office located on Bell Blvd. in Bayside, NY across from Bay Terrace Shopping Center. Available immediately. Reply Dept. 440 c/o NYSJM.

NORTH SHORE: Long Island's Gold coast. Professional home/office. Custom designed 8 year Ranch on 1 acre in prestigious Roslyn Harbor. 4 bdrms., 2 baths, formal dining room, large country kitchen, den/stone fplc. Adjoining professional wing has 2 large rooms and bath. Owner relocating. Priced for quick sale . . . $595,000. Merrill Lynch Realty, (516) 484-6767.

PARK AVENUE—LOWER 70's. Elegantly furnished, air conditioned, well equipped, compact office with private entrance. Available full time on Wednesday, weekends and evenings. Flexible hours on other days. Ideal as second office. Suitable for surgeon, neurologist or other specialist. Please call Tuesday or Thursday morning. (212) 876-3286.

MISCELLANEOUS

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuarial certification and employee statements of participation—partnership agreements and professionals incorporation—No insurance required—references upon request—Wachstock and Dienstag Attorneys at Law, 122 Cutter Mill Road, Great Neck, NY 11021 (516) 773-3322.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta, GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

PROFESSIONAL MISCONDUCT ATTORNEYS. William L. Wood Jr., formerly Executive Director of the New York State Office of Professional Discipline and Anthony Z. Scher, formerly Director of Prosecutions, announce the opening of their law office, Wood & Scher. Our recent tenure as chief enforcement officers has given us experience which will allow us to represent physicians in professional misconduct proceedings, malpractice licensure, license restoration, controlled drug proceedings, reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practitioners. Wood & Scher, Attorneys at Law, One Chase Road, Scarsdale, New York 10583. Telephone 914-723-3500.

MISCELLANEOUS—CONT'D

HAVE YOU RECEIVED ANY COMMUNICATION FROM THE OFFICE OF PROFESSIONAL MEDICAL CONDUCT? If affirmative, contact Susan Kaplan, Attorney-at-Law, (212) 877-5998. Practice limited to the crucial legal problems directly affecting your license to practice medicine—with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professions), and as an Assistant District Attorney in Nassau County. Susan Kaplan, Esq., 165 West End Avenue, Suite 27P, New York, N.Y. 10023.

TUNA THERAPY SEMINARS: Does your brain need a rest, your body need exercise, are your lungs in need of fresh air? Giant tuna fishing from Montauk Point, New York, aboard Montauk's finest charter boat "Dawn". Charters available for 1 to 4 serious medical anglers. Capt. Greg Beacher, RD-2, Box 16, Benson Drive, Montauk, NY 11954, (516) 668-9056.

COMPUTERIZE YOUR BILLING—Remarkably easy-to-use software automatically prints bills, statements, insurance forms. Menu-driven. Reports ageing balances, referral sources, income by time period, procedure & more. Installs automatically. IBM and compatibles. Solo practice, only $399. Group practice, only $499. Free information. Demonstration disk with complete manual, $19 plus $3 shipping, credited to purchase. Call or write REM Systems, Inc., Dept. NY5, 281 Dug Hill Road, Brownsboro, AL 35741 (205) 534-3457. VISA, MC Accepted.

PORTABLE RADIOLOGY SERVICE: abdominal + pelvic ultrasound, cardiac echos including 2D + m-mode. portable x-rays. NYC and suburbs. Schedule weekly/bi-weekly in your office or visits to your patient's home. New portable hi-tech equipment. Hi-quality like standard office images, far superior to hospital portable images. No lead shielding needed for x-rays. University Radiology Service, (212) 928-5321 or (718) 339-5363.

CRAIG PERSONNEL AGENCY, INC., Medical placement specialists: medical office managers, secretaries, assistants and technicians; full-time, part-time, temp. NYC. (212) 302-8686, 25 West 43 Street, NY, NY.

ARE YOU HAVING A HEADACHE WITH THIRD PARTY REIMBURSEMENT? I offer my independent medical services in billing, negotiating and collecting. Coding and filing expertise to Medicaid, Medicare and all commercial carriers as well as patient collections. Long Island and New York practitioners please call (516) 944-9578.

Classified Advertising

New York State Journal of Medicine

GENERAL INFORMATION

All classified advertisements are payable in advance, except for government agencies and advertising agencies; a 15% commission to recognized advertising agencies.

Frequency: Monthly
Mailing date: 7th of the month
Closing date: 30 days before mailing date

Circulation: 27,000 (mainly physicians & medical libraries)

DISPLAY ADVERTISING

	1 time	*6 time*	*12 time*	*Width*		*Depth*
1/4-page	$540	$480	$420	3-1/2″	×	4-7/8″
1/6-page	252	228	204	2-1/8″	×	4-7/8″
1/8-page	228	204	180	3-1/2″	×	2-3/8″
1/16-page	114	102	90	3-1/2″	×	1-1/8″
1/18-page	84	72	60	2-1/8″	×	1-1/2″

PARAGRAPH ADVERTISING

The first several words are set in bold face caps.

Rates: $35.00 per insertion for 50 words or less; additional words are 40 cents each. On request, Dept. numbers will be assigned by the Journal for replies to be forwarded to the advertiser. Dept. numbers are 50 cents per insertion. There is no frequency discount for paragraph ads.

Counting Words: Two initials, each abbreviation, figures consisting of 5 numerals or less, are counted as separate words. For replies, your name and address, or telephone number should appear at the end of the ad and be counted in the total number of words.

For further information, please call the Advertising Department at 516-488-6100.

CLASSIFIED ADVERTISING ORDER FORM

New York State Journal of Medicine
Attn: Classified Advertising
420 Lakeville Rd., P.O. Box 5404
Lake Success, N.Y. 11042

Date____________________

Name____________________________________ Payment enclosed: $__________

Address__________________________________ Number of Insertions: __________

City/State/Zip____________________________ Assign Dept. Number: __________

Please make check payable to New York State Journal of Medicine.

THE MOUNT SINAI CENTER FOR FACIAL NERVE DISORDERS

A multi-disciplinary team of physicians and surgeons at The Mount Sinai Hospital for the complete evaluation and treatment of facial nerve disorders.

PROGRAM DIRECTORS

AVRIM R. EDEN, M.D.
CHIEF OF OTOLOGY
THE MOUNT SINAI MEDICAL CENTER

DAVID SIMPSON, M.D.
ASSISTANT PROFESSOR OF NEUROLOGY
CO-DIRECTOR, EMG LABORATORY

Address for patients and correspondence:

FACIAL NERVE CENTER
The Mount Sinai Hospital
19 East 98th Street
Suite 6A, Box 1191
New York, NY 10029

FOR PATIENT APPOINTMENTS OR INFORMATION:

(212) 650-2309

Obstetricians and Gynecologists, 600 Maryland Ave, SW, Suite 300 East, Washington, DC 20024-2588.

June 27–28. **9th Annual Poison Control Center Symposium on Management of Drug Overdose and Poisoning.** 14 Cat 1 Credits. San Francisco. Contact Continuing Education S/P, Room S-926, University of California, San Francisco, CA 94143-0446. Tel: (415) 476-4194.

COLORADO

June 22–26. **The Physician in Management Seminar I and Seminar II.** 31 Cat 1 Credits. The Antlers Hotel, Colorado Springs. Contact: Sherry Mason, American Academy of Medical Directors, 4830 W Kennedy Blvd, Suite 648, Tampa, FL 33609. Tel: (813) 873-2000.

IDAHO

June 28–July 5. **Effective Management of Common Sports Injuries.** Middle Fork of the Salmon River. Contact: Kathy Rairigh, Epic Expeditions, PO Box 209, Sun Valley, ID 83353. Tel: (208) 788-4995.

ILLINOIS

June 7–13. **Doppler and 2-D Echocardiography.** 39 Cat 1 Credits. Chicago. Contact: Lisa Krehbiel, Institute for Medical Studies, 30131 Town Center Drive, Suite 215, Laguna Niguel, CA 92677. Tel: (714) 495-4499.

MASSACHUSETTS

June 19–20. **Upper Extremity Disorders in Children.** Cambridge. Contact: American Society for Surgery of the Hand, 3025 South Parker Rd, Suite 65, Aurora, CO 80014.

MISSOURI

June 29–July 2. **Advanced Symposium on the Plastic Surgery of the Aging Face.** 35 Cat 1 Credits. Adam's Mark Hotel, St Louis. Contact: American Academy of Facial Plastic and Reconstructive Surgery, 1101 Vermont Ave, NW, Suite 404, Washington, DC 20005.

PENNSYLVANIA

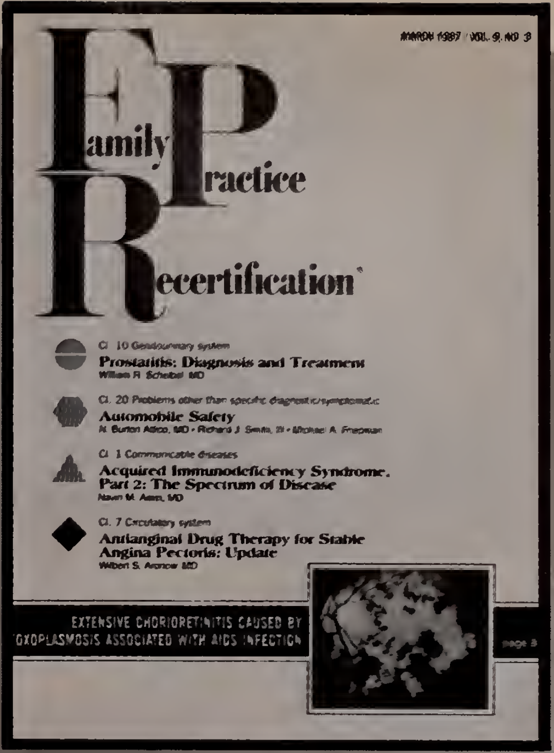
June 4–6. **Plastic Surgery Educational Foundation: Choices and Aesthetics.** 18 Cat 1 Credits. Vista International Hotel, Pittsburgh. Contact: PSEF, 233 North Michigan Ave, Suite 1900, Chicago, IL 60601.

June 10–12. **Current Concepts in Oncology and Hematologic Malignancies.** 17½ Cat 1 Credits. Holiday Inn Center City, Philadelphia. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

June 29–July 1. **Pharmacology, Biochemistry and Pathophysiology of Leukotrienes.** Wyndham Franklin Plaza Hotel, Philadelphia. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd Street, New York, NY 10021.

The complete journal for family practice physicians

- Reaches 79,000 family physicians monthly
- Presents the most commonly seen patient problems in family practice
- Written by physicians for physicians
- The most current clinical updates in:
  - Cardiology
  - Diabetes
  - Pediatrics
  - Ob/Gyn
  - Psychiatry
  - Gastroenterology
- Provides 20 hours of CME Category 1 Credit


MARCH 1987 VOL. 9, NO 3
Family Practice Recertification
Cl. 10 Genitourinary system
Prostatitis: Diagnosis and Treatment
Cl. 20 Problems other than specific diagnostic/symptomatic
Automobile Safety
Cl. 1 Communicable diseases
Acquired Immunodeficiency Syndrome, Part 2: The Spectrum of Disease
Cl. 7 Circulatory system
Antianginal Drug Therapy for Stable Angina Pectoris: Update
EXTENSIVE CHORIORETINITIS CAUSED BY TOXOPLASMOSIS ASSOCIATED WITH AIDS INFECTION
page 5

PRACTICAL ▪ CLINICAL ▪ EDUCATIONAL ▪ CURRENT

SOUTH CAROLINA

June 1–5. **13th Annual Family Medicine Update.** 29 Cat 1 Credits. Omni Hotel, Charleston. *June 8–13.* **Intensive Review in Family Medicine.** 45 Cat 1 Credits. Wild Dunes, Charleston. Contact: Dawne Ryals, Ryals and Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

TEXAS

June 17–20. **Menopause: Consequences and Treatment Strategies.** South Padre Island Hilton Resort, South Padre Island. Contact: Office of Continuing Medical Education, Scott and White, Temple, TX 76508. Tel: (817) 774-4073.

VIRGINIA

June 3–7. **The 11th Annual Postgraduate Course on Rehabilitation of the Brain-Injured Adult and Child.** The Williamsburg Hilton, Williamsburg. Contact: Kathy Martin, Office of Continuing Medical Education, Medical College of Virginia, Box 48, MCV Station, Richmond, VA 23298-0001. Tel: (804) 786-0494.

WASHINGTON

June 25–27. **Second National Conference on Cancer Prevention and Detection.** 15 Cat 1 Credits. Westin Hotel, Seattle. Info: American Cancer Society, Second National Conference on Cancer Prevention and Detection, 90 Park Ave, New York, NY 10016.

AROUND THE WORLD

CANADA

June 4–6. **Basic Ultrasound.** Chateau Frontenac, Quebec City, Quebec. Contact: The American College of Obstetricians and Gynecologists, 600 Maryland Ave, SW, Suite 300 East, Washington, DC 20024-2588.

June 12–13. **Toronto Neurology Update 1987.** 14 Cat 1 Credits. L'Hotel, Toronto. Contact: Organising Secretary, Sunnybrook Hospital, University of Toronto Clinic, 2075 Bayview Ave, Toronto, Ontario M4N 3M5. Tel: 480-4468.

June 15–19. **Advances in Internal Medicine.** 27 Cat 1 Credits. Banff Centre, Banff, Alberta. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

FINLAND/USSR

June 20–July 6. **Advanced Imaging Tutorials in Bone and Chest Diagnosis, Including MRI, CT, Arthrography, Absorptiometry, and the Plain Film.** 26 Cat 1 Credits. Helsinki, Finland and Leningrad, Suzdal, Moscow, USSR. Contact: Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

FRANCE

June 1–8. **Advances in Anesthesiology.** Paris. Contact: George Silvay, MD, PhD, Department of Anesthesiology, Mount Sinai Medical Center, One Gustav L. Levy Place, New York, NY 10029. Tel: (212) 650-5954.

We Make Money
For People
Who Make Money.

We're Bankers Trust's Private Clients Group. All the resources for customized money management assembled in one place. Professionals in portfolio management, sophisticated lending, forward planning and forty other financial services, at your command to go to work to get your money to work. Let us show you how we put it all together and how you can benefit from our unique capabilities.

For information and our "Guide to Services," write or call: Robert DiNapoli, Vice President, Private Clients Group, 280 Park Avenue, New York, N.Y. 10017. Tel. 212/850-3169.

Bankers Trust Company
Private Clients Group
We Make Money For People Who Make Money

© 1987 Bankers Trust Company
Member FDIC

Increase your interest in Israel
Ampal now pays 8%-10%

Invest in fixed income, variable rate or zero coupon securities and profit while you strengthen Israel's economy.

For 45 years, a driving financial force, Ampal-American Israel Corporation enables Israel to grow productively.

For further information and a prospectus, call:
Michael Lukin
Representative, Ampal Securities Corporation
26 Vanderbilt Way, No. Woodmere, NY 11581
(718) 258-6719 or 1-800 556-8766, Operator 456

This is neither an offer to sell nor a solicitation of an offer to buy securities. The offer is made only by the Prospectus which may be obtained in any state wherein the underwriter may lawfully offer the securities

PROVIDING A FOUNDATION ON WHICH ISRAEL BUILDS
AMPAL

This space contributed as a public service.

CANCER. IT'S SIMPLY NOT WHAT IT USED TO BE.

Over the last 40 years, research programs supported by the American Cancer Society have made increasing progress in the treatment, detection and prevention of cancer.

In 1986 alone, the Society funded over 700 projects conducted by the most distinguished scientists and research institutions in the country.

Which is why, this year, hundreds of thousands of people will be successfully treated for the disease.

We are winning.

But we need you to help keep it that way.

AMERICAN CANCER SOCIETY®
Help us keep winning.

Created as a public service by
Ally Gargano/MCA Advertising LTD.

right on TARGET

Consider a few features of **The Western Journal of Medicine:**

- **Epitomes of Progress** — Each month this section highlights the major advances in a different specialty with 15 or 20 brief epitomes by experts in the field.
- **Clinical Investigation** — Five major clinical research societies coordinate important new research findings, emphasizing clinical value.
- **Socioeconomics** — WJM is often the first to predict, examine and evaluate socioeconomic changes, trends.
- **Yearly Special Issue** — Each December's issue is devoted to a topic vital to physicians: clinical nutrition (1979), cost of care (1980), geriatrics (1981), occupational/environmental (1982), cross-cultural medicine (1983), personal health maintenance (1984), high-tech medicine (1985), medical informatics (1986).

NYSJM 5-87

Enclosed is $________ to cover ________ subscriptions.
Subscription renewal ☐ Regular subscription ☐
Foreign subscription ☐

TO: ______________________________

Street ______________________________

City/State/Zip ______________________________

Specialty ______________________________

Gift Card to read "From ______________________________"

The Western Journal of Medicine

Please mail this coupon with
enclosed payment to:

Circulation Department
P.O. Box 7602, San Francisco,
California 94120-7602

Subscriptions, 12 issues Per Year
U.S. and Canada $30 1 year,
$50 2 years

All other countries $50 1 year,
$90 2 years

MONDAY TO FRIDAY
OCTOBER 5-9, 1987

FIVE DAY EMERGENCY MEDICINE BOARD REVIEW COURSE

NYU Medical Center
Post-Graduate Medical School

Bellevue Hospital Center
Emergency Services

An intensive, five-day course providing:
- a concentrated review and update of Emergency Medicine topics
- analysis of relevant articles from the past six years of Emergency Medicine literature
- interaction regarding clinical controversies in management techniques
- sessions in written test-taking, and oral problem solving in ED medicine
- group sessions to assist participants with Oral Board preparation
- review of sample multiple choice questions with emphasis on test taking techniques
- special opportunity for 1 on 1 sessions with Board certified faculty to be provided on an ongoing basis throughout the course

TUITION: $600
Accreditation: 36.5 AMA Category I credit hours

For further information: NYU Post-Graduate Medical School, 550 First Avenue, New York, N.Y. 10016 (212) 340-5295 (24-hour service)

NYSJ5/87

HOUSE PHYSICIAN

Full time immediate, New York State License or Limited Permit. For Limited Permit foreign graduate must have completed 3 year approved residency U.S., United States Graduate 1 year approved residency U.S.

SHEEHAN MEMORIAL HOSPITAL
425 Michigan Ave., Buffalo, NY 14203 (716) 842-2200 Ext. 115

"SELLING PRACTICES IS OUR BUSINESS"

Want to maximize the return on your investment? Before you buy or sell, call for an appraisal to assure the best financial and transfer terms. We are a full service practice broker, not simply a listing service. Since 1981, Countrywide has been instrumental in helping hundreds of doctors like yourself buy and sell their practices. We guide you through the entire sales process from initial meeting to closing. Our offices serve New York, New Jersey and New England.

Countrywide Business Brokerage, Inc.
319 East 24th Street, Suite 23-G, New York, NY 10010
(212) 686-7902 (203) 868-3666 (617) 232-1075

MEDLINE:

Online Computer Search
Training for Health Professionals

One-day session to be held at:

THE NEW YORK ACADEMY OF MEDICINE
NEW YORK CITY

JUNE 16, 1987

For information please call:

Patricia Kennedy at (212) 876-8763

Doctor . .

Do you, or a physician you know, have a problem with alcohol, drugs, or emotional illness?

If so, please contact the Medical Society of the State of New York's Committee for Physicians' Health, whose function is to assist, *confidentially.*

Telephone: 516-488-7777.

Index to Advertisers

Ampal Securities Corporation	20
Ayerst Laboratories	4A, 5A, 6A, 23A, 24A, 25A, 26
Bankers Trust Company	20
St. Clare's Hospital	7
Classified Advertising	10A, 16A, 20A, 22
Countrywide Business Brokers	22
Family Practice Recertification Magazine	19
Mary Ann Hamburger Associates	14
Knoll Pharmaceuticals	15A, 16
Eli Lilly and Company	12
Marion Laboratories	1A, 2
Mt. Sinai Center	18
NYU Medical Center	22
Roche Laboratories	8A, 9A, 3rd & 4th Cove
Upjohn Company	2nd Cove

To show you how many hypertensives stayed on

INDERAL® LA

(PROPRANOLOL HCl)

after a major nationwide trial...

...we had to find just the right room.

60,073 patients (90%) who started on INDERAL® LA stayed on INDERAL LA.[1*]

Surprising? Not really.

Because most patients on INDERAL LA (propranolol HCl) don't even know it's working.

A recent double-blind, placebo-controlled, crossover study in 138 hypertensive patients[2] revealed that INDERAL LA has a side effects profile unsurpassed by atenolol or metoprolol — which shows how well-tolerated once-daily INDERAL LA can be.

Sole therapy or concomitant therapy? Fifty-nine percent of the time, INDERAL LA stood on its own.

The patients in the nationwide compliance trial were no different from yours. Generally when the antihypertensive regimen is complicated, compliance may become a problem. So, the effectiveness of INDERAL LA as once-daily monotherapy is a big plus. Of the remaining hypertensives in the program, 36% were treated merely with the addition of a diuretic to INDERAL LA.

For the noncompliant patients in your practice, INDERAL LA may well be the answer.

Almost 20,000 of the patients in the nationwide compliance trial were identified as having been noncompliant with their previous antihypertensive therapy. Their physicians reported that 88% showed improved compliance when placed on once-daily INDERAL LA.

Control, comfort, and compliance

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl) LONG ACTING CAPSULES

Like conventional INDERAL Tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree, and bronchial asthma.

*After a 30-day trial with INDERAL LA, physicians reported that 90% of the patients would remain on INDERAL LA.

The one you know best keeps looking better

NOW AVAILABLE...

NEW LOW DOSE

INDERAL LA 60 mg

(PROPRANOLOL HCl) LONG ACTING CAPSULES

Please see next page for brief summary of prescribing information

The one you know best keeps looking better

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. INDERAL LA is formulated to provide a sustained release of propranolol hydrochloride. INDERAL LA is available as 60 mg, 80 mg, 120 mg, and 160 mg capsules.

CLINICAL PHARMACOLOGY. INDERAL is a nonselective, beta-adrenergic receptor-blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor-stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately.

INDERAL LA Capsules (60, 80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate. Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours. When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL Tablets. The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially.

INDERAL LA should not be considered a simple mg-for-mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period.

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension; it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies.

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris.

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache. The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use.

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope. INDERAL LA also improves exercise performance. The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary.

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first-degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL.

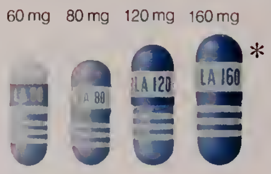
WARNINGS. CARDIAC FAILURE: Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl)

LONG ACTING CAPSULES



IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible).

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following *abrupt* discontinuance of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned, the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

MAJOR SURGERY: The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, eg, dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA: Beta blockers should be used with caution in diabetic patients if a beta-blocking agent is required. Beta blockers may mask tachycardia occurring with hypoglycemia, but other manifestations such as dizziness and sweating may not be significantly affected. Following insulin-induced hypoglycemia, propranolol may cause a delay in the recovery of blood glucose to normal levels.

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol may change thyroid function tests, increasing T_4 and reverse T_3, and decreasing T_3.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case, this resulted after an initial dose of 5 mg propranolol.

PRECAUTIONS. GENERAL: Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies.

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

CLINICAL LABORATORY TESTS: Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Caution should be exercised when patients receiving a beta blocker are administered a calcium-channel-blocking drug, especially intravenous verapamil, for both agents may depress myocardial contractility or atrioventricular conduction. On rare occasions, the concomitant intravenous use of a beta blocker and verapamil has resulted in serious adverse reactions, especially in patients with severe cardiomyopathy, congestive heart failure or recent myocardial infarction.

Aluminum hydroxide gel greatly reduces intestinal absorption of propranolol.

Ethanol slows the rate of absorption of propranolol.

Phenytoin, phenobarbitone, and *rifampin* accelerate propranolol clearance.

Chlorpromazine, when used concomitantly with propranolol, results in increased plasma levels of both drugs.

Antipyrine and *lidocaine* have reduced clearance when used concomitantly with propranolol.

Thyroxine may result in a lower than expected T_3 concentration when used concomitantly with propranolol.

Cimetidine decreases the hepatic metabolism of propranolol, delaying elimination and increasing blood levels.

Theophylline clearance is reduced when used concomitantly with propranolol.

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY: Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug.

PREGNANCY: Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose.

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

NURSING MOTHERS: INDERAL is excreted in human milk. Caution should be exercised when INDERAL (propranolol HCl) is administered to a nursing woman.

PEDIATRIC USE: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy.

Cardiovascular: Bradycardia; congestive heart failure; intensification of AV block; hypotension; paresthesia of hands; thrombocytopenic purpura; arterial insufficiency, usually of the Raynaud type.

Central Nervous System: Light-headedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia; visual disturbances; hallucinations; vivid dreams; an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics. For immediate formulations, fatigue, lethargy, and vivid dreams appear dose related.

Gastrointestinal: Nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

Allergic: Pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress.

Respiratory: Bronchospasm.

Hematologic: Agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura.

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported.

Miscellaneous: Alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily. If patients are switched from INDERAL Tablets to INDERAL LA Capsules, care should be taken to assure that the desired therapeutic effect is maintained. INDERAL LA should not be considered a simple mg-for-mg substitute for INDERAL. INDERAL LA has different kinetics and produces lower blood levels. Retitration may be necessary, especially to maintain effectiveness at the end of the 24-hour dosing interval.

HYPERTENSION—*Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic. The dosage may be increased to 120 mg once daily or higher until adequate blood-pressure control is achieved. The usual maintenance dosage is 120 to 160 mg once daily. In some instances a dosage of 640 mg may be required. The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks.

ANGINA PECTORIS—*Dosage must be individualized.* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three- to seven-day intervals until optimal response is obtained. Although individual patients may respond at any dosage level, the average optimal dosage appears to be 160 mg once daily. In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established.

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS).

MIGRAINE—*Dosage must be individualized.* The initial oral dose is 80 mg INDERAL LA once daily. The usual effective dose range is 160-240 mg once daily. The dosage may be increased gradually to achieve optimal migraine prophylaxis. If a satisfactory response is not obtained within four to six weeks after reaching the maximal dose, INDERAL LA therapy should be discontinued. It may be advisable to withdraw the drug gradually over a period of several weeks.

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily.

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use.

*The appearance of these capsules is a registered trademark of Ayerst Laboratories.

REFERENCES:

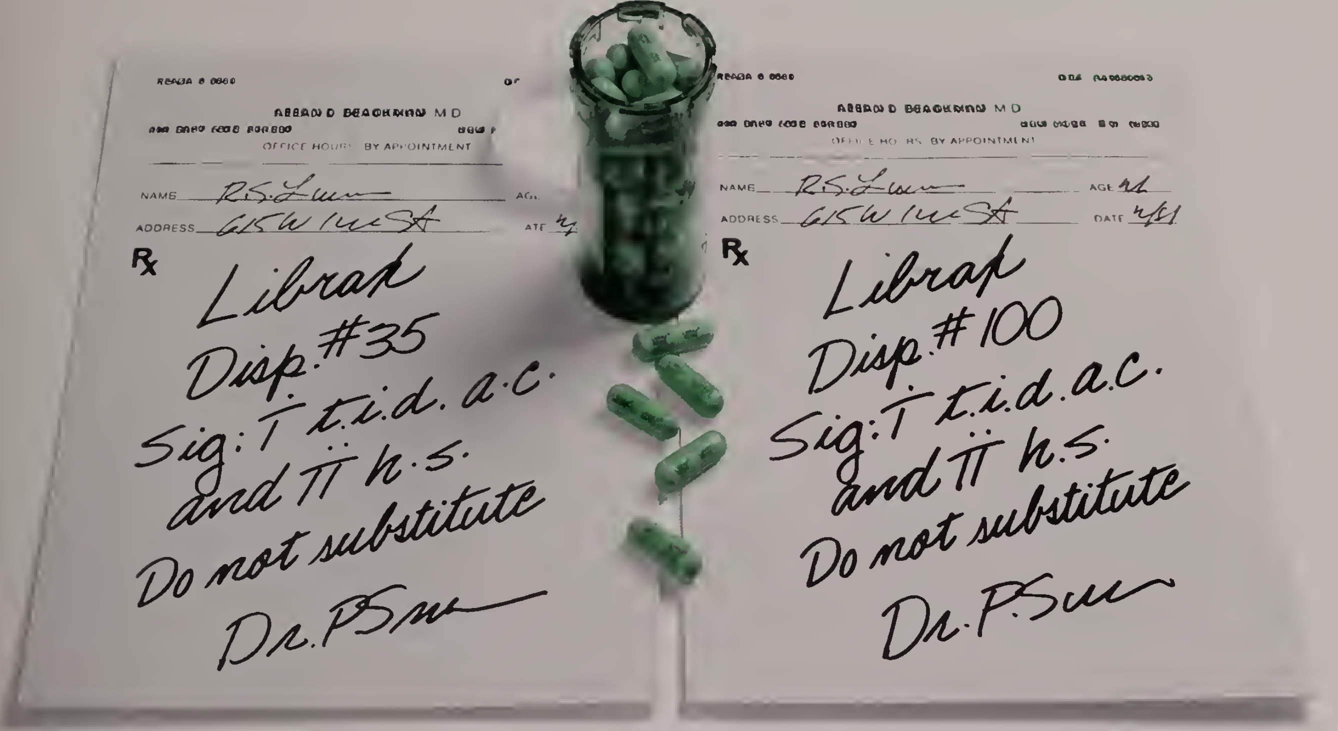
1. INDERAL LA National Compliance Evaluation Program. Data on file, Ayerst Laboratories.
2. Ravid M, Lang R, Jutrin I: The relative antihypertensive potency of propranolol, oxprenolol, atenolol, and metoprolol given once daily. *Arch Intern Med* 1985; 145:1321-1323.

R7119/587

Ayerst®
AYERST LABORATORIES
New York, NY 10017

© 1987 Ayerst Laboratories

SPECIFY LIBRAX®



OFFICE HOURS BY APPOINTMENT

NAME R.S. L

ADDRESS 615 W 1 St

℞

Librax

Disp. #35

Sig: i t.i.d. a.c.

and ii h.s.

Do not substitute

Dr. P.S

OFFICE HOURS BY APPOINTMENT

NAME R.S. L

ADDRESS 615 W 1 St

℞

Librax

Disp. #100

Sig: i t.i.d. a.c.

and ii h.s.

Do not substitute

Dr. P.S

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide.

Please consult complete prescribing information, a summary of which follows:

* Indications: Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:
"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl/Roche) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.

As with all anticholinergics, inhibition of lactation may occur.

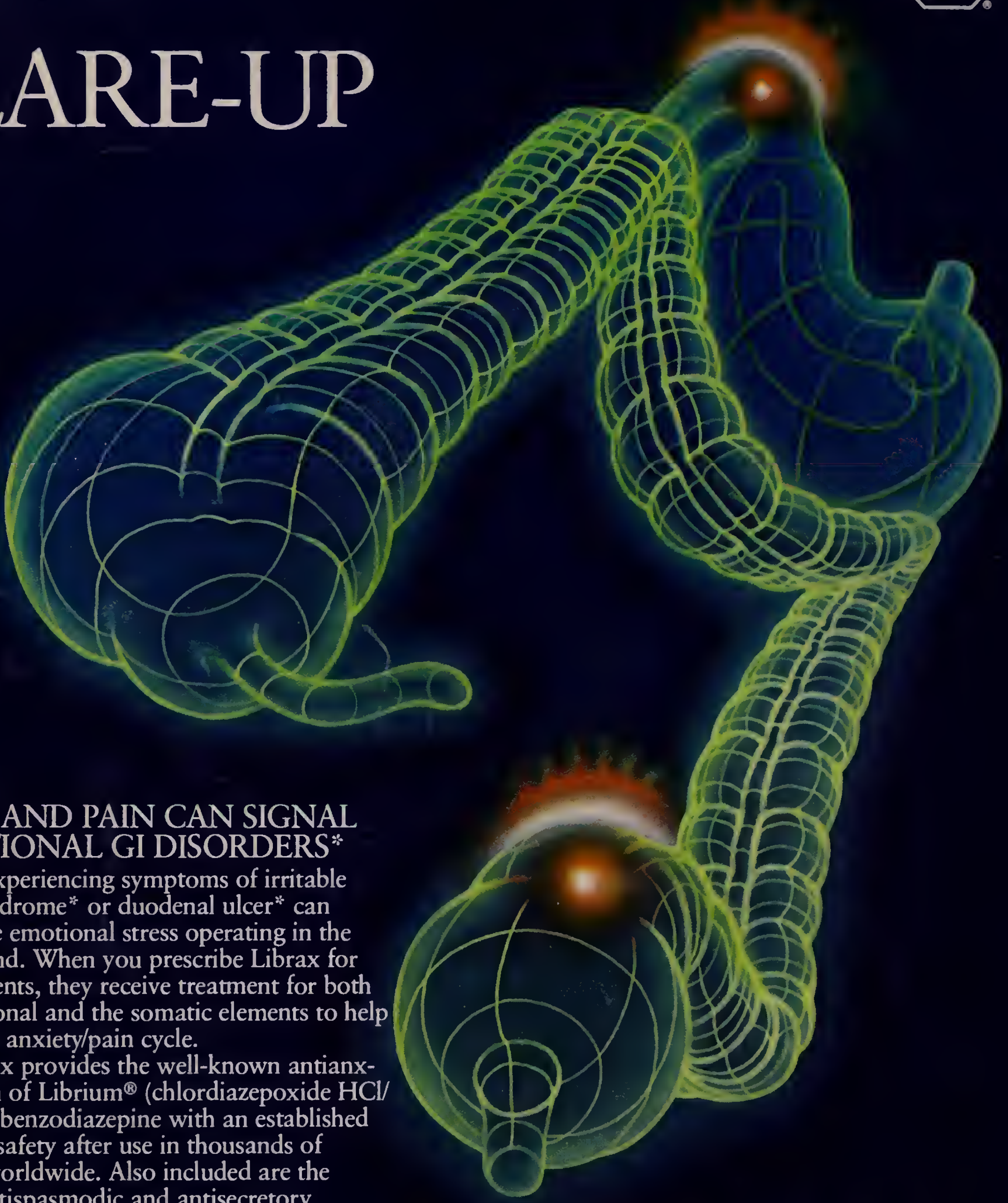
Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

ROCHE®
Roche Products Inc.
Manati, Puerto Rico 00701

ROCHE®

FLARE-UP



SPASM AND PAIN CAN SIGNAL FUNCTIONAL GI DISORDERS*

Patients experiencing symptoms of irritable bowel syndrome* or duodenal ulcer* can often have emotional stress operating in the background. When you prescribe Librax for these patients, they receive treatment for both the emotional and the somatic elements to help relieve the anxiety/pain cycle.

Librax provides the well-known antianxiety action of Librium® (chlordiazepoxide HCl/Roche), a benzodiazepine with an established record of safety after use in thousands of patients worldwide. Also included are the proven antispasmodic and antisecretory actions of Quarzan® (clidinium bromide/Roche), the component which helps to reduce colonic spasm and hypersecretion and helps also to alleviate the pain they cause.

LIBRAX: FOR THE DUAL PROBLEMS OF FUNCTIONAL GI DISORDERS.

SPECIFY ADJUNCTIVE

LIBRAX®

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide.

ANTIANXIETY
ANTISECRETORY
ANTISPASMODIC

---

*Librax has been evaluated as possibly effective as adjunctive therapy in the treatment of duodenal ulcer and the irritable bowel syndrome.

Copyright © 1983 by Roche Products Inc. All rights reserved.

Please see summary of prescribing information on following page.

STACKS

NEW YORK STATE JOURNAL OF MEDICINE

JUNE 1987 • Volume 87, Number 6

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA
JUN 17 1987
M.D.S.

Contents

COMMENTARIES

Preventing bicycle-related head injuries 319
BARRY D. WEISS, MD

Diagnostic approaches to Pancoast's syndrome 320
PETER E. KRUMPE, MD

Amiodarone: Therapeutic but toxic 322
FRANCIS E. MARCHLINSKI, MD

The status of amiodarone 324
RICHARD N. FOGOROS, MD

RESEARCH PAPERS

The role of fiberoptic bronchoscopy and transbronchial biopsy in the diagnosis of Pancoast's tumor 326
ROGER A. MAXFIELD, MD, CONRADO P. ARANDA, MD

Oral ciprofloxacin in the treatment of 14 patients with bacterial pneumonia 330
CHRISTINE M. WOLLSCHLAGER, MD; SUHAIL RAOOF, MB; FAROQUE A. KHAN, MB

HISTORY

Arthur Donaldson Smith, MD: Physician, explorer, naturalist, and diplomat—Part IV 334
PASCAL JAMES IMPERATO, MD

ESSAYS IN PEDIATRICS

Etiology of infectious mononucleosis: Solving the riddle 352
CARL POCHEDLY, MD

SPECIAL ARTICLE

Death of a dentist 356
NAOMI R. BLUESTONE, MD, MPH

CASE REPORTS

Serum creatinine increase associated with amiodarone therapy 358
MICHAEL B. JACOBS, MD

Fatal *Branhamella catarrhalis* pneumonia 359
LORRAINE COLI, MD; SUNNY WONG, MD; WILLIAM SCHRAFT, MD; FRANK E. IAQUINTA, MD; ROBERT D. BRANDSTETTER, MD

Bilateral pregnancy in a uterus didelphys 361
STEPHEN W. GIORLANDO, MD, FAKHRY Y. ALEXANDER, MD

The neuroleptic malignant syndrome after open heart surgery: Successful treatment with bromocriptine 362
ABRAHAM LIEBERMAN, MD; PETER PASTERNACK, MD; STEPHEN COLVIN, MD

LETTERS TO THE EDITOR

Patient-physician relationship 364
NORMAN S. BLACKMAN, MD

Medicaid billing 365
DONALD R. MOY, JD

Carbon dioxide laser surgery of female genital, perineal, and anal condyloma acuminata 365
HALUK CAGLAR, MD, MEHMET ERK, MD, MYROSLAW M. HRESHCHYSHYN, MD

Diarrhea caused by group G streptococcus 366
GREGORY BARD, MD; ERNEST B. VISCONTI, MD; JOHN P. CURRAN, MD; PASCAL J. DE CAPRARIIS, MD

BOOK REVIEWS 368
NEWS BRIEFS 371
MEDICAL SOCIETY NEWS 373
OBITUARIES 374
MEDICAL MEETINGS AND LECTURES 3A

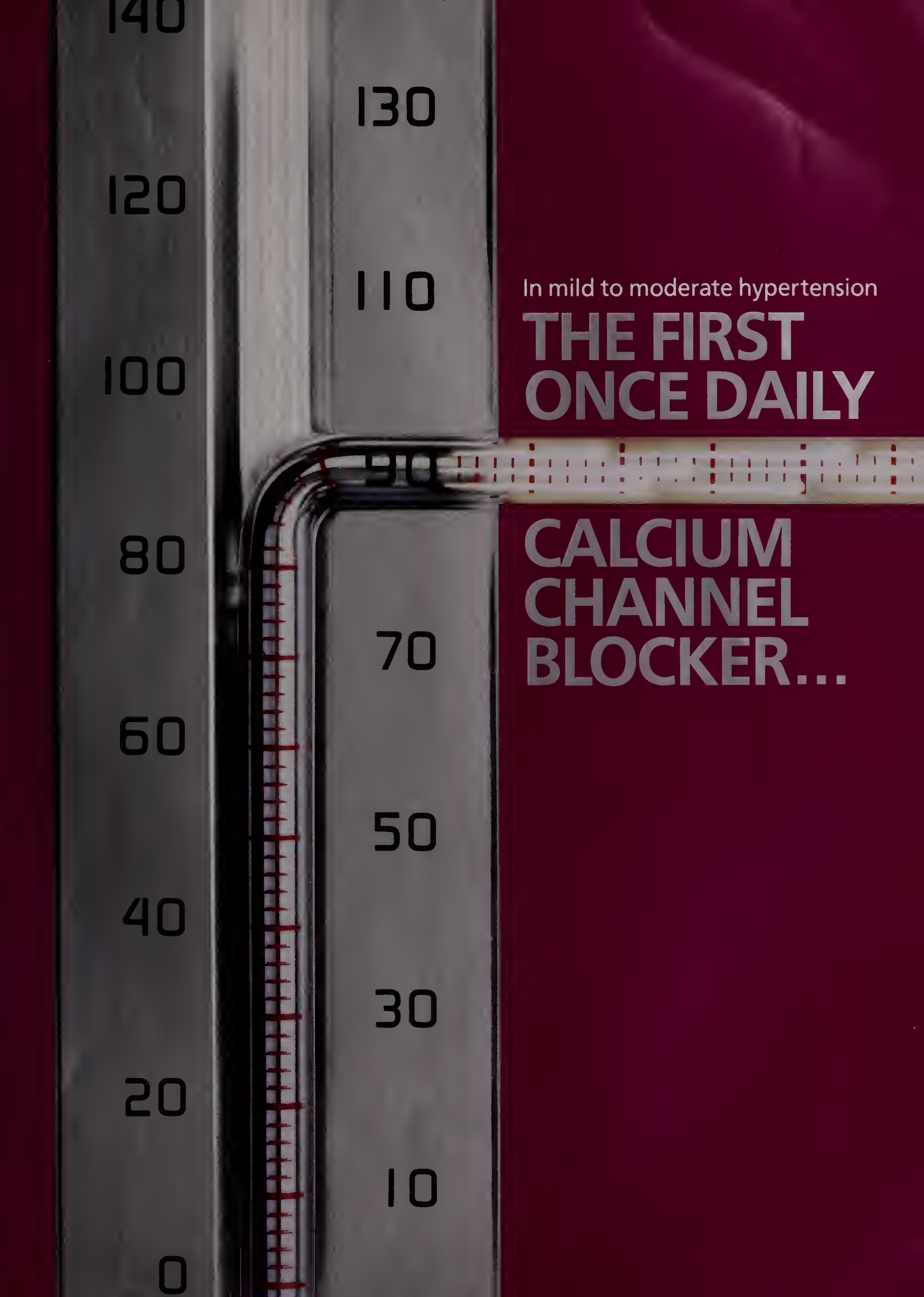
Motrin® 800 TABLETS mg
ibuprofen

Extra strength
Convenience
Economy

Upjohn
A Century
of Caring
1886·1986

©1986 The Upjohn Company

J-6138 January 1986


140
130
120
110
100
90
80
70
60
50
40
30
20
10
0
In mild to moderate hypertension
THE FIRST ONCE DAILY
CALCIUM CHANNEL BLOCKER...

NEW
ONCE DAILY

ISOPTIN SR*

(verapamil HCl/Knoll)

240 mg scored, sustained-release tablets

JAMES B.
38, black male, heavy smoker. Prescribed a diuretic by another physician last year for hypertension.

YOUR CONCERNS
Presents with "smoker's cough." Workup reveals a BP of 150/107.

A LOGICAL CHOICE FOR CONTROL OF HIS BP
ISOPTIN® (verapamil HCl/Knoll) because...

- Black hypertensives often have low plasma renin activity and generally do not respond favorably to beta blockers.
- Beta blockers may increase the likelihood of bronchospasm.

ALICE W.
65, diabetic, overweight. Her BP has elevated to 190/98.

YOUR CONCERNS
She's on daily insulin.

A LOGICAL CHOICE FOR CONTROL OF HER BP
ISOPTIN® (verapamil HCl/Knoll) because...

- Unlike most beta blockers and diuretics, ISOPTIN has no adverse effects on serum glucose levels.
- Unlike most beta blockers, ISOPTIN does not mask the symptoms of hypoglycemia.

THOMAS G.
70, asthmatic. In the past, BP adequately controlled with 25 mg hydrochlorothiazide daily.

YOUR CONCERNS
Today patient presents with symptoms of gout. Workup reveals high uric acid level, low serum potassium, and BP elevated to 180/98.

A LOGICAL CHOICE FOR CONTROL OF HIS BP
ISOPTIN® (verapamil HCl/Knoll) because...

- Unlike diuretics, ISOPTIN will not decrease serum potassium levels or elevate uric acid levels.
- Unlike beta blockers, ISOPTIN can be used safely in asthma and COPD patients.

JOHN K.
42, Annual physical uncovered diastolic BP of 102... confirmed on three successive office visits. Unresponsive to nonpharmacologic intervention.

YOUR CONCERNS
Salesman, spends many hours of his working day in car... total cholesterol level 300, HDL 35.

A LOGICAL CHOICE FOR CONTROL OF HIS BP
ISOPTIN® (verapamil HCl/Knoll) because...

- Unlike diuretics, ISOPTIN does not cause urinary urgency.
- Unlike either beta blockers or diuretics, ISOPTIN will not adversely affect his already seriously compromised lipid profile.
- Unlike with propranolol, fatigue and impotence are rarely reported.

Antihypertensive therapy you and your patients can live with

*A product of Knoll research.

© 1986, BASF K & F Corporation

2538/2-87

Knoll Pharmaceuticals
A Unit of BASF K&F Corporation
Whippany, New Jersey 07981

BASF Group

Knoll®

Printed in U.S.A.
Please see adjacent page for brief summary.

In mild to moderate hypertension

Brief Summary

THE FIRST ONCE DAILY CALCIUM CHANNEL BLOCKER

ISOPTIN® SR
(verapamil HCl/Knoll)
240 mg scored, sustained-release tablets

CONTRAINDICATIONS: 1) Severe left ventricular dysfunction (see WARNINGS), 2) Hypotension (less than 90 mmHg systolic pressure) or cardiogenic shock, 3) Sick sinus syndrome or 2nd or 3rd degree AV block (except in patients with a functioning artificial ventricular pacemaker).

WARNINGS: Heart Failure: ISOPTIN should be avoided in patients with severe left ventricular dysfunction (see DRUG INTERACTIONS). Patients with milder ventricular dysfunction should, if possible, be controlled before verapamil treatment. Hypotension: ISOPTIN (verapamil HCl) may produce occasional symptomatic hypotension. Elevated Liver Enzymes: Elevations of transaminases with and without concomitant elevations in alkaline phosphatase and bilirubin have been reported. Periodic monitoring of liver function in patients receiving verapamil is therefore prudent. Accessory Bypass Tract (Wolff-Parkinson-White): Patients with paroxysmal and/or chronic atrial flutter or atrial fibrillation and a coexisting accessory AV pathway have developed increased antegrade conduction across the accessory pathway producing a very rapid ventricular response or ventricular fibrillation after receiving intravenous verapamil. While this has not been reported with oral verapamil, it should be considered a potential risk. Treatment is usually D.C.-cardioversion. Atrioventricular Block: The effect of verapamil on AV conduction and the SA node may cause asymptomatic 1st degree AV block and transient bradycardia. Higher degrees of AV block, while infrequent (0.8%), may require a reduction in dosage or, in rare instances, discontinuation of verapamil HCl. Patients with Hypertrophic Cardiomyopathy (IHSS): Although verapamil has been used in the therapy of patients with IHSS, severe cardiovascular decompensation and death have been noted in this patient population.

PRECAUTIONS: Impaired Hepatic or Renal Function: Verapamil is highly metabolized by the liver with about 70% of an administered dose excreted in the urine. In patients with impaired hepatic or renal function verapamil should be administered cautiously and the patients monitored for abnormal prolongation of the PR interval or other signs of excessive pharmacological effects (see OVERDOSAGE).

Drug Interactions: Beta Blockers: Concomitant use of ISOPTIN and oral beta-adrenergic blocking agents may be beneficial in certain patients with chronic stable angina or hypertension, but available information is not sufficient to predict with confidence the effects of concurrent treatment in patients with left ventricular dysfunction or cardiac conduction abnormalities. Digitalis: Clinical use of verapamil in digitalized patients has shown the combination to be well tolerated if digoxin doses are properly adjusted. However, chronic verapamil treatment increases serum digoxin levels by 50 to 75% during the first week of therapy and this can result in digitalis toxicity. Upon discontinuation of ISOPTIN (verapamil HCl), the patient should be reassessed to avoid underdigitalization. Antihypertensive Agents: Verapamil administered concomitantly with oral antihypertensive agents (e.g., vasodilators, angiotensin-converting enzyme inhibitors, diuretics, beta blockers, prazosin) will usually have an additive effect on lowering blood pressure. Patients receiving these combinations should be appropriately monitored. Disopyramide: Disopyramide should not be administered within 48 hours before or 24 hours after verapamil administration. Quinidine: In patients with hypertrophic cardiomyopathy (IHSS), concomitant use of verapamil and quinidine resulted in significant hypotension. There has been a report of increased quinidine levels during verapamil therapy. Nitrates. The pharmacologic profile of verapamil and nitrates as well as clinical experience suggest beneficial interactions. Cimetidine: Two clinical trials have shown a lack of significant verapamil interaction with cimetidine. A third study showed cimetidine reduced verapamil clearance and increased elimination to 1/2. Anesthetic Agents: Verapamil may potentiate the activity of neuromuscular blocking agents and inhalation anesthetics. Carbamazepine: Verapamil may increase carbamazepine concentrations during combined therapy. Rifampin: Therapy with rifampin may markedly reduce oral verapamil bioavailability. Lithium: Verapamil may lower lithium levels in patient on chronic oral lithium therapy. Carcinogenesis, Mutagenesis, Impairment of Fertility: There was no evidence of a carcinogenic potential of verapamil administered to rats for two years. Verapamil was not mutagenic in the Ames test. Studies in female rats did not show impaired fertility. Effects on male fertility have not been determined. Pregnancy (Category C): There are no adequate and well-controlled studies in pregnant women. ISOPTIN crosses the placental barrier and can be detected in umbilical vein blood at delivery. This drug should be used during pregnancy, labor, and delivery, only if clearly needed. Nursing Mothers: ISOPTIN is excreted in human milk, therefore, nursing should be discontinued while verapamil is administered. Pediatric Use: Safety and efficacy of ISOPTIN in children below the age of 18 years have not been established.

ADVERSE REACTIONS: Constipation 8.4%, dizziness 3.5%, nausea 2.7%, hypotension 2.5%, edema 2.1%, headache 1.9%, CHF/pulmonary edema 1.8%, fatigue 1.7%, bradycardia 1.4%, 3° AV block 0.8%, flushing 0.1%, elevated liver enzymes (see WARNINGS). The following reactions, reported in less than 1.0% of patients, occurred under conditions (open trials, marketing experience) where a causal relationship is uncertain; they are mentioned to alert the physician to a possible relationship: angina pectoris, arthralgia and rash, AV block, blurred vision, cerebrovascular accident, chest pain, claudication, confusion, diarrhea, dry mouth, dyspnea, ecchymosis or bruising, equilibrium disorders, exanthema, gastrointestinal distress, gingival hyperplasia, gynecomastia, hair loss, hyperkeratosis, impotence, increased urination, insomnia, macules, muscle cramps, myocardial infarction, palpitations, paresthesia, psychotic symptoms, purpura (vasculitis), shakiness, somnolence, spotty menstruation, sweating, syncope, urticaria. Treatment of Acute Cardiovascular Adverse Reactions: Whenever severe hypotension or complete AV block occur following oral administration of verapamil, the appropriate emergency measures should be applied immediately, e.g., intravenously administered isoproterenol HCl, levarterenol bitartrate, atropine (all in the usual doses), or calcium gluconate (10% solution). If further support is necessary, inotropic agents (dopamine or dobutamine) may be administered. Actual treatment and dosage should depend on the severity and the clinical situation and the judgment and experience of the treating physician.

OVERDOSAGE: Treatment of overdosage should be supportive. Beta-adrenergic stimulation or parenteral administration of calcium solutions may increase calcium ion flux across the slow channel, and have been used effectively in treatment of deliberate overdosage with verapamil. Clinically significant hypotensive reactions or fixed high degree AV block should be treated with vasopressor agents or cardiac pacing, respectively. Asystole should be handled by the usual measures including cardiopulmonary resuscitation.

Knoll Pharmaceuticals
A Unit of BASF K&F Corporation
Whippany, New Jersey 07981

knoll®

BASF Group

©1986, BASF K&F Corporation 2474/11/86 Printed in U.S.A.

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices unless the information is submitted at least three months prior to the event.*

JULY 1987

AROUND THE STATE

BUFFALO

July 16–18. **The Great Lakes Regional Conference on Infertility.** Buffalo Waterfront Hilton Hotel, Buffalo. Contact: Jordan M. Phillips, MD, General Chairman, AAGL, 13021 East Florence Ave, Santa Fe Springs, CA 90670. Tel: (213) 946-8774.

SARATOGA SPRINGS

July 9–10. **Update on Stroke.** Ramada Renaissance. Contact: Barbara G. Post, Program Coordinator, Regional Medical Education Program, Sunnyview Hospital and Rehabilitation Center, 1270 Belmont Ave, Schenectady, NY 12308. Tel: (518) 382-4500.

AROUND THE NATION

ALASKA

July 4–11. **Imaging of Musculoskeletal Trauma.** 18 Cat 1 Credits. Cruise the Inland Passage. Info: Alaska '87—Cruise the Inland Passage, 21915 Roscoe Blvd, Suite 222, Canoga Park, CA 91304. Tel: (818) 340-0580, ext 280.

CALIFORNIA

July 24–25. **Debates & Updates in Pediatric Emergencies.** 14 Cat 1 Credits. San Francisco. *July 28–August 8.* **Summer Seminar in Medical Ethics and Humanities.** San Francisco. Contact: School of Medicine, Extended Programs in Medical Education, University of California—San Francisco, San Francisco, CA 94143. Tel: (415) 476-4251.

COLORADO

July 9–11. **The Pituitary in Aspen.** The Given Institute of Pathobiology, Aspen. *July 12–17.* **Twenty-Third Annual In-**

(*continued on p 9A*)

NEW YORK STATE JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD	JOHN T. PRIOR, MD
ELIZA H. CALDWELL, MD	GITA S. SINGH*
JOSEPH F. MURATORE, MD	STANFORD WESSLER, MD

**Medical student*

Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Consulting Editor and Book Review Editor	RICHARD B. BIRRER, MD
Consulting Editor	NAOMI R. BLUESTONE, MD, MPH
Consulting Editor	CARL POCHEDLY, MD
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Managing Editor	CAROL L. MOORE
Advertising Director	ROBERT MILLER
Consulting Medical Writer	VICKI GLASER
Editorial Assistant	MILDRED J. AREMANN
Secretary	ELIZABETH J. SOMERS
Librarian	ELLA ABNEY
Assistant Librarian	ELEANOR BURNS

ASSOCIATE EDITORIAL BOARD 1987

MICHAEL E. BERLOW, MD	ROBERT LANDESMAN, MD
RANDALL BLOOMFIELD, MD	JAMES M. MORRISSEY, ESQ
ROBERT D. BRANDSTETTER, MD	STEPHEN NORDLICHT, MD
JOHN S. DAVIS, MD	JOSEPH SCHLUGER, MD
MYLES S. GOMBERT, MD	BJORN THORBJARNARSON, MD
ALFRED P. INGEGNO, MD	RODRIGO E. URIZAR, MD
FLORENCE KAVALER, MD	NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. United Media Associates, Inc, 16 Bruce Park Ave, Greenwich, CT 06830. Telephone: (203) 661-9702.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

SAMUEL M. GELFAND, MD,	*President*
JOHN A. FINKBEINER, MD,	*Past-President*
CHARLES D. SHERMAN, JR, MD,	*President-Elect*
JOSEPH F. SHANAPHY, MD,	*Vice-President*
JOHN H. CARTER, MD,	*Secretary*
GEORGE LIM, MD,	*Assistant Secretary*
MORTON KURTZ, MD,	*Treasurer*
ROBERT A. MAYERS, MD,	*Assistant Treasurer*
CHARLES N. ASWAD, MD,	*Speaker*
SEYMOUR R. STALL, MD,	*Vice-Speaker*

Councilors

Term Expires 1988

JAMES H. COSGRIFF, JR, MD,	Erie
RICHARD A. HUGHES, MD,	Warren
ANTONIO F. LASORTE, MD,	Broome
SIDNEY MISHKIN, MD,	Nassau

Term Expires 1989

ROBERT E. FEAR, MD,	Suffolk
STANLEY L. GROSSMAN, MD,	Orange
THOMAS D. PEMRICK, MD,	Rensselaer
RALPH E. SCHLOSSMAN, MD,	Queens

Term Expires 1990

DAVID M. BENFORD, MD,	New York
DUANE M. CADY, MD,	Onondaga
WILLIAM A. DOLAN, MD,	Monroe
ROBERT E. GORDON, MD,	Kings

Resident Councilor (representing the resident physician membership)

KATHLEEN E. SQUIRES, MD,	New York

Student Councilor (representing the medical student membership)

MICHAEL PACIOREK,	Onondaga

Trustees

RICHARD D. EBERLE, MD, (*Chairman*)	Onondaga
EDGAR P. BERRY, MD,	New York
JAMES M. FLANAGAN, MD,	Wayne
ALLISON B. LANDOLT, MD,	Westchester
DANIEL F. O'KEEFFE, MD,	Warren
BERNARD J. PISANI, MD,	New York
VICTOR J. TOFANY, MD,	Monroe

Executive Vice-President	DONALD F. FOY
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Vice-President Emeritus	GEORGE J. LAWRENCE, JR, MD
Director, Division of Policy Coordination	IRMA A. ERICKSON
Director, Division of Scientific Publications	PASCAL JAMES IMPERATO, MD
Director, Division of Communications	EDWARD A. HYNES
Director, Division of Computer Services	IVAN H. NAJMAN
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Governmental Affairs	GERARD L. CONWAY, ESQ
General Counsel	DONALD R. MOY, JD
Director, Division of Management and Meeting Services	ROBERT MILLER
Director, Division of Membership Support Services	EUNICE M. SKELLY
Director, Division of Scientific and Educational Activities	PETER B. FARNSWORTH, MD
Director, Division of Socio-Medical Economics	MARIO V. MENGHINI
Medical Director, Committee for Physicians' Health	BARTON A. HARRIS, MD
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York. Copyright © 1987, Medical Society of the State of New York. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.

Un-switch-ables

Patients and physicians alike prefer

Transderm-Nitro®
nitroglycerin

Over half a billion* patches prescribed in the U.S. since 1982

The only patch with a rate-limiting membrane

Familiar, distinctive tan color and unique shape recognized by patients everywhere

There's no substitute for experience

CIBA

(See Brief Summary of Prescribing Information on the next page.)

*Data on file, CIBA Pharmaceutical Co.

629-7750-A

Transderm-Nitro®
nitroglycerin
Transdermal Therapeutic System

BRIEF SUMMARY (FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE INSERT)

INDICATIONS AND USAGE
This drug product has been conditionally approved by the FDA for the prevention and treatment of angina pectoris due to coronary artery disease The conditional approval reflects a determination that the drug may be marketed while further investigation of its effectiveness is undertaken. A final evaluation of the effectiveness of the product will be announced by the FDA.

CONTRAINDICATIONS
Intolerance of organic nitrate drugs, marked anemia, increased intraocular pressure or increased intracranial pressure.

WARNINGS
In patients with acute myocardial infarction or congestive heart failure, Transderm-Nitro system should be used under careful clinical and/or hemodynamic monitoring
In terminating treatment of anginal patients, both the dosage and frequency of application must be gradually reduced over a period of 4 to 6 weeks to prevent sudden withdrawal reactions, which are characteristic of all vasodilators in the nitroglycerin class
Transdermal nitroglycerin systems should be removed before attempting defibrillation or cardioversion because of the potential for altered electrical conductivity which may enhance the possibility of arcing, a phenomenon associated with the use of defibrillators

PRECAUTIONS
Symptoms of hypotension, such as faintness, weakness or dizziness, particularly orthostatic hypotension may be due to overdosage. When these symptoms occur, the dosage should be reduced or use of the product discontinued
Transderm-Nitro system is not intended for immediate relief of anginal attacks. For this purpose occasional use of the sublingual preparations may be necessary

ADVERSE REACTIONS
Transient headaches are the most common side effect, especially when higher doses of the drug are used These headaches should be treated with mild analgesics while Transderm-Nitro therapy is continued When such headaches are unresponsive to treatment, the nitroglycerin dosage should be reduced or use of the product discontinued
Adverse reactions reported less frequently include hypotension, increased heart rate, faintness, flushing, dizziness, nausea and vomiting. These symptoms are attributable to the known pharmacologic effects of nitroglycerin, but may be symptoms of overdosage When they persist the dose should be reduced or use of the product discontinued In some patients, dermatitis may occur.

DOSAGE AND ADMINISTRATION
Therapy should be initiated with application of one Transderm-Nitro 5 system to the desired area of skin Many patients prefer the chest, if hair is likely to interfere with system adhesion or removal, it can be clipped prior to placement of the system Each system is designed to remain in place for 24 hours, and each successive application should be to a different skin area Transderm-Nitro system should not be applied to the distal parts of the extremities
The usual dosage is one Transderm-Nitro 5 system every 24 hours Some patients, however, may require the Transderm-Nitro 10 system If a single Transderm-Nitro 5 system fails to provide adequate clinical response, the patient should be instructed to remove it and apply either two Transderm-Nitro 5 systems or one Transderm-Nitro 10 system. More systems may be added as indicated by continued careful monitoring of clinical response The Transderm-Nitro 2.5 system is useful principally for decreasing the dosage gradually, though it may provide adequate therapy for some patients when used alone
The optimal dosage should be selected based upon the clinical response, side effects, and the effects of therapy upon blood pressure The greatest attainable decrease in resting blood pressure that is not associated with clinical symptoms of hypotension especially during orthostasis indicates the optimal dosage. To decrease adverse reactions, the size and/or number of systems should be tailored to the individual patient's needs
Do not store above 86°F (30°C).

PATIENT INSTRUCTIONS FOR APPLICATIONS
A patient leaflet is supplied with the systems.

HOW SUPPLIED

Transderm-Nitro System Rated Release *in vivo*	Total Nitroglycerin in System	System Size	Carton Size
2.5 mg/24 hr	12.5 mg	5 cm²	30 Systems (NDC 0083-2025-26) *100 Systems (NDC 0083-2025-30)
5 mg/24 hr	25 mg	10 cm²	30 Systems (NDC 0083-2105-26) *100 Systems (NDC 0083-2105-30)
10 mg/24 hr	50 mg	20 cm²	30 Systems (NDC 0083-2110-26) *100 Systems (NDC 0083-2110-30)
15 mg/24 hr	75 mg	30 cm²	30 Systems (NDC 0083-2115-26) *100 Systems (NDC 0083-2115-30)

*Hospital Pack 100's

C85-35 (Rev 11/85)

Dist. by:
CIBA Pharmaceutical Company
Division of CIBA-GEIGY Corporation
Summit, New Jersey 07901

All transdermal nitroglycerin products are being marketed pending final evaluation of effectiveness by the FDA.

© 1986, CIBA 629-7750-A

C I B A

Classified Advertising

PHYSICIANS WANTED

PEDIATRICIAN, Board eligible/certified, to work in a community health center in the Finger Lakes Region of New York State. The Center is an established program with a record of excellence. Associated with the University of Rochester, this can be an exciting opportunity. Send CV to Constantino Fernandez, MD, Acting Medical Director, Jordan Health Center, 82 Holland Street, P.O. Box 876, Rochester, NY 14603, EOE, M/F.

INTERNIST OR G.P. WANTED for partnership in old established Queens Practice. Yugoslavian preferred, but not a must. (718) 728-1244.

DO YOU WANT TO SHARE A THRIVING DERMATOLOGY PRACTICE: Do you enjoy year around outdoor recreation? You should contact us—a progressive multi-specialty group of 68 physicians in a pleasant, growing community. We offer a liberal financial package and outstanding benefits. Send curriculum vitae and references to: Michael T. Anderson, M.D., Willmar Medical Center, 101 Willmar Ave. SW, Willmar, MN 56201.

EMERGENCY MEDICINE POSITIONS—Full/part time emergency medicine physicians sought by multi state professional association for openings in metropolitan NY, PA, MD, DC, FL, New England and throughout U.S. Contact or send CV to Liberty Healthcare Corporation, 399 Market Street, Suite 400, Philadelphia, PA 19106, (215) 592-7400 or outside Pa (800) 331-7122.

ARIZONA-BASED PHYSICIAN RECRUITMENT firm has quality opportunities coast to coast. Available positions in most primary care and surgical specialties to include OB/GYN, Orthopedics, ER, and ENT. "Quality Physicians for Quality Clients since 1972." Call 602-990-8080; or send CV to: Mitchell & Associates, Inc., PO Box 1804, Scottsdale, AZ 85252.

PRIMARY CARE PRACTITIONERS (BC/BE) needed for unique practice situation, with ownership potential, in Long Island. Please reply with full particulars including telephone number to Dept. 443 c/o NYSJM.

POSITIONS WANTED

ROUTINE X-RAY DIAGNOSIS: Mature, U.S. trained, Board Certified Radiologist will have time available commencing summer of 1987, to participate in radiographic installation in New York City Metropolitan area. Not interested in large volume, salary commensurate. Private practice coverage would also be suitable. Reply Dept. 441 c/o NYSJM.

PHYSICAL MEDICINE AND REHABILITATION. Physiatrist seeks full or part time position. Long Island/New York City area. Board Eligible, American Medical Graduate, Chief Resident. Available July 1. (516) 794-9622.

PRACTICES AVAILABLE

FLORIDA FAMILY/MEDICAL PRACTICE—N.E. Fort Lauderdale. Physician retiring, established 20 years. High gross, desirable demographic patient profile. Fully equipped including x-ray. Top location, terms (305) 946-2472 evenings. Excellent opportunity to get started, by-pass long years of building.

PRACTICE FOR SALE. Excellent opportunity to join and buy into large active 30 year old I.M. and F.M. practice. Bergen County. Plan early partnership and retirement after introduction. Please send curriculum and needs. Reply Dept. 442 c/o NYSJM.

EQUIPMENT

50% OFF PREVIOUSLY OWNED MEDICAL—Laboratory, x-ray, ultrasound equipment. We buy, sell, broker, repair. Appraisals available by certified surgical consultant. Medical Equipment Resale & Repair, Inc., 24026 Haggerty Road, Farmington Hills, MI 48018. 1-800-247-5826. (313) 477-6880.

DISCOUNT HOLTZER SCANNING SERVICES starting at $35.00. Spacelabs holter recorders available from $1,275.00. Smallest and lightest holters update. Fast service (24–48 hours) turnover. Hook-up kits starting at $4.95. Special introductory offer of three free tests at the purchase or lease of the holter. Stress test electrodes available at 34 cents. Scanning paper available at $18.95. Cardiologist overread available at $15.00. If interested call (301) 870-3626.

EQUIPMENT FOR SALE: Ritter hydraulic chair, Sorensen De-Luxe treatment unit and cabinet, Bausch & Lomb Ferree-Rand perimeter, Marita cuspidor, Bausch & Lomb examiners stool, Castle sterilizer, two Bausch & Lomb equipment stands—all in excellent condition. Contact Jerome Lehner, M.D., 141 Hooker Avenue, Poughkeepsie, New York 12601.

REAL ESTATE FOR SALE OR RENT

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

EAST SIXTIES, PROFESSIONAL BUILDING. The preferred location for internists, cardiologist, rheumatologist. 7 treatment rooms, 2 reception areas to share with three other internists. Share NYC licensed lab, radiology, thermography. Full or part time. (212) 838-2860.

EAST NORTHPORT: Office space for medical practitioner or group. Can provide from 700 to 8000 sq. ft. if necessary. Housed in large white colonial building in East Northport, NY midway between Huntington and Smithtown, Long Island. Almost any sort of financial arrangement can be worked out. Please contact: Robert Smirnow, D.D.S., 7 Pulaski Road, East Northport, L.I., New York 11731, (516) 261-1068.

MANHATTAN, 7 EAST 68th STREET, medical office sub-lease. Lots of rooms, R.M.S., busy office with referrals, flex hours. (212) 879-4260.

HIV Positive Confirmed. Then What?

AIDS is tough to treat. Ruthless to an increasing patient population, it challenges physicians and nurses as never before in their commitment to quality care.

The Spellman Center for HIV-Related Disease at St. Clare's Hospital and Health Center in New York City, accepts the challenge posed by AIDS and HIV infection. The first facility in the tri-state region to devote its resources solely to the care of AIDS patients, the Spellman Center designates its highest priority as compassionate patient care. A dedicated staff provides comprehensive medical, nursing, psychosocial, and dental treatment in an atmosphere that is warm and understanding.

The Spellman Center—a 60-bed Acute Care unit, Emergency Service, Outpatient, Ambulatory Infusion and Dental Clinics—accepts referrals of patients with CDC-confirmed AIDS or ARC (AIDS-related complex), and those who are seropositive for HIV. Spellman also reaches out to offer testing, counseling and treatment to persons at risk for AIDS.

If you need consultation concerning your patients, or more information, please call Dr Debra Spicehandler, the Medical Director, at (212) 307-1161.

The Spellman Center also maintains an information hotline for patients with questions about AIDS: 1-800-433-AIDS.

The Spellman Center
for HIV-Related Disease
A Designated AIDS Treatment Facility
St. Clare's Hospital and Health Center

SC

415 W 51st St
New York, NY 10019

An Affiliate of New York Medical College

MLMIC
Medical Liability Mutual
Insurance Company

Still the right choice for professional liability insurance when you consider all the facts.

Experience: MLMIC is the largest professional liability carrier in New York, and the only one which has met the test of time. The experience of MLMIC's approved defense counsel is unrivaled in the state. These advantages provide an important edge to a doctor confronting the challenge of defending a lawsuit.

Peer Review: Due to the strength of its Peer Review mechanism, MLMIC is the only company that will **not** be using an automatic surcharge schedule to determine which doctors to surcharge and how much. You cannot be cancelled or surcharged on the basis of claims history without prior peer review.

Claims Management: All claims against you are evaluated by your peers and non-meritorious cases are vigorously defended.

Management: MLMIC's dedicated physician board members establish all Company policy and supervise the activities of the most experienced liability insurance professionals in the state.

Non-Assessable Policies: MLMIC policies remain, as always, non-assessable.

Claims Made Insurance: MLMIC has offered claims made insurance since 1982, as long as any insurance company in the state. Doctors insured with other companies can easily change to MLMIC. Changing to MLMIC will not reduce time credit toward free retirement coverage (tail).

In addition, MLMIC continues to offer:

- Superior Value
- Free Risk Management and Legal Hotline
- Full Featured P.C. and Partnership Coverage
- Part-time Coverage
- First Year Physician Rate Reduction
- Informative Newsletter

And more . . .

For more information and an application, contact Medical Liability Mutual Insurance Company at:

MLMIC
250 Harrison St.
Syracuse, NY 13202
(315) 428-1188

MLMIC
2 Park Ave.
New York, NY 10016
(212) 576-9800

THE MOUNT SINAI CENTER FOR FACIAL NERVE DISORDERS

A multi-disciplinary team of physicians and surgeons at The Mount Sinai Hospital for the complete evaluation and treatment of facial nerve disorders.

PROGRAM DIRECTORS

AVRIM R. EDEN, M.D.
CHIEF OF OTOLOGY
THE MOUNT SINAI MEDICAL CENTER

DAVID SIMPSON, M.D.
ASSISTANT PROFESSOR OF NEUROLOGY
CO-DIRECTOR, EMG LABORATORY

Address for patients and correspondence:

FACIAL NERVE CENTER
The Mount Sinai Hospital
19 East 98th Street
Suite 6A, Box 1191
New York, NY 10029

FOR PATIENT APPOINTMENTS OR INFORMATION:

(212) 650-2309

ternal Medicine Program. YMCA of the Rockies, Estes Park. *July 27–31.* **Dynamic Psychotherapy. The Therapeutic Bond.** The Given Institute of Pathobiology, Aspen. *July 31–August 2.* **Pediatric Sports Medicine.** The Given Institute of Pathobiology, Aspen. Contact: Colorado School of Medicine, Office of Continuing Medical Education, 4200 E 9th Ave, Box C-295, Denver, CO 80262. Tel: (313) 394-5195.

MASSACHUSETTS

July 16–18. **Advances in Cardiology.** 16 Cat 1 Credits. Country Inn and Conference Center at Jiminy Peak, Hancock. Contact: Berkshire AHEC, 725 North St, Pittsfield, MA 01201. Tel: (413) 499-5161, ext 2417.

MINNESOTA

July 23–25. **Cardiology Update.** 15 Cat 1 Credits. Minneapolis. Contact: Lisa Krehbiel, Institute for Medical Studies, 30131 Town Center Dr, Suite 215, La-

(*continued on p 11A*)

It's Here At Last...
A Choice of Two Low-Cost Practice Evaluations.

Basic Practice Evaluation
Provides a benchmark for your practice.

Expanded Option Evaluation
Provides 15 different option categories, with 180 evaluations, under a variety of conditions.

Here's Why You Should Consider a Pro-Tech Practice Evaluation:

- Analysis can be used in buying, selling, merging or dissolving of a practice.
- Analysis can serve to confirm other appraisals.
- Analysis is processed quickly — usually within 10 days.

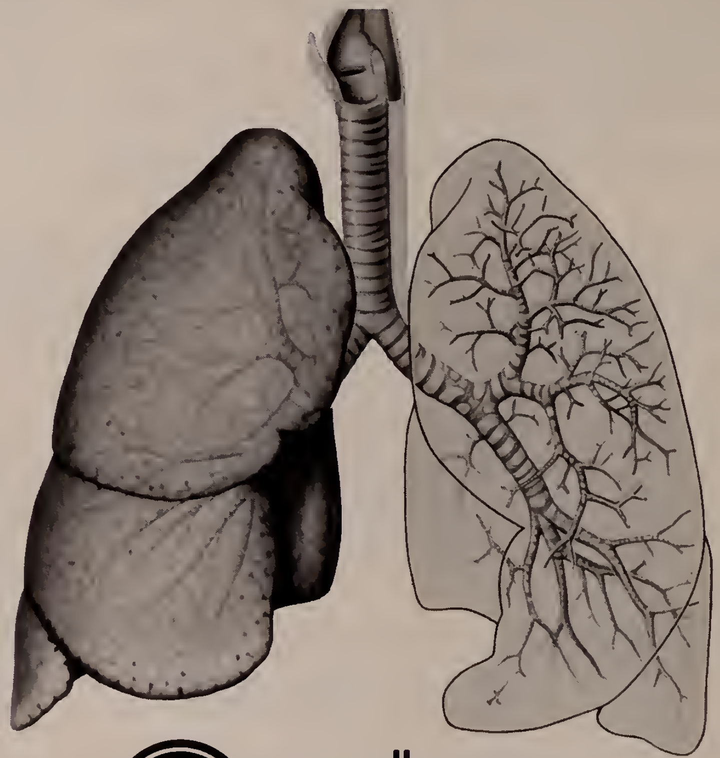
Get the hard facts.

Name ____________ Phone ____________
Address ____________
City ____________ State ______ Zip ______

Pro-Tech Information Systems℠
© 1986 All Rights Reserved
240 Cedar Knolls Rd., Suite 310, Cedar Knolls, NJ 07927-9990

Exclusive Marketing Agents: Sarantos & Co., Inc.

Consider the causative organisms...



Ceclor®
cefaclor

250-mg Pulvules® t.i.d.

offers effectiveness against the major causes of bacterial bronchitis

Haemophilus influenzae, Streptococcus pneumoniae
(ampicillin-susceptible and ampicillin-resistant)

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

Ceclor® (cefaclor)

Summary. Consult the package literature for prescribing information.

Indications: Lower respiratory infections, including pneumonia, caused by susceptible strains of *Streptococcus pneumoniae, Haemophilus influenzae,* and *Streptococcus pyogenes* (group A β-hemolytic streptococci).

Contraindication:
Known allergy to cephalosporins.

Warnings:
CECLOR SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY. POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS.

Administer cautiously to allergic patients.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea. Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.

Precautions:
- Discontinue Ceclor in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of nonsusceptible organisms.
- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- Ceclor should be administered with caution in the presence of markedly impaired renal function. Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk. Exercise caution in prescribing for these patients.

Adverse Reactions: (percentage of patients)

Therapy-related adverse reactions are uncommon. Those reported include:
- Gastrointestinal (mostly diarrhea): 2.5%.
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
- Hypersensitivity reactions (including morbilliform eruptions, pruritus, urticaria, and serum-sickness-like reactions that have included erythema multiforme [rarely, Stevens-Johnson syndrome] or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever): 1.5%; usually subside within a few days after cessation of therapy. Serum-sickness-like reactions have been reported more frequently in children than in adults and have usually occurred during or following a second course of therapy with Ceclor. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.
- Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.
- As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely.
- Rarely, reversible hyperactivity, nervousness, insomnia, confusion, hypertonia, dizziness, and somnolence have been reported.
- Other: eosinophilia, 2%; genital pruritus or vaginitis, less than 1%; and, rarely, thrombocytopenia.

Abnormalities in laboratory results of uncertain etiology
- Slight elevations in hepatic enzymes.
- Transient fluctuations in leukocyte count (especially in infants and children).
- Abnormal urinalysis; elevations in BUN or serum creatinine.
- Positive direct Coombs' test.
- False-positive tests for urinary glucose with Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (glucose enzymatic test strip, Lilly).

[072886R]

PA 8794 AMP

©1987, ELI LILLY AND COMPANY

Lilly

Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285.

Eli Lilly Industries, Inc
Carolina, Puerto Rico 00630

700241

NEW YORK STATE
JOURNAL OF MEDICINE

June 1987 • Volume 87, Number 6

COMMENTARIES

Preventing bicycle-related head injuries

Bicycle accidents are common. Each year in the United States approximately 1,300 persons die from injuries sustained while riding a bicycle.[1] Nearly half of these deaths occur among children. Among children less than ten years old, the death rate from bicycling injuries exceeds that from many other causes that receive considerably more public attention, such as accidental poisonings,[2] falls,[3] firearms accidents,[3] and suffocation caused by aspiration of small toys or parts of toys.[4] The death rate from childhood bicycling mishaps is similar to the death rate from illnesses such as *Haemophilus influenzae* meningitis, a condition for which a major nationwide vaccination campaign has recently been launched.[5]

Bicycle mishaps also cause a large number of nonfatal injuries. The US Consumer Product Safety Commission estimates that each year, more than 350,000 children are treated in hospital emergency rooms because of injuries sustained while bicycling. Emergency room visits are precipitated by bicycling mishaps more frequently than by other childhood recreational activities that are generally considered to be more dangerous, such as skateboarding, sledding, and roller and ice skating.[4]

In nearly all bicycle-related deaths, and in the majority of serious nonfatal bicycling mishaps, head trauma is involved. Head trauma (usually intracranial hemorrhage) is the single most frequent cause of death in fatal bicycling accidents, occurring in approximately 75% of cases.[6-8] Head trauma is also frequently seen in serious nonfatal bicycling injuries, and is the most common reason for hospitalization after a bicycling mishap (nearly 70% of cases).[6,9]

Despite the importance of bicycle-related head injury as a cause of childhood morbidity and mortality, and despite the extraordinarily large number of children who ride bicycles, the issues of bicycle safety and prevention of bicycle-related head injuries have received remarkably little attention. Physicians do not include bicycle safety in the preventive health education they provide to patients.[10] Fewer than one in seven children injured while bicycling report ever having received formal bicycle safety instruction.[9] In fact, only one third have ever received bicycle safety training from their parents.[9] Even when school curricula include bicycle safety as part of the health or safety material taught to children, the instruction that is provided does not always give appropriate emphasis to the importance of preventing head injuries. In particular, school safety programs that include instruction in bicycle safety generally do not teach children about the importance of using bicycle helmets. Rather, they tend to focus on traffic safety. Although good traffic safety skills are important, emphasis on this topic fails to acknowledge that most bicycle-related injuries do not involve collisions with automobiles.[8,9]

Routine use of bicycle helmets is probably the most important intervention that could be made to decrease the incidence of serious bicycle-related injuries. Bicycle helmets have been widely available for more than a decade. In recent years, the design and construction of bicycle helmets have been the subject of intense investigation. Currently available helmets have been subjected to testing similar to that used for motorcycle and football helmets.[11] Laboratory tests suggest that helmets made with a hard plastic outer shell and an expanded polystyrene (styrofoam) inner liner can protect bicyclists from serious head injuries.[11] Helmets with this type of construction are marketed by numerous manufacturers, including Bell, Hanna Pro, Kiwi, Mountain Safety Research (MSR), NJL-Abaddon, OGK, Toys-R-Us, Land Tool Co, Targa Sport, Velta, and others.[12]

Despite the widespread availability of bicycle helmets, however, they are rarely used, particularly by children. Observational studies have shown that fewer than 2% of school-aged children wear a helmet when bicycling.[13] Helmet use rates among young adults are not much greater; only 10% of university-aged students wear helmets when riding a bicycle.[13]

It is unfortunate that bicycle helmets are used so infrequently. In addition to the physiomechanical testing referred to above, which indicates that helmets are capable of preventing injury, there are now studies which suggest that helmet-wearing bicyclists are less likely to sustain serious head injuries than those who do not wear helmets.[8]

Why don't bicyclists wear helmets? The reasons are probably multifactorial, and although little investigative work has been done, several factors have been suggested. One possible reason is that bicycle helmets are expensive, averaging $35–45 for a good quality helmet. This would be a deterrent to many families, although it is important to note that the size of a helmet can be modified by changing fitting pads inside the helmet. Helmets, therefore, are not outgrown on a yearly basis like shoes and clothes. In fact helmets can be used for many years. Since children's head sizes are nearly at their adult size long before their full skeletal height is reached, a helmet purchased at age seven or eight may be used into and through adolescence, merely by changing the fitting pads on an occasional basis.

Another concern is that "peer pressure" negatively influences the tendency of children to wear helmets. If wearing helmets is not perceived as being in style, children may be reluctant to use them. This, of course, can be addressed with proper advertising and "image-making."

One other occasionally mentioned concern is that helmets are uncomfortable because they prevent ventilation of the head. Newer helmet designs, however, address this problem, and most helmets currently available provide excellent air flow about the head.

In the only systematic study of helmet use patterns, pediatricians and family physicians were questioned to determine their opinions about why children do not wear helmets. Surprisingly, the majority of physicians cited none of the reasons mentioned above. Rather, they believed that children fail to wear helmets because of a lack of parental education; their parents had never been told that wearing bicycle helmets was an important thing for children to do![10]

The implications of this finding are obvious. Schools, media, and health professionals could be used to educate the public about the importance of wearing bicycle helmets. If this education were aimed at children in the primary grades, life-long patterns of helmet use could be established.

As physicians, we are letting the public down if we do not use our professional influence to see that a public education campaign is undertaken to increase the rate of helmet usage. The US Public Health Service has recommended that every two-year-old child in the country be vaccinated to prevent *Haemophilus influenzae* meningitis, but it has recommended nothing about bicycle helmets, even though *Haemophilus influenzae* meningitis and bicycling accidents claim similar numbers of children's lives each year. The offices of pediatricians abound with posters and brochures that inform the public about ways to prevent accidental poisonings and aspiration of foreign bodies, but most physicians do nothing to educate the public about bicycle safety, even though bicycle accidents claim the lives of many more children than do poisonings and foreign body aspirations combined.

We have a tremendous opportunity to diminish the death toll from childhood bicycling accidents. Simple educational measures are likely to be effective. All we have to do is act.

BARRY D. WEISS, MD
Chief, Section of Family Medicine

Assistant Professor
Department of Family and
Community Medicine
University of Arizona
Health Sciences Center
Tucson, AZ 86719

1. *Accident Facts*. Chicago, National Safety Council, 1982, pp 45–91.
2. National Center for Health Statistics. *Vital Statistics of the United States*, vol 2, part A. US Dept of Health and Human Services publication No. (PHS) 83-1101. Government Printing Office, 1982.
3. Rivara FP: Traumatic deaths of children in the United States: Currently available prevention strategies. *Pediatrics* 1985; 75:456–462.
4. Toy safety—United States, 1984. *MMWR* 1985; 34:755–762.
5. Advisory Committee for Immunization Practices: Polysaccharide vaccine for prevention of Haemophilus influenza type b disease. *MMWR* 1985; 34:201–205.
6. Guichon DM, Myles ST: Bicycle injuries: One year sample in Calgary. *J Trauma* 1975; 15:504–546.
7. Bouvier R: Bicycle accidents in childhood. Social and preventive aspects. *Aust Fam Physician* 1984; 13:287–289.
8. Weiss BD: Prevention of bicycle-related head injuries. *Am J Prev Med* 1986; 2:330–333.
9. Selbst SM, Alexander D, Ruddy R: Bicycle-related injuries. *Am J Dis Child* 1987; 141:140–144.
10. Weiss BD, Duncan B: Bicycle helmet use by children: Knowledge and behavior of physicians. *Am J Pub Health* 1986; 76:1022–1023.
11. Bishop PJ, Briand BD: Impact performance of bicycle helmets. *Can J Appl Sport Sci* 1984; 9:94–101.
12. Hurt HH, Thom DR: Laboratory tests and accident performance of bicycle safety helmets. Presented at the 29th Annual Proceedings, American Association for Automotive Medicine, October 7–9, 1985, Washington, DC.
13. Weiss BD: Bicycle helmet use by children. *Pediatrics* 1986; 77:677–679.

Diagnostic approaches to Pancoast's syndrome

Henry K. Pancoast, MD,[1,2] a Philadelphia radiologist, first reported the association of upper lung malignancy and involvement of the cervical sympathetic and brachial plexus nerves in the American literature in 1924 and 1932. He described ipsilateral Horner's syndrome (miosis, ptosis, and facial anhidrosis), dystrophic changes and pain of the arm and hand muscles, and tumor extension to the cervical skeletal structures. Although Hare[3] and Tobias[4] had noted similar associations, the syndrome is referred to as Pancoast's syndrome. These tumors were erroneously thought to arise from "a vestigial fissure formed during the embryonal development of the right upper lobe during migration of the azygous vein," the so-called "superior sulcus."[5] Pancoast also postulated that the cells of origin of the tumors were embryonal epithelial rests from the fifth branchial cleft. In fact, the superior sulcus does not exist as an anatomic structure, and the tumors arising in the uppermost lung zones are of bronchogenic origin. Pancoast's tumors have the same cell types as do solitary pulmonary malignancies occurring elsewhere in the periphery of the lung. They are uncommon tumors, accounting for less than 5% of all bronchogenic carcinomas; until the 1950s they had an extremely poor prognosis.

In the 1950s Shaw[6,7] and Chardack[8] reported several cases in which preoperative radiotherapy and extensive surgical resection of the tumor and adjacent involved structures resulted in long survival times. This approach has resulted in current five-year survival rates of 30–35%. The use of extensive radiotherapy alone has also resulted in five-year survival rates of 40% in the absence of lymph node or bone involvement,[9] although surgery followed by irradiation offered no improvement.[8] These uncharacteristically favorable responses to therapy have been attributed to the presurgical "localization" of the tumor by radiotherapy. It is speculated that the lymphatic drainage of the uppermost lung zones may be toward the neck lymphatics rather than toward the hilum, and therefore might delay the spread of mediastinal and distant metastases.

A variety of other conditions also can present as Pancoast's syndrome. These include granulomatous infections of the lung apex, aneurysms of the axillary or carotid vessels, and amyloid accumulation of the apical pleura. Other malignancies can simulate the syndrome by local extension from primary tumors of the thyroid, larynx, and pleura. Although uncommon, Pancoast's syndrome has been reported to occur due to small cell carcinoma,[9,10] and from plasmacytomas arising from the cervical ribs.[11,12] Clearly, preoperative radiotherapy without a tissue diagnosis would be inappropriate in these situations. Furthermore, the survival rate of patients with mediastinal spread of tumors is extremely low, even when the combined radiotherapy-surgery approach is used.

It is therefore important to establish a diagnosis of non-small-cell bronchogenic carcinoma before radiotherapy, and to stage the tumors of all patients so as to avoid radical surgery in those with mediastinal spread.[13] The likelihood of patients having malignant pulmonary nodules can be estimated clinically using a Bayesian analysis.[14] Mediastinal node metastases, invasion of subclavian veins, and vertebral body involvement by tumor can be detected by computed tomography, thereby avoiding unnecessary surgery.[15] Percutaneous needle aspiration using a transcervical approach has been able to establish a diagnosis in 95% of patients.[16] Indeed, in one such patient, a pneumothorax occurred after needle biopsy, which caused the pulmonary nodule to separate from the apical pleura. This patient underwent lobectomy without preoperative radiotherapy.[17] These advances in pretherapeutic diagnosis and staging techniques result in improved selection of patients for the combined therapy approach.

In this issue of the *Journal*, Maxfield and Aranda[18] report their experience using the fiberoptic bronchoscope for the pretherapeutic evaluation of ten patients with Pancoast's tumors. In five of the ten patients, a tissue diagnosis was obtained by transbronchial forceps biopsy under fluoroscopic guidance. In one patient an unsuspected adenocarcinoma was found elsewhere in the lung during fiberoptic examination. In our experience at the Martinez Veterans Administration Medical Center, an incidence of unsuspected findings at bronchoscopy for peripheral lung nodules was 10%. Thus in populations with a history of heavy smoking and a high prevalence of lung cancer, there may be important advantages to the use of fiberoptic bronchoscopy as the initial approach to the diagnosis of Pancoast's tumors.

In order to enter the upper lobe airways with the biopsy forceps, the fiberscope must be flexed to almost 180 degrees. It is often difficult to advance a stiff forceps through the biopsy channel with the fiberscope in full flexion. Recently Wang[19–21] has proposed the use of flexible needles for transbronchial aspiration biopsy of mediastinal and paratracheal lymph nodes for the staging of lung cancer, and for the biopsy of peripheral nodules for diagnosis. In 11 of 23 patients (47%) with peripheral nodules, this method yielded cytologic specimens positive for malignancy. The transbronchial needle aspirate was the only source of positive material in seven of the 23 patients (35%).[21] In our experience, the flexible plastic biopsy needle is capable of easy maneuverability through the bronchoscope even under 180 degrees of flexion. Thus the diagnostic yields reported by Maxfield and Aranda[18] may even underestimate the value of fiberoptic bronchoscopy in the diagnosis and staging of Pancoast's syndrome.

PETER E. KRUMPE, MD
Associate Professor of Medicine
in Residence
University of California
Davis, School of Medicine

Chief, Pulmonary Medicine Section
Martinez Veterans Administration
Medical Center
Martinez, CA 94553

1. Pancoast HK: Superior pulmonary sulcus tumor. Tumor characterized by pain, Horner's syndrome, destruction of bone and atrophy of hand muscles. *JAMA* 1932; 99:1391–1396.
2. Pancoast HK: Importance of careful roentgen-ray investigations of apical chest tumors. *JAMA* 1924; 83:1407–1411.
3. Hare ES: Tumors involving certain nerves. *London Med Gaz* 1838; 23:16–18.
4. Tobias JW: Sindrome ápico-costo-vertebral doloroso por tumor apexiano. Su valos diagnóstico en el cáncer primitivo pulmonar. *Rev Med Latino-Am* 1932; 17:1522–1556.
5 Teixeira JP: Concerning the Pancoast tumor: What is the superior pulmonary sulcus? [editorial]. *Ann Thorac Surg* 1983; 35:577–578.
6. Shaw RR, Paulson DL, Kee JL Jr: Treatment of the superior sulcus tumor by irradiation followed by resection. *Ann Surg* 1961; 154:29–40.
7. Shaw RR: Pancoast's tumor. *Ann Thorac Surg* 1984; 37:343–345.
8. Chardack WM: Pancoast's tumor. *Ann Thorac Surg* 1985; 39:96.
9. Van Houtte P, MacLennan I, Poulter C, et al: External radiation in the management of superior sulcus tumor. *Cancer* 1984; 223–227.
10. Johnson DH, Hainsworth JD, Greco FA: Pancoast's syndrome and small cell lung cancer. *Chest* 1982; 82:602–606.
11. Brenner B, Carter A, Freidin N, et al: Pancoast's syndrome in multiple myeloma. *Acta Haematol* (*Basel*) 1984; 71:353–355.
12. Chen KT, Padmanabhan A: Pancoast syndrome caused by extramedullary plasmacytoma. *J Surg Oncol* 1983; 24:117–118.
13. Stanford W, Barnes RP, Tucker AR: Influence of staging in superior sulcus (Pancoast) tumors of the lung. *Ann Thorac Surg* 1980; 29:406–409.
14. Cummings SR, Lillington GA, Richard RJ: Estimating the probability of malignancy in solitary pulmonary nodules. A Bayesian approach. *Am Rev Respir Dis* 1986; 134:449–452.
15. Webb WR, Jeffrey RB, Godwin JD: Thoracic computed tomography in superior sulcus tumors. *J Comput Assist Tomogr* 1981; 5:361–365.
16. Paulson DL, Weed TE, Rian RL: Cervical approach for percutaneous needle biopsy of Pancoast tumors. *Ann Thorac Surg* 1985; 39:586–587.
17. Rockoff SD: Apical lung cancer masquerading as a Pancoast tumor. *AJR* 1982; 139:163–165.
18. Maxfield RA, Aranda CP: The role of fiberoptic bronchoscopy and transbronchial biopsy in the diagnosis of Pancoast's tumor. *NY State J Med* 1987; 87:326–329.

19. Wang KP, Brower R, Haponik EF, et al: Flexible transbronchial needle aspiration for staging of bronchogenic carcinoma. *Chest* 1983; 84:571-576.
20. Wang KP, Terry PB: Transbronchial needle aspiration in the diagnosis and staging of bronchogenic carcinoma. *Am Rev Respir Dis* 1983; 127:344-347.
21. Wang KP, Haponik EF, Britt EJ, et al: Transbronchial needle aspiration of peripheral pulmonary nodules. *Chest* 1984; 86:819-823.

Amiodarone: Therapeutic but toxic

Early in 1980 a patient asked me, "If this amiodarone is so good at preventing heart rhythm problems, why don't they just put it in the drinking water? It would be like fluorinated water for preventing cavities." I believe this query reflected the enthusiasm that many clinical investigators in this country had for the then-experimental agent amiodarone. This enthusiasm was conveyed to the patient. The enthusiasm reflected the difficulty in establishing control of many life-threatening, sustained ventricular tachyarrhythmias using the available antiarrhythmic agents and therefore the readiness to try a new therapy if there was a suggestion of efficacy during short-term follow-up. The enthusiasm also stemmed from the fact that the drug is easy to administer (with a once-a-day dosing regimen) and is, in general, well tolerated during short-term administration.

Years of experience have tempered the enthusiasm for the use of amiodarone. Although the agent has proven effective in both acute and chronic management of many sustained ventricular arrhythmias, long-term control of these arrhythmias has not been achieved. We have learned much about the methods for predicting long-term control of sustained ventricular arrhythmias with amiodarone. It has been established that abolishing frequent and complex spontaneous ventricular ectopic activity has been associated with control of sustained ventricular arrhythmias.[1,2]

The role of electrophysiologic testing in assessing the efficacy of amiodarone initially appeared controversial.[3,4] In most patients, sustained ventricular arrhythmia remains inducible during treatment with amiodarone. Results of long-term follow-up of large numbers of patients suggest that prevention of inducibility of sustained ventricular tachyarrhythmia with amiodarone therapy may be associated with an excellent prognosis.[5] More importantly, because most arrhythmias remain inducible during amiodarone therapy, patients can be divided into two well-defined risk groups based on whether the induced arrhythmia is well-tolerated hemodynamically.[5,6] Horowitz et al[5] found that none of the 56 patients treated with amiodarone in whom the induced arrhythmia was tolerated hemodynamically at the time of programmed stimulation died suddenly during a mean follow-up period of one year. It is important to note that 46% of these patients had a recurrence of hemodynamically well-tolerated, sustained ventricular arrhythmia.

Despite the improved ability to identify patients who are most likely to have control of their sustained arrhythmia and not experience sudden cardiac death during amiodarone therapy, the drug is still reserved for patients whose arrhythmias prove refractory to other antiarrhythmic agents. The use of amiodarone in the management of less serious ventricular arrhythmias, such as symptomatic nonsustained ventricular tachycardias or symptomatic ventricular ectopic activity, cannot be recommended unless all other antiarrhythmic agents are unsuccessful in controlling the arrhythmia. The basis for this cautious, conservative approach to the use of amiodarone is related to the potential for serious adverse effects during long-term administration of the drug.

Adverse effects related to amiodarone therapy occur in upwards of 80% of patients during chronic administration of the drug.[7-12] Although many of these side effects have limited clinical significance, serious side effects warranting withdrawal of therapy occur in up to 25% of patients during long-term follow-up.[7-10] Table I lists the possible side effects associated with amiodarone therapy with the reported incidence of the adverse reaction. The work by Jacobs[13] in this issue of the *Journal* documents the development of an increase in serum creatinine associated with the institution of amiodarone. The incidence of this adverse effect may be more common than previously recognized.[7] Direct nephrotoxicity with an associated inflammatory process—based on pathologic findings—has not been demonstrated, and thus the reason for the observed laboratory abnormalities can only be speculated.

The pathogenesis of many of the toxic manifestations of amiodarone remains unclear. Lamellated cytoplasmic inclusion bodies identical to those seen in phospholipidosis have been demonstrated in tissue from organs involved with the toxic inflammatory process associated with amiodarone therapy.[14-16] However, these same inclusion bodies have been found in the absence of an associated inflammatory process. It is not known whether these inclusions are essential for the development of many of the toxic manifestations of amiodarone or whether they represent a nonspecific finding present in most or possibly all patients who receive the drug.

Unfortunately, the ability to identify patients at risk for toxicity appears limited. Cigarette smokers and those patients who manifest abnormalities on baseline pulmonary function tests may be at greatest risk for the pulmonary alveolitis that probably represents the most serious toxic manifestation associated with amiodarone therapy.[8,14] As noted by Jacobs,[13] some of the toxic manifestations appear to depend on serum concentration. Dermatologic, ocular, neuromuscular, and hepatic abnormalities appear to occur more commonly in association with increased dosages of amiodarone and elevated serum amiodarone con-

TABLE I. Adverse Effects of Amiodarone

Side Effect	Estimated Incidence
Dermatologic	
Bluish discoloration	2–5%
Photosensitivity	10–55%
Rash	2–5%
Ocular	
Asymptomatic corneal microdeposits	30–90%
Halo vision	2–5%
Pulmonary	
Alveolitis/interstitial fibrosis	2–5%
Cardiac	
Worsening heart failure	2–10%
Symptomatic bradyarrhythmia	1–2%
Torsade de pointe	<1%
Hepatic	
Serum transaminase elevation	15–50%
Fulminant hepatic necrosis	1–2%
Gastrointestinal	
Anorexia	2–10%
Constipation	20–50%
Endocrinologic	
Hypothyroidism	3–30%
Hyperthyroidism	1–5%
Neurologic	
Proximal muscle weakness	1–6%
Ataxia, tremor, dizziness	5–40%
Peripheral neuropathy	<1–3%
Renal	
Serum creatinine elevation	1–9%
Epididymitis	<1–6%

centrations.[8,17-19] In a recent study, steady-state serum levels of amiodarone and its major metabolite, desethylamiodarone, were correlated to the development of toxicity in more than 100 patients.[20] Although no definite relationship was established between any individual adverse effect and the serum concentration, a serum amiodarone concentration of greater than 2.5 mg/L had a positive predictive value of 76% for identifying patients with any adverse effect. The results of this study suggest that if the serum concentration is greater than 2.5 mg/L, the patient should be monitored closely for adverse effects. Clearly, if the arrhythmia being treated is not life threatening, a reduction in dosage is indicated when such levels of amiodarone are noted.

The interaction of amiodarone with other cardioactive agents such as procainamide, flecainide, digoxin, phenytoin, and warfarin can also result in increased serum concentrations and pharmacologic effects of these commonly used agents.[21,22] It is frequently difficult to predict the degree of interaction of amiodarone with these agents. Thus, these agents—which have low therapeutic-to-toxic ratios in their own right—often cannot be used.

Any discussion of the toxic manifestations of amiodarone would be incomplete without mention of the drug's breakdown products. The drug is mainly excreted in the bile. Following chronic oral administration, amiodarone has a biphasic elimination pattern.[23] Fifty percent of the compound is eliminated within ten days; however, the terminal elimination phase is markedly prolonged, with a half-life of over 50 days for amiodarone. The half-life of the drug's major metabolite, desethylamiodarone, appears to be even longer. The desethyl metabolite formed in the liver clearly contributes to the electrophysiologic effects of amiodarone and may contribute to the toxic manifestations of the drug. The markedly prolonged half-life of amiodarone makes the management of serious forms of toxicity difficult.

A detailed bibliography of all the work published over the last ten years related to the antiarrthymic efficacy of and toxicity associated with amiodarone would fill every page of the *Journal*. Despite the plethora of investigations, we still lack important information. Can we hasten the metabolism of the drug when toxicity becomes apparent? Can we identify factors that better predict the development of toxicity? These are just two of the questions that need to be addressed. Amiodarone has not been placed in the drinking water reservoirs, but its efficacy in controlling serious cardiac arrhythmias has been placed in the well of the practicing physician. The physician should draw from this well with some trepidation. Familiarity with all of amiodarone's many toxic manifestations is essential for optimal patient care.

FRANCIS E. MARCHLINSKI, MD
Clinical Electrophysiologic Laboratory
Hospital of the University of
Pennsylvania
Philadelphia, PA 19104

Acknowledgments. The author thanks Faith Marie Hope for her skillful preparation of this manuscript.

Supported in part by grants from the American Heart Association; the National Heart, Lung and Blood Institute (HL24278, HL28093); and the McCabe Fund.

1. Marchlinski FE, Buxton AE, Flores BT, et al: Value of Holter monitoring in identifying risk for sustained ventricular arrhythmia recurrence on amiodarone. *Am J Cardiol* 1985; 55:709–712.
2. Kim SG, Felder SD, Figura I, et al: Value of Holter monitoring in predicting long-term efficacy and inefficacy of amiodarone used alone and in combination with class 1A antiarrhythmic agents in patients with ventricular tachycardia. *J Am Coll Cardiol* 1987; 9:169–174.
3. Waxman HL, Groh WG, Marchlinski FE, et al: Amiodarone for control of sustained ventricular tachyarrhythmias: Clinical and electrophysiologic effects in 51 patients. *Am J Cardiol* 1982; 50:1066–1074.
4. Hamer AW, Finerman WB Jr, Peter T, et al: Disparity between the clinical and electrophysiologic effects of amiodarone in the treatment of recurrent ventricular tachyarrhythmias. *Am Heart J* 1981; 102:992–1000.
5. Horowitz LN, Greenspan AM, Spielman SR, et al: Usefulness of electrophysiologic testing in evaluation of amiodarone therapy for sustained ventricular tachyarrhythmias associated with coronary heart disease. *Am J Cardiol* 1985; 55:367–371.
6. Kadish AH, Buxton AE, Waxman HL, et al: Usefulness of electrophysiologic study to determine the clinical tolerance of arrhythmic recurrences during amiodarone therapy. *J Am Coll of Cardiol* (in press).
7. Fogoros RN, Anderson KP, Winkle RA, et al: Amiodarone: Clinical efficacy and toxicity in 96 patients with recurrent, drug-refractory arrhythmias. *Circulation* 1983; 68:88–94.
8. Greene HL, Graham EL, Werner JA, et al: Toxic and therapeutic effects of amiodarone in the treatment of cardiac arrhythmias. *J Am Coll Cardiol* 1983; 2:1114–1128.
9. Harris L, McKenna WJ, Rowland E, et al: Side effects of long-term amiodarone therapy. *Circulation* 1983; 67:45–51.
10. Haffajee CI, Love JC, Alpert JS, et al: Efficacy and safety of long-term amiodarone in treatment of cardiac arrhythmias: Dosage experience. *Am Heart J* 1983; 106:935–943.
11. Nademanee K, Singh BN, Callahan B, et al: Amiodarone, thyroid hormone indexes and altered thyroid function: Long-term serial effects in patients with cardiac arrhythmias. *Am J Cardiol* 1986; 58:981–986.
12. Albert SG, Alves LE, Rose EP: Thyroid dysfunction during chronic amiodarone therapy. *J Am Coll Cardiol* 1987; 9:175–183.
13. Jacobs MB: Serum creatinine increase associated with amiodarone therapy. *NY State J Med* 1987; 87:358–359.
14. Marchlinski FE, Gansler TS, Waxman HL, et al: Amiodarone pulmonary toxicity. *Ann Intern Med* 1982; 97:839–845.
15. Delage C, Lagace R, Huard J: Pseudocyanotic pigmentation of the skin

induced by amiodarone: A light and electron microscopic study. *Can Med Assoc J* 1975; 112:1205-1208.
16. Rigas B, Rosenfeld LE, Barwick KW, et al: Amiodarone hepatotoxicity. *Ann Intern Med* 1986; 104:348-351.
17. Rotmensch HH, Belhassen B, Swanson BN, et al: Steady-state serum amiodarone concentrations: Relationships with antiarrhythmic efficacy and toxicity. *Ann Intern Med* 1984; 101:462-469.
18. Haffajee CI, Love JC, Canada AT, et al: Clinical pharmacokinetics and efficacy of amiodarone for refractory tachyarrhythmias. *Circulation* 1983; 67:1347-1355.
19. Heger JJ, Prystowski EN, Zipes DP: Relationships between amiodarone dosage, drug concentrations, and adverse side effects. *Am Heart J* 1983; 106:931-935.
20. Falik R, Flores BT, Shaw L, et al: The relationship of steady-state serum concentrations of amiodarone and desethylamiodarone to therapeutic efficacy and adverse effects. *Am J Med* 1987; 82:1102-1108.
21. Saal AK, Werner JA, Greene HL, et al: Effect of amiodarone on serum quinidine and procainamide levels. *Am J Cardiol* 1984; 53:1264-1267.
22. Nademanee K, Kannan R, Hendrickson J, et al: Amiodarone-digoxin interaction: Clinical significance, time course of development, potential pharmacokinetic mechanisms and therapeutic implications. *J Am Coll Cardiol* 1984; 4:111-116.
23. Holt DW, Tucker GT, Jackson PR, et al: Amiodarone pharmacokinetics. *Am Heart J* 1983; 106:840-847.

The status of amiodarone

The appropriate use of amiodarone remains controversial a year after its release in the United States, and more than 20 years after its introduction in much of the rest of the world. Opinions in the literature vary markedly regarding the efficacy and toxicity of this novel antiarrhythmic agent. The reasons for the apparently diverse experiences with amiodarone have to do both with the unusual characteristics of the drug itself and with the unusual manner in which amiodarone came into use in the United States.

Amiodarone was first synthesized more than 20 years ago in France and was quickly marketed for the treatment of angina. Over the next several years, physicians noted that amiodarone seemed to display significant antiarrhythmic properties. Formal study of these properties began overseas in the early 1970s, and reports from these studies began to appear in the American literature later in the decade.[1-3] These reports were astoundingly favorable, each remarking on amiodarone's high rate of efficacy against both atrial and ventricular tachyarrhythmias, and on the drug's low incidence of toxicity. Such reports excited investigators in the US. Accordingly, many American investigators independently sought and obtained permission to prescribe amiodarone on a compassionate clearance basis. During the early 1980s, the use of amiodarone in the US became widespread for treatment of arrhythmias.

The use of amiodarone in this country remained uniquely outside the auspices of any American pharmaceutical house. Consequently, without this usual coordinating force, the reporting of results with amiodarone was at best haphazard, and depended on those investigators who cared to have their data published. Because of this lack of a centralized data-gathering system, accessible data regarding amiodarone was incomplete, and was subject much more than usual to variations in patient selection, dosing, therapeutic and toxic endpoints, follow-up methods, and data analysis. Any attempt to interpret the literature on amiodarone must take into account these inherent limitations.

The pharmacokinetic features of amiodarone are complex and poorly understood, and they contribute to the difficulty in evaluating the drug's efficacy and toxicity. Such features include the following.[4-5] Bioavailability with oral dosing is variable (between 20% and 60%). The pharmacologic distribution of amiodarone is complex and speculative—usually a three- or four-compartment model is invoked to describe its kinetics. Despite the existence of several compartments of distribution, the importance of specific compartments to either efficacy or toxicity is not understood. The complex distribution of the drug results in an apparently prolonged loading period (generally believed to be four to six weeks) before maximum efficacy is realized. The complex distribution of the drug results in a long half life (between 13 and 107 days), with measurable drug levels persisting as long as nine months after the drug is discontinued. Amiodarone has a major metabolite (desethylamiodarone), the antiarrhythmic activity effects and pharmacokinetics of which are unknown at this time. Blood levels of amiodarone or its desethyl metabolite have not correlated with either efficacy or toxicity.

Despite the complex pharmacokinetics of amiodarone, no systematic studies have been done to optimize either loading or maintenance dosing schedules. Physicians using the drug have had to rely on their best judgment and personal experience to regulate dosages. The resultant diverse dosing schedules make it all the more difficult to interpret the amiodarone literature.

In light of this information, it is not surprising that reports of amiodarone's efficacy are conflicting. Early studies in the US almost universally agreed with the overseas literature that amiodarone was remarkably effective in the treatment of tachyarrhythmias.[6-9] Much of this early data was flawed by relatively small numbers of patients, short-term follow-up, and the failure to employ actuarial methods in analyzing longitudinal data. Not surprisingly, later reports using actuarial analysis have suggested that amiodarone may not be as uniquely efficacious as previously thought in the treatment of patients with malignant arrhythmias such as sustained ventricular tachycardia or fibrillation (VT/VF).[10-12]

A major problem in assessing amiodarone's efficacy has been the widespread practice of empirically using amiodarone in patients with malignant arrhythmias. This practice has resulted from the belief that inducing VT/VF during electrophysiologic testing with amiodarone (in contrast to all other antiarrhythmic agents) does not predict a poor outcome during chronic therapy. This phenomenon may in fact not be unique to amiodarone. As pointed out by Winkle,[13] a substantial proportion of patients who receive chronic treatment with drugs predicted to be ineffective by electrophysiologic testing do not have recurrent VT/VF. In addition, we have compared actuarial rates of arrhythmia recurrence in patients with drug-refractory VT/VF treated empirically with amiodarone, versus similar patients treated with drugs that were predicted by electrophysiologic testing to be ineffective.[12] We found that while amiodarone was more protective than "ineffective" drugs for the first six months of therapy, the two-year actuarial risk of having recurrent VT/VF was the same in both groups. This preliminary data would indicate that long-term empiric therapy with amiodarone is no more effective at preventing recurrent VT/VF than is therapy with "ineffective" drugs. These data support the growing consensus that empiric therapy with amiodarone is not adequate for patients with malignant ventricular arrhythmias. Unfortunately, proposed methods to make the use of amiodarone less empiric (including a renewed interest in electrophysiologic testing) have not been validated.

It is becoming clear that amiodarone presents toxic potential which is unique for an antiarrhythmic agent. Early reports in the US again supported the experience overseas and found little or no significant toxicity with the drug.[6-9] As time went by, however, well-documented reports appeared of serious toxicity involving most organ systems, including the heart, lungs, skin, liver, gastrointestinal tract, skeletal muscles, and the central and peripheral nervous systems. In addition, multiple drug interactions were reported. Some of these side effects (such as pneumonitis) now appear to be relatively frequent and potentially lethal.

Unfortunately, the lack of a centralized data-gathering system for amiodarone has made it next to impossible to determine the true incidence of most side effects. The toxicity reported from center to center continues to vary, and some types of toxicity may remain unnoticed or ignored. A case in point is the report by Jacobs[14] in this issue of the *Journal*, in which a convincing case of renal toxicity due to amiodarone is described. While renal toxicity with amiodarone was first described four years ago,[10] it has since gone virtually unnoticed. Whether this toxicity has gone unnoticed because it is infrequent, or because physicians have simply not recognized it, cannot be stated on the basis of available data.

Validated methods for reducing the risk of amiodarone toxicity have not been described, but will clearly not be as simple as following serum drug levels. As is not surprising for a drug whose metabolism is described by a multicompartment model, various toxicities are likely related to different factors. Some side effects (pneumonitis, skin discoloration) appear to be related to cumulative dose rather than daily dose.[15] Other side effects (gastrointestinal) may be related to daily dose or method of administration, and yet other side effects (skin photosensitivity) may be related to environmental influences. The safe use of amiodarone cannot be assured by any known method. It must be assumed that any patient started on amiodarone is at risk for a multitude of side effects, and must be carefully observed for the duration of therapy—and possibly after therapy is stopped.

Amiodarone is a novel antiarrhythmic agent which has engendered much enthusiasm in the US. While this enthusiasm is now tempered by questions regarding the drug's unique efficacy and by a growing list of toxic effects, amiodarone is still generally acknowledged to have superior antiarrhythmic activity. Unfortunately, the lack of a coordinated system for collecting amiodarone-related data, the complicated and poorly understood pharmacokinetics of the drug, and the incompletely defined multiorgan toxicity of amiodarone currently make the safe use of this drug difficult. Despite the drug's release for general use, amiodarone should still be reserved for patients with incapacitating or life-threatening arrhythmias who cannot benefit from other antiarrhythmic agents or from nonpharmacologic therapy.

RICHARD N. FOGOROS, MD
Assistant Professor of Medicine
University of Pittsburgh
School of Medicine

Director, Clinical Electrophysiology
Division of Cardiology
Presbyterian-University Hospital
Pittsburgh, PA 15213

1. Rosenbaum MB, Chiale PA, Halpren MS, et al: Clinical efficacy of amiodarone as an antiarrhythmic agent. *Am J Cardiol* 1976; 38:934-944.
2. Rowland E, Krikler DM: Electrophysiological assessment of amiodarone in treatment of resistant supraventricular arrhythmias. *Br Heart J* 1980; 44:82-90.
3. Kaski JC, Girotti LA, Messuti H, et al: Long-term management of sustained, recurrent symptomatic ventricular tachycardia with amiodarone. *Circulation* 1981; 64:273-279.
4. Holt DW, Tucker GT, Jackson PR, et al: Amiodarone pharmacokinetics. *Am Heart J* 1983; 106:840-847.
5. Siddoway LA, McAllister CB, Wilkinson GR, et al: Amiodarone dosing: A proposal based on its pharmacokinetics. *Am Heart J* 1983; 106:951-956.
6. Marcus F, Fontaine GH, Frank R, et al: Clinical pharmacology and therapeutic applications of the antiarrhythmic agent amiodarone. *Am Heart J* 1981; 101:480-493.
7. Nademanee K, Hendrickson JA, Cannom DS, et al: Control of refractory life-threatening ventricular tachyarrhythmias by amiodarone. *Am Heart J* 1981; 101:759-768.
8. Groh WC, Kastor JA, Josephson ME, et al: Amiodarone: An effective drug for refractory ventricular tachycardia. *Circulation* 1980; 62(suppl III): III-152 [abstract].
9. Peter T, Hamer A, Mandel WJ, et al: Evaluation of amiodarone therapy in the treatment of drug-resistant cardiac arrhythmias: Long-term follow-up. *Am Heart J* 1983; 106:943-950.
10. Fogoros RN, Anderson KP, Winkle RA, et al: Amiodarone: Clinical efficacy and toxicity in 96 patients with recurrent, drug-refractory arrhythmias. *Circulation* 1983; 68:88-94.
11. Veltri EP, Reid PR, Platia EV, et al: Amiodarone in the treatment of life-threatening ventricular tachycardia: Role of holter monitoring in predicting long-term clinical efficacy. *J Am Coll Cardiol* 1985; 6:806-813.
12. Fogoros RN, Fiedler SB, Elson JJ: Empiric amiodarone vs "ineffective" drug therapy in patients with inducible ventricular tachyarrhythmias. *PACE* 1986; 9(2):143.
13. Winkle RA: Amiodarone and the American way [editorial]. *J Am Coll Cardiol* 1985; 6:822-824.
14. Jacobs MB: Serum creatinine increase associated with amiodarone therapy. *NY State J Med* 1987; 87:358-359.
15. Mason JW: Toxicity of amiodarone. *Circulation* 1985; 72(suppl):III-272 [abstract].

The role of fiberoptic bronchoscopy and transbronchial biopsy in the diagnosis of Pancoast's tumor

ROGER A. MAXFIELD, MD, CONRADO P. ARANDA, MD

ABSTRACT. To assess the role of fiberoptic bronchoscopy in the diagnosis of Pancoast's tumor, records were reviewed from ten patients with confirmed Pancoast's tumor who had undergone fiberoptic bronchoscopy and transbronchial biopsy. No endobronchial tumor extension was seen. Transbronchial biopsy revealed bronchogenic carcinoma in four patients; one biopsy revealed an acid-fast organism as well. Bronchial washings and brushings provided no definitive diagnoses. Sputum for cytologic studies revealed malignant cells in two of seven patients. Following negative results from transbronchial biopsy and cytologic analysis, fine needle aspiration biopsy was performed in two patients, revealing malignant cells in both patients. A patient with suspected but unconfirmed Pancoast's tumor was found to have an unsuspected contralateral endobronchial adenocarcinoma. No complications were associated with fiberoptic bronchoscopy and transbronchial biopsy. These procedures can safely provide a histologic diagnosis in some patients with Pancoast's tumor, while providing other useful information regarding second primary tumors or concomitant infection which might not be detected by needle aspiration alone.

(NY State J Med 1987; 87:326–329)

Tumors of the superior pulmonary sulcus, or Pancoast's tumors,[1] are usually bronchogenic carcinomas that arise from the apex of the lung. These tumors spread by direct invasion to the adjacent soft tissues of the thoracic inlet, including the brachial plexus. Because the tumor invades nerve and soft tissue, most patients with Pancoast's tumor present with shoulder or arm pain, and many have Horner's syndrome.[2–4]

Based on the characteristic clinical and radiographic presentation, a clinical diagnosis of Pancoast's tumor due to bronchogenic carcinoma can be made with 90–95% accuracy.[2,5] Preoperative radiation therapy followed by en bloc resection of the tumor and involved portions of the chest wall has been considered the treatment of choice for patients in whom the tumor has not spread to the mediastinum.[2,5] Surgical biopsy procedures are believed to interfere with successful radiotherapy, so treatment has been advocated based on a clinical diagnosis alone.[2] However, many conditions other than bronchogenic carcinoma can cause Pancoast's syndrome. These conditions include direct spread of laryngeal or thyroid carcinoma,[4] Hodgkin's disease,[4] mesotheliomas,[4] metastatic carcinoma,[6,7] myeloma,[8] actinomycosis,[9] tuberculosis,[10] and rupture of an echinococcal cyst.[11]

Because of the diversity of conditions capable of producing Pancoast's syndrome, pretreatment histologic diagnosis would be desirable if it could be obtained without interfering with therapeutic plans. Rigid bronchoscopy does not permit biopsy of the apex, and fiberoptic bronchoscopy has similarly been considered to be of little value in obtaining tissue for a histologic diagnosis in patients with this tumor.[2,12,13] In contrast, a high diagnostic yield has been reported with thin needle aspiration biopsy in patients with Pancoast's tumor.[14]

The purpose of this study was to review the role of fiberoptic bronchoscopy with transbronchial biopsy in establishing a histologic diagnosis of Pancoast's tumor of the lung. The study is based on the experience over the last seven years at an urban Veteran's Administration hospital. Previous studies have not directly addressed this issue.

PATIENTS AND METHODS

Because there is no precise anatomic site consisting of the "superior pulmonary sulcus," criteria for diagnosing Pancoast's tumor have not been uniform.[15,16] In Pancoast's original series, all of the patients had "roentgenographic evidence of a small, homogenous shadow at the extreme apex" with "more or less rib destruction," and "pain around the shoulder and down the arm, Horner's syndrome, and atrophy of the muscles of the hand."[1] For the purpose of this study, cases were defined by Paulson's criteria, which do not require Horner's syndrome or bone involvement, but do include characteristic pain and a roentgenographic shadow at the extreme apex or thoracic inlet.[2]

Cases were identified by reviewing all bronchoscopy records from 1978 through 1984. Clinical records, chest radiographs, pathology reports, and cytology reports were reviewed from all of the ten patients with confirmed carcinoma of the superior pulmonary sulcus who had undergone fiberoptic bronchoscopy with transbronchial biopsy during this seven-year period. These ten patients all met the clinical and roentgenographic criteria for Pancoast's tumor.

Bronchoscopies were performed with a flexible fiberoptic bronchoscope (Olympus BF B3) under topical anesthesia. Transbronchial biopsy from the apex was performed using a fenestrat-

---

From the Chest Service and the Department of Medicine, Veterans Administration Medical Center, New York University School of Medicine, New York, NY.

Address correspondence to Dr Aranda, Veterans Administration Medical Center, Room 14107 S, First Ave at 24th St, New York, NY 10010.

ed transbronchial biopsy forceps (Olympus FB-21C) under fluoroscopic guidance. Because of the extreme bend in the bronchoscope required to reach the apex, the forceps were advanced 1 cm beyond the tip of the bronchoscope before entering the apical segment. This facilitated passage of the forceps into the apex. In some cases it was necessary to push the bend of the bronchoscope down on a carina, using this carina as a fulcrum to achieve a more acute angular bend with the bronchoscope.

Apical bronchial washings for cytologic analysis were obtained from all of the patients, and bronchial brushings from the apex were obtained from seven patients. Sputum for cytologic analysis was obtained from seven patients. In two patients, following negative results from transbronchial biopsy, needle aspiration biopsy was performed percutaneously using a 20–21 gauge spinal needle under local anesthesia with fluoroscopic guidance.

RESULTS

The patients ranged in age from 45 to 70 years, with a mean age of 59 years (Table I). Eight patients reported ipsilateral shoulder or arm pain, and three of these patients had Horner's syndrome. One patient had upper chest and neck pain, and another reported pain only in the lateral chest wall but had Horner's syndrome as well. All of the patients smoked cigarettes.

Chest roentgenograms of all patients revealed masses at the apex of the lung, with eight masses on the right lung and two on the left lung. The tumor densities ranged from 1 cm to 6 cm in vertical diameter, with a mean vertical diameter of 3.9 ± 1.7 cm. Four patients demonstrated radiographic evidence of destruction of the upper ribs.

TABLE I. Clinical and Roentgenographic Characteristics in Ten Patients with Pancoast's Tumor

Patient	Age (yr)	Symptoms	Results of Chest Roentgenogram
1	66	Right arm pain	3-cm right apical mass
2	70	Right chest, shoulder, and arm pain; right arm weakness; Horner's syndrome	6-cm right apical mass with 2nd rib destruction
3	59	Right lateral chest pain; Horner's syndrome	4-cm right apical mass
4	45	Left shoulder and arm pain	1-cm left apical mass
5	58	Right upper chest and neck pain	6-cm right apical mass
6	59	Left shoulder pain; left arm weakness; Horner's syndrome	3-cm left apical mass
7	64	Right shoulder pain; right arm weakness	5-cm right apical mass with 1st–4th rib destruction
8	49	Right shoulder pain	2-cm right apical mass
9	62	Right neck and shoulder pain; Horner's syndrome	5-cm right apical mass with 1st–2nd rib destruction
10	62	Right shoulder, upper chest, and back pain	4-cm right apical mass with 1st rib erosion

The results of bronchoscopy and other diagnostic studies are shown in Table II. No endobronchial tumor extension was noted. One patient had what appeared to be bulging of the right tracheal wall due to extrinsic compression without endobronchial invasion. The endobronchial inspection was normal in the other patients.

Four of the ten apical transbronchial biopsies were positive for carcinoma. Three revealed squamous cell carcinoma, and one showed poorly differentiated carcinoma. One of the biopsies revealed an acid-fast bacillus in addition to the carcinoma. The vertical diameter of the tumors from patients with positive transbronchial biopsies was 3.7 ± 1.7 cm compared to 4 ± 1.8 cm for those with a negative biopsy ($p > 0.4$). With regard to the four patients with rib involvement, two transbronchial biopsies were positive for carcinoma and two were negative. Similarly, from the four patients with Horner's syndrome, two transbronchial biopsies were positive for malignancy and two were negative.

Cytologic analysis of apical bronchial washings and brushings provided no definitive diagnoses. Two washings and one brushing were suspicious for squamous cell carcinoma, with the remainder of these studies providing negative results.

Sputum samples were collected prior to bronchoscopy in five patients; sputum analysis revealed squamous cell carcinoma in one patient. This patient had a negative transbronchial biopsy, but the diagnosis was confirmed at surgery. Sputum samples were collected after bronchoscopy in two patients, both of whom had positive transbronchial biopsies. One of these sputum samples yielded positive findings, and the other was highly suspicious for squamous cell carcinoma.

Specimens obtained by percutaneous needle aspiration were positive for carcinoma in both of the patients who underwent this procedure following negative findings on transbronchial biopsy and cytologic analysis. One aspirate revealed squamous cell carcinoma, with the other showing an undifferentiated, non-small cell carcinoma. In the remainder of the patients with negative transbronchial biopsies, a final diagnosis was made at thoracotomy in two patients and by cervical lymph node biopsy in one.

A patient who was not included in the above analysis because of lack of tissue confirmation had a clinical and roentgenographic syndrome compatible with Pancoast's tumor on the right lung. Transbronchial biopsy of the apex of the right lung was negative, but bronchoscopy revealed an unsuspected endobronchial adenocarcinoma of the left upper lobe bronchus. Left supraclavicular lymph node biopsy was also positive for adenocarcinoma.

No complications arose from any of the bronchoscopic procedures or transbronchial biopsies performed in these patients.

DISCUSSION

The results of this study indicate that fiberoptic bronchoscopy and transbronchial biopsy can provide valuable diagnostic information in patients with Pancoast's tumors. Previous studies have found bronchoscopy to be of little value in obtaining a histologic diagnosis in this tumor, and most authors have dismissed bronchoscopy without providing specific data on diagnostic yield from this procedure.[2,12,13] One study reported no positive diagnoses from ten transbronchial biopsies obtained from patients with Pancoast's tumor.[14]

In contrast to these reports, transbronchial biopsy in the present series revealed a positive histologic diagnosis in 40% of the patients undergoing this procedure. The likelihood of obtaining a positive diagnosis in these patients was not dependent on the size of the tumor or on the extent of bone or nerve involvement. In one case reported in the literature, transbronchial biopsy provided a histologic diagnosis of a Pancoast's tumor that was too small to be seen on a plain chest roentgenogram.[17] Another study reported histologic diagnoses made with transbronchial biopsy in five patients with Pancoast's tumor.[18] However, the num-

TABLE II. Results of Diagnostic Studies in Ten Patients with Pancoast's Tumor

Patient	Transbronchial Biopsy	Cytologic Analysis of Sputum	Cytologic Analysis of Bronchial Brushing	Cytologic Analysis of Bronchial Washing	Needle Aspiration Biopsy	Final Diagnosis
1	Negative	Negative (prebronchoscopy)	Negative	Negative	—	Squamous cell carcinoma (thoracotomy)
2	Poorly differentiated squamous cell carcinoma	Squamous cell carcinoma (post-bronchoscopy)	Suspicious squamous cell carcinoma	Negative	—	Poorly differentiated squamous cell carcinoma
3	Negative	Suspicious squamous cell carcinoma (pre-bronchoscopy)	Negative	Negative	—	Poorly differentiated squamous cell carcinoma (lymph node)
4	Negative	Negative (pre-bronchoscopy)	Negative	Negative	—	Large cell carcinoma (thoracotomy)
5	Negative	Squamous cell carcinoma (pre-bronchoscopy)	Negative	Suspicious poorly differentiated squamous cell carcinoma	—	Squamous cell carcinoma
6	Poorly differentiated carcinoma	—	Negative	Negative	—	Poorly differentiated carcinoma
7	Negative	—	Negative	Negative	Squamous cell carcinoma	Squamous cell carcinoma
8	Squamous cell carcinoma and acid-fast bacilli	Highly suspicious (post-bronchoscopy)	—	Highly suspicious squamous cell carcinoma	—	Squamous cell carcinoma
9	Negative	—	—	Negative	Non-small cell carcinoma	Non-small cell carcinoma
10	Squamous cell carcinoma	Negative (pre-bronchoscopy)	—	Negative	—	Squamous cell carcinoma

ber of bronchoscopic procedures performed was not reported, so the diagnostic yield could not be calculated.

Cytologic analysis of bronchial washings and brushings was of no value in these patients. The finding of cells suspicious for malignancy in three patients added no diagnostic information, because in this group of patients there was already a high degree of suspicion of malignancy based on the clinical and roentgenographic findings. Other authors have reported similarly low yields from bronchial washings.[18] Cytologic analysis of sputum has been reported to yield a positive diagnosis in 11–18% of patients with superior sulcus tumors.[3,12,14] The results from cytologic analysis of sputum samples in the present study are consistent with these reports.

Needle aspiration biopsy was performed in only two patients in the present study. However, this technique provided a positive tissue diagnosis of malignancy in both these patients. In a larger series, needle aspiration biopsy provided a positive diagnosis of malignancy in 26 of 27 cases of Pancoast's tumor.[14] Pneumothorax occurred in three of 30 procedures performed on these 27 patients.

Although needle aspiration biopsy provides a higher diagnostic yield than fiberoptic bronchoscopy, bronchoscopy may have advantages as the primary invasive diagnostic procedure in patients with superior sulcus tumors. In contrast to needle aspiration, bronchoscopy permits visualization of the entire upper airway and tracheobronchial tree, which can provide information regarding the extent of tumor spread and the presence of second primary tumors. An unexpected, contralateral second primary tumor was found in a patient from this institution who was undergoing bronchoscopy for a suspected Pancoast's tumor. In population groups with a history of heavy smoking, second primary tumors are not uncommon. In a study of solitary pulmonary nodules, nine of 101 patients had tumors remote from their nodules which were detected by fiberoptic bronchoscopy.[19] Knowledge of the presence of a second primary tumor in patients with Pancoast's tumor would influence plans for initiating therapy with local radiation and surgery.

Pneumothorax is reported to occur as a complication in 10–39% of patients who have undergone needle aspiration biopsies.[20-25] In a series limited to Pancoast's tumors, pneumothorax occurred in only 10% of patients who underwent this biopsy procedure.[14] This contrasts with a pneumothorax rate of 2–5% following bronchoscopy with transbronchial biopsy.[25-28] The pneumothorax rate following transbronchial biopsy falls to less than 1% if fluoroscopic guidance is used for all biopsies.[29] Therefore, with respect to pneumothorax, it appears that transbronchial biopsy may be a safer procedure than needle aspiration biopsy.

Although there is a theoretical risk of spreading tumor cells in the needle track while performing needle aspiration biopsy, there has been only one well-documented case of this complication reported with current fine needle techniques.[30,31] Most cases of needle track dissemination were reported following biopsies with larger Vin Silverman or Franklin Silverman needles.[32–34] In one report of tumor spread by needle aspiration,[35] the cytologic interpretation on which the diagnosis of dissemination was made has been questioned.[36]

The biopsy specimen from a transbronchial biopsy is larger than that obtained from fine needle aspiration. These larger specimens may allow the diagnosis of granulomas or acid-fast bacilli more readily than a needle aspiration specimen.

Based on the findings of this study and a review of the literature, the following diagnostic sequence is recommended for establishing a histologic diagnosis in patients with suspected Pancoast's tumor: First, a sputum sample should be obtained for cytologic analysis, followed by bronchoscopy. If cytologic analysis of the sputum is negative, transbronchial biopsy under fluoroscopic guidance should be performed during the bronchoscopy. If the cytologic studies and transbronchial biopsy findings are negative, then fine needle aspiration biopsy should be performed. If needle aspiration biopsy is initially performed to establish a diagnosis, bronchoscopy is still recommended to exclude endobronchial extension or a second primary tumor. A variety of conditions other than bronchogenic carcinoma can produce Pancoast's syndrome.[4,6–11] Therefore, a definitive tissue diagnosis is desirable prior to starting therapy with radiation followed by surgery. The diagnostic sequence outlined can safely provide a specific diagnosis in most cases.

Transbronchial needle aspiration has been introduced as another bronchoscopic technique for obtaining tissue specimens from peripheral masses.[37] This technique could potentially increase the value of bronchoscopy in this group of patients. However, the diagnostic yield and pneumothorax rate from transbronchial needle aspiration in patients with apical tumors has not yet been defined.

REFERENCES

1. Pancoast HK: Superior pulmonary sulcus tumor; tumor characterized by pain, Horner's syndrome, destruction of bone and atrophy of hand muscles. *JAMA* 1932; 99:1391–1461.
2. Paulson DL: Carcinomas in the superior pulmonary sulcus. *J Thorac Cardiovasc Surg* 1975; 70:1095–1104.
3. Hepper NG, Herskovic T, Witten DM, et al: Thoracic inlet tumors. *Ann Intern Med* 1966; 64:979–989.
4. Herbut PA, Watson JS: Tumor of the thoracic inlet producing the Pancoast syndrome; report of 17 cases and reviews of literature. *Arch Path* 1946; 42:88–103.
5. Shaw RR: Pancoast's tumor. *Ann Thorac Surg* 1984; 37:343–345.
6. Goldman SM, Fajardo AA, Naraval RC, et al: Metastatic transitional cell carcinoma from the bladder: Radiographic manifestations. *AJR* 1979; 132:419–425.
7. Omenn GS: Pancoast syndrome due to metastatic carcinoma from the uterine cervix. *Chest* 1971; 60:268–270.
8. Brenner B, Carter A, Freidin N, et al: Pancoast's syndrome in multiple myeloma. *Acta Haematol* (Basel) 1984; 71:353–355.
9. Eiben C, Indihar FJ, Hunter SW: Thoracic actinomycosis mimicking the Pancoast syndrome. *Minn Med* 1983; 66:541–544.
10. Vamos G, Papp A: Pancoast's syndrome and pulmonary tuberculosis. *Schweiz Z Tuberk* 1960; 17:423–430.
11. Aletras H, Papaconstantinou C: Pancoast's syndrome following an intrapleural rupture of a hepatic echinococcus cyst. *Scan J Thorac Cardiovasc Surg* 1982; 16:283–287.
12. Attar S, Miller JE, Satterfield J, et al: Pancoast's tumor: Irradiation or surgery? *Ann Thorac Surg* 1979; 28:578–586.
13. Johnson DH, Hainsworth JD, Greco FA: Pancoast's syndrome and small cell lung cancer. *Chest* 1982; 82:602–606.
14. Walls WJ, Thornbury JR, Naylor B: Pulmonary needle aspiration biopsy in the diagnosis of Pancoast tumors. *Radiology* 1974; 111:99–102.
15. Teixeira JP: Concerning the Pancoast tumor: What is the superior pulmonary sulcus? [editorial]. *Ann Thorac Surg* 1983; 35:577–578.
16. Paulson DL: Carcinoma in the superior pulmonary sulcus [editorial]. *Ann Thorac Surg* 1979; 28:3–4.
17. Pratt DS: Diagnosis of a thoracic inlet tumour by transbronchial biopsy. *Thorax* 1978; 33:803–805.
18. Komaki R, Roh J, Cox JD, et al: Superior sulcus tumors: Results of irradiation of 36 patients. *Cancer* 1981; 48:1563–1568.
19. Hadley JL, Krumpe PE: Incidence of unanticipated findings from fiberoptic bronchoscopy in patients with solitary pulmonary nodules. *Am Rev Respir Dis* 1984; 129:A59 (Annual Meeting Abstracts).
20. Westcott JL: Direct percutaneous needle aspiration of localized pulmonary lesions: Results in 422 patients. *Radiology* 1980; 137:31–35.
21. Landman S, Burgener FA, Lim GH: Comparison of bronchial brushing and percutaneous needle aspiration biopsy in the diagnosis of malignant lung lesions. *Radiology* 1975; 115:275–278.
22. Zavala DC, Bedell GN: Percutaneous lung biopsy with a cutting needle. An analysis of 40 cases and comparison with other biopsy techniques. *Am Rev Respir Dis* 1972; 106:186–193.
23. Lalli AF, McCormack LJ, Zelch M, et al: Aspiration biopsies of chest lesions. *Radiology* 1978; 127:35–40.
24. Sinner WN: Transthoracic needle biopsy of small peripheral malignant lung lesions. *Invest Radiol* 1973; 8:305–314.
25. Herman PG, Hessel SJ: The diagnostic accuracy and complications of closed lung biopsies. *Radiology* 1977; 125:11–14.
26. Fulkerson WJ: Fiberoptic bronchoscopy. *N Engl J Med* 1984; 311:511–515.
27. Herf SM, Suratt PM, Arora NS: Deaths and complications associated with transbronchial lung biopsy. *Am Rev Respir Dis* 1977; 115:708–711.
28. Pereira W, Kovnat DM, Snider GL: A prospective cooperative study of complications following flexible fiberoptic bronchoscopy. *Chest* 1978; 73:813–816.
29. Ellis JH: Transbronchial lung biopsy via the fiberoptic bronchoscope. Experience with 107 consecutive cases and comparison with bronchial brushing. *Chest* 1975; 68:524–532.
30. Sinner WN, Zajicek J: Implantation metastasis after percutaneous transthoracic needle aspiration biopsy. *Acta Radiolog* [Diagn] (Stockh) 1976; 17:473–480.
31. Sinner WN: Complications of percutaneous transthoracic needle aspiration biopsy. *Acta Radiolog* [Diagn] (Stockh) 1976; 17:813–828.
32. Allbritten FF, Nealon T, Gibbon JH Jr, et al: Symposium on safeguards in surgical diagnosis: Diagnosis of lung cancer. *S Clin North America* 1952; 32:1657–1662.
33. Dutra FR, Geraci CL: Needle biopsy of the lung. *JAMA* 1954; 155:21–24.
34. Wolinsky H, Lischner MW: Needle track implantation of tumor after percutaneous lung biopsy. *Ann Intern Med* 1969; 71:359–362.
35. Berger RL, Dargan EL, Huang BL: Dissemination of cancer cells by needle biopsy of the lung. *J Thorac Cardiovasc Surg* 1972; 63:430–432.
36. Naylor B: Dissemination of cancer cells after needle biopsy of the lung. *J Thorac Cardiovasc Surg* 1972; 64:324.
37. Shure D, Fedullo PF: Transbronchial needle aspiration of peripheral masses. *Am Rev Respir Dis* 1983; 128:1090–1092.

See also pp 320–322.

Oral ciprofloxacin in the treatment of 14 patients with bacterial pneumonia

CHRISTINE M. WOLLSCHLAGER, MD; SUHAIL RAOOF, MB; FAROQUE A. KHAN, MB

ABSTRACT. Ciprofloxacin is a new synthetic quinolone antibiotic with broad, in vitro antibacterial activity. As part of a clinical trial, we treated 14 patients with bacteriologically proven pneumonia with oral ciprofloxacin in a dose of 750 mg twice daily. Nine men and five women with a mean age of 46 years received the drug for an average of 11.5 days. Thirteen patients had underlying lung disease or other predisposing factors for pneumonia. Cure was achieved in 12 of 14 patients (86%). The two clinical failures included one patient with inoperable lung cancer and one with suspected anaerobic empyema. None of the 17 bacterial isolates obtained from sputum from these patients showed in vitro resistance to ciprofloxacin; however, three isolates showed intermediate zones of sensitivity (two isolates of *Streptococcus pneumoniae* and one isolate of α-hemolytic streptococcus). Persistence of the pathogenic organism occurred in only two patients, involving *S pneumoniae* and *Serratia marcescens*. Adverse reactions were mild and self-limiting. Although the study population was small, the results show that ciprofloxacin is a safe, effective therapy for the treatment of bacterial pneumonia and has, in addition, the advantages of twice daily oral dosing and broad in vitro antibacterial activity.

(NY State J Med 1987; 87:330–333)

Ciprofloxacin is a new synthetic quinolone antibiotic derived from nalidixic acid.[1] The drug is undergoing clinical evaluation as a parenteral and oral antimicrobial agent.[2-4] Ciprofloxacin has shown in vitro activity against gram-negative bacteria that are resistant to multiple antibacterial agents, such as *Pseudomonas aeruginosa*, as well as activity against methicillin-resistant *Staphylococcus aureus*.[5-7] Another potential advantage of this drug is its extended spectrum of activity, covering gram-positive and gram-negative aerobic bacteria. Although its clinical relevance remains unknown, ciprofloxacin has also demonstrated in vitro activity against unusual organisms such as chlamydia, *Mycobacterium avium-intracellulare*, and *Legionella*.[7-11] Oral ciprofloxacin is 100 times more active than nalidixic acid, and primary resistance to ciprofloxacin is rare.[3-8]

These properties combine to make ciprofloxacin an attractive antimicrobial agent, particularly in the treatment of lower respiratory tract infections. We are evaluating the efficacy and safety of ciprofloxacin in patients with bacterial pulmonary infections. In this paper, we report the results of oral ciprofloxacin therapy in 14 patients with community- or hospital-acquired pneumonia.

PATIENTS AND METHODS

Patient Population. We reviewed the medical records of 14 patients treated for bacterial pneumonia with oral ciprofloxacin at Queens Hospital Center, Jamaica, NY, between September 1984 and June 1985. The diagnosis of bacterial pneumonia was established on clinical evidence (fever, productive cough, abnormal breath sounds, leukocytosis, and a chest film consistent with pneumonia) and microbiologic confirmation (from a sputum specimen obtained within 24 hours of starting treatment). No patient had received antibiotics in the previous 48 hours, and all patients gave informed written consent obtained in accordance with the institutional Human Subjects Research Committee. Patients were specifically excluded from receiving ciprofloxacin if they were less than 18 years of age, had a psychiatric illness, were pregnant, or were allergic to quinolones (nalidixic acid). In addition, patients were excluded from the trial if they were deemed so unstable or toxic that they required intravenous therapy.

Drug Administration. Ciprofloxacin was supplied by Miles Pharmaceuticals, West Haven, Conn, as 750 mg tablets. It was administered twice daily on an empty stomach. Care was taken to insure that no antacids, especially those containing magnesium, were given with the study drug.[12]

Data Collection. The following information was collated from the medical records.

- A daily record of fever, cough, sputum production, hemoptysis, dyspnea, and other pertinent clinical history and the results of daily physical examinations were recorded.
- The results of the complete blood count, and liver and renal function tests done prior to and during the therapy were tabulated.
- The results of pre- and post-therapy sputum cultures were collected. Sputum was obtained in all patients by spontaneous expectoration or was induced by ultrasonic nebulization with 5% saline. Gram's staining was performed; only specimens that contained fewer than 25 squamous epithelial cells and 25 or more neutrophils per low power field were accepted for culture analysis. Specimens were inoculated onto trypticase soy agar supplemented with 5% sheep blood, chocolate agar, and MacConkey agar plates and were incubated at 35°C in 5% carbon dioxide for 18–24 hours. Disc diffusion sensitivities were performed using the standard Kirby-Bauer technique.[13] The interpretative criteria for ciprofloxacin zone size were: resistant ≤ 15 mm, intermediate 16–20 mm, and sensitive ≥ 21 mm. Minimum inhibitory concentrations of antibiotics were

---

From the Division of Pulmonary Medicine, Department of Medicine, Queens Hospital Center Affiliation of the Long Island Jewish Medical Center, and Health Sciences Center, State University of New York at Stony Brook.

Address correspondence to Dr Khan, Chairman, Department of Medicine, Nassau County Medical Center, 2201 Hempstead Tpke, East Meadow, NY 11554.

determined by the agar dilution technique as outlined by the National Committee for Clinical Laboratory Standards (NCCLS) and with the use of two-fold dilutions of the antibiotics. The minimum inhibitory concentration (MIC) was defined as the lowest concentration of antibiotic at which there was no evidence of visible growth.

Definition of Efficacy. We evaluated therapeutic efficacy only in patients who received therapy with ciprofloxacin for at least five days. Cases in which the drug was discontinued before five days because of worsening of the patient's condition were considered a drug failure. The clinical and bacterial efficacy of ciprofloxacin was evaluated as follows.

Clinical response. We categorized the patients' clinical response to antibiotic therapy for their pneumonia in one of two ways. Cure was marked by the disappearance of signs and symptoms of infection, normalization of temperature, clearing of the chest film, and decrease of an elevated leukocyte count. Failure was defined as insufficient lessening of the signs and symptoms and objective evidence of infection to qualify as improvement.

Bacteriologic outcome. Bacteriologic response was graded as eradication, colonization, or persistence. Eradication was said to be achieved when pathogenic organisms were absent at the end of therapy. Colonization was noted when a new organism was isolated at the end of therapy. Persistence was said to occur when the causative organism was still present at the end of therapy.

RESULTS

Nine men and five women with pneumonia were treated with ciprofloxacin. The pneumonia was community acquired in 11 patients and nosocomial in three patients. The average age of these patients was 46 years (range, 26–72 years). Eleven patients had one or more of the following risk factors for pneumonia: chronic obstructive pulmonary disease (COPD) (four patients), potential aspiration due to alcohol or drug use (seven patients), and bronchogenic carcinoma with a permanent tracheostomy (one patient) (Table I). All patients were considered clinically and hemodynamically stable and thus were eligible for oral antibiotic therapy. The majority of patients had a segmental or lobar consolidation and temperature ranging from 100°F to 103°F. They received ciprofloxacin in an oral dose of 1,500 mg per day for an average of 11.5 days (range, 5–19 days).

Clinical Response. Oral ciprofloxacin successfully cured bacterial pneumonia in 12 of 14 patients (86%). The average time for defervescence to <99°F was 4.8 days (Table I). The total white blood count in the cured group was >10,000/mm^3 in nine patients and became normal in seven of nine patients within 7–12 days. In 11 of 14 patients, the radiographic abnormality was a segmental or single lobe infiltrate. Of the 12 patients ultimately cured, 42% showed significant radiographic clearance in two weeks and all radiographs were cleared in three weeks. The two patients who did not respond to ciprofloxacin (nos. 6, 13) showed clinical deterioration during the antibiotic therapy.

TABLE I. Clinical Characteristics of Patients Treated with Ciprofloxacin

Patient No.	Age/Sex	Predisposing Factors	Organism	Where Acquired	Entry Temp(°F)	Days in Which Afebrile	Entry X-ray	X-ray at 2 or 3 Weeks	Clinical Outcome	Adverse Reactions
1	50/F	Methadone maintenance	*S pneumoniae*	Community	101.4	8	Single lobe[†]	Significantly improved	Cure, slow improvement	Diarrhea
2	67/M	Chronic obstructive pulmonary disease (COPD)	*E coli*	Hospital	100.2	3	Single lobe[**]	Cleared	Cure	—
3	72/M	COPD, congestive heart failure, alcohol abuse	*S pneumoniae* *H parainfluenzae*	Community	99.8	9	Single lobe[†]	Significantly improved	Cure	—
4	34/F	Alcohol abuse, heroin addict	*P mirabilis* *K pneumoniae*	Community	101.8	2	Bilobar[†]	Partial clearing	Cure	Nausea
5	26/F	Bronchial asthma	*E cloacae*	Hospital	100.4	5	Single lobe[‡]	Cleared	Cure	Nausea, pruritis
6	33/M	Bronchogenic carcinoma, tracheostomy	*S marcescens*	Hospital	99.8	Remained febrile	Single lobe[†]	Unchanged	Failure	Bitter taste
7	71/M	COPD, bronchiectasis	*H parainfluenzae* *P aeruginosa* *S pneumoniae*	Community	100.4	3	Single lobe[†]	Cleared	Cure	Palpitations, tremor
8	63/F	COPD	*H influenzae*	Community	101.6	4	Single lobe[‡]	Cleared	Cure	Difficulty swallowing large pills
9	36/M	None	*S pneumoniae*	Community	104	3	Single lobe[†]	Cleared	Cure	Diarrhea
10	44/F	Bronchial asthma	*H parainfluenzae*	Community	103	3	Single lobe[†]	Cleared	Cure	—
11	32/M	Alcohol abuse, bronchiectasis	β-streptococcus	Community	101.8	7	Single lobe[†]	Significantly improved	Cure	—
12	33/M	Alcohol abuse	*S pneumoniae*	Community	101.4	8	Bilobar[**]	Significantly improved	Cure, slow improvement	Diarrhea
13	48/M	Alcohol abuse	Anaerobes[*]	Community	102.4	Remained febrile	Bilobar[†]	Pleural effusion, abscess	Failure	—
14	33/M	Alcohol abuse	α-streptococcus	Community	100.6	4	Lobar[**]	Significantly improved	Cure	—

[*] No organism isolated from sputum or empyema fluid.

[†] Alveolar filling process.

[**] Patchy pneumonitis.

[‡] Segmental consolidation.

Adverse reactions to ciprofloxacin were mild and did not interfere with completion of therapy (Table I). Diarrhea occurred in 21% of patients and nausea in 14%. In one patient (no. 7), theophylline toxicity was suspected based on the patient's symptoms of palpitations and tremors. His serum theophylline level was 10.9 μg/mL before ciprofloxacin therapy and 15 μg/mL at one point during the treatment. Crystalluria was noted in one patient.

Bacteriologic Outcome. The etiology of the pneumonia was determined by Gram's stain and cultures of sputum in 13 of 14 patients; 17 isolates were obtained (Table II). None of these patients was found to have bacteremia. The most common organisms were pneumococci, accounting for 29% of isolates, and *Hemophilus* species, accounting for 24% of the isolates. One patient (no. 13) had an anaerobic pneumonia diagnosed on the basis of history, clinical examination, Gram's stain, and presence of foul smelling pleural fluid on thoracentesis from which no organism could be isolated. Patient no. 14 had a heavy growth of a partially resistant α streptococcal species that was isolated from multiple sputum specimens. Although not usually considered a cause of pneumonia, this organism was believed to be pathogenic in view of clinical and radiographic evidence of pneumonia and the organism's unusual growth and sensitivity patterns. Patients with COPD or asthma had the following isolates: *Hemophilus* sp (four patients), *Streptococcus pneumoniae* (two patients), and other gram-negative bacteria (three patients). The organisms implicated in the three nosocomial pneumonias were *Escherichia coli*, *Enterobacter cloacae*, and *Serratia marcescens*. No isolates were resistant to ciprofloxacin at the beginning of the trial, but two isolates of *Streptococcus pneumoniae* and one α-streptococcus showed intermediate zones of sensitivity. Minimum inhibitory concentrations (MICs) were available on 15 of 17 isolates. In general, MICs were higher for gram-positive organisms, and pneumococci in particular. Gram-negative bacteria, particularly *Hemophilus* spp and *E coli*, had extremely low MICs.

Bacterial eradication was achieved in 11 of 13 patients (85%). Persistence of pneumococcus and *S marcescens* occurred in patients 1 and 6, respectively. The isolate from patient no. 6 developed intermediate resistance to ciprofloxacin (on disc diffusion), and its MIC increased from 0.03 μg/mL before therapy to 0.25 μg/mL after therapy. Finally, colonization with a new organism, *Klebsiella pneumoniae*, that was sensitive to ciprofloxacin, only developed in patient no. 1 (8%). This occurred on day 17 of therapy for treatment of a community-acquired pneumococcal pneumonia.

DISCUSSION

We found oral ciprofloxacin to be an effective therapy for bacterial pneumonia as evidenced by a clinical cure rate of 86%. The drug was well tolerated by a diverse group of patients and resulted in prompt clinical and radiographic improvement. In two patients the pneumonia did not respond to ciprofloxacin therapy; however, their illnesses were complex. Patient no. 13 had a presumed anaerobic aspiration pneumonia that was diagnosed on the basis of previous similar episodes, a history of alcohol abuse, failure to isolate any organisms from sputum or purulent empyema fluid, and radiographic evidence of abscess formation within five days. Patient no. 6 had bronchogenic carcinoma and a permanent tracheostomy. In the preceding three months, he had received multiple antibiotics for nosocomial infections. He did not respond to 18 days of treatment with ciprofloxacin, and *S marcescens* persisted in his sputum. As noted, this isolate was sensitive to ciprofloxacin at the beginning of the trial and became partially resistant after therapy.

In this hospital, the three most common gram-negative bacteria seen during exacerbations of COPD are *Hemophilus*, *Pseudomonas*, and *Klebsiella*. All of these organisms were highly sensitive to ciprofloxacin, with MICs $<$ 0.03 μg/mL in all cases. Thus, ciprofloxacin can be expected to be very efficacious in the infected patient with COPD. In this study, pretreatment in vitro sensitivity tests showed all bacterial isolates to be sensitive to ciprofloxacin, except *S pneumoniae* and α-streptococcus, which showed intermediate sensitivity. This broad spectrum in vitro sensitivity is well described with ciprofloxacin.[5-7] The MICs available for *S pneumoniae* were 1.0 μg/mL in two isolates and 4.0 μg/mL in two other isolates. Despite these relatively high MICs, eradication of *S pneumoniae* from the sputum occurred in four of five cases (80%), and, more importantly, all these patients were judged to be cured based on their definite clinical and radiographic improvement. Studies with enoxacin and other quinolone antibiotics have shown that concentration of this drug in the bronchial mucosa was 40 times higher than the plasma concentration, and was much higher than the MICs for common respiratory pathogens.[14] Thus, serum concentrations of enoxacin, and perhaps of ciprofloxacin as well, may markedly underestimate the local lung concentrations. This potential

TABLE II. Bacteriologic Data from Sputum Isolates

		Pretreatment in vitro Tests				Posttreatment Culture Results	
Organism	No. Isolates	Sensitive	Intermediate	Resistant	MICs(μg/mL)*	Eradication	Persistence
S pneumoniae	5	3	2	—	1.0, 1.0, 4.0, 4.0	4	1
β-hemolytic streptococcus	1	1	—	—	.5	1	—
α-streptococcus	1	—	1	—	.5	1	—
Hemophilus spp	4	4	—	—	<.008 <.008 <.008	4	—
P aeruginosa	1	1	—	—	.03	1	—
K pneumoniae	1	1	—	—	.015	1	—
S marcescens	1	1	—	—	.03	—	1
E cloacae	1	1	—	—	.008	1	—
E coli	1	1	—	—	<.008	1	—
P mirabilis	1	1	—	—	.015	1	—

* MICs = minimum inhibitory concentrations (not available in one isolate each of *S pneumoniae* and *E coli*).

concentration gradient could explain the good clinical response to ciprofloxacin in infections with gram-positive organisms.

There were few adverse reactions to ciprofloxacin in this small group of patients. Mild, self-limiting side effects did not necessitate discontinuance of the drug in any patient. Difficulty in swallowing the large tablet was a manageable complaint.

The therapeutic success encountered in this clinical study is similar to favorable results published by other investigators.[2-4] This study supports a significant clinical potential for ciprofloxacin in the treatment of community- and hospital-acquired gram-negative bacterial pneumonia. The role of ciprofloxacin in gram-positive bacterial pulmonary infections requires further study. However, the clinical results in the seven patients with gram-positive infections described here were encouraging—including the cures achieved in the five patients with *S pneumoniae* infection. This excellent clinical response, as well as the lack of serious toxicity, broad spectrum antibacterial activity, and the inherent advantage of a twice daily dosage, are added positive features that combine to make ciprofloxacin an important antibiotic of the future.

Acknowledgment. The authors thank the staff of the Department of Microbiology, Queens Hospital Center, for their involvement in this study, and Miss Terry Ishman for her expert secretarial assistance.

REFERENCES

1. Riesenberg DE: New quinolone generation active over wide spectrum. *JAMA* 1986; 255:176.
2. Eron LJ, Harvey L, Hixon DL, et al: Ciprofloxacin therapy of infections caused by Pseudomonas aeruginosa and other resistant bacteria. *Antimicrob Agents Chemother* 1985; 28:308-310.
3. Ramirez CA, Poran JL, Meja CR, et al: Open, prospective study of the clinical efficacy of ciprofloxacin. *Antimicrob Agents Chemother* 1985; 28:128-132.
4. Ernst JA, Sy ER, Lucca H, et al: Ciprofloxacin in the treatment of pneumonia. *Antimicrob Agents Chemother* 1986; 29:1088-1089.
5. Fass RJ: In vitro activity of ciprofloxacin (Bay 0 9867). *Antimicrob Agents Chemother* 1983; 24:568-574.
6. Wise R, Andrews JM, Edwards LJ: In vitro activity of Bay 09867, a new quinolone derivative, compared with those of other antimicrobial agents. *Antimicrob Agents Chemother* 1983; 23:559-564.
7. Van Caekenberghe DL, Pattyn SR: In vitro activity of ciprofloxacin compared with those of other new fluorinated piperazinyl-substituted quinolone derivatives. *Antimicrob Agents Chemother* 1984; 25:518-521.
8. Smith SM: In vitro comparison of A-55619, A-56620, amifloxacin, ciprofloxacin, enoxacin, norfloxacin and ofloxacin against methicillin-resistant Staphylococcus aureus. *Antimicrob Agents Chemother* 1986; 29:325-326.
9. Heessen FW, Muytjens HL: In vitro activities of ciprofloxacin, norfloxacin, pipemidic acid, cinoxacin, and nalidixic acid against Chlamydia trachomatis. *Antimicrob Agents Chemother* 1984; 25:123-124.
10. Fenlon CH, Cynamon MH: Comparative in vitro activities of ciprofloxacin and other 4-quinolones against *Mycobacterium tuberculosis* and *Mycobacterium intracellulare. Antimicrob Agents Chemother* 1986; 29:386-388.
11. Bure A, Desplaces N, Pangon B, et al: In vitro activity of ciprofloxacin, perfloxacin and ofloxacin against Legionella. 14th International Congress of Chemotherapy, Kyote, Japan, June 23-28, 1985, pp 37-74.
12. Ratcliffe NT, Smith JT: Effects of magnesium on the activity of 4-quinolone antibacterial agents. *J Pharm Pharmacol* 1983; 35:61.
13. Bauer AW, Kerby WM, Sherris JC, et al: Antibiotic susceptibility testing by standardized single dose method. *Am J Clin Pathol* 1966; 45:493-496.
14. Marlin GE, Braude PD, Whelan AJ, et al: The effect of drug pharmokinetics in treatment of lower respiratory tract infections. 19th International Congress of Infectious and Parasitic Diseases, Munich, Germany, July 20-26, 1986, p 1190 [abstract].

FROM THE LIBRARY

THE ROLE OF THE BRONCHOSCOPIST IN THE DIAGNOSIS AND TREATMENT OF DISEASES OF THE BRONCHIAL PULMONARY TRACT

Because of the demands of the rapid progress of medical science during the past decade, bronchoscopy has come to play an important role in the diagnosis and treatment of bronchial pulmonary diseases. To quote that famous teacher of bronchoscopy, Chevalier Jackson: " 'Look and see' is the order of the day. In every accessible region of the body, the aid of direct vision is called upon to contribute to diagnosis and treatment."

In the diagnosis of thoracic diseases, the internist by means of external inspection, percussion, and auscultation, usually obtains sufficient information to establish a tentative diagnosis. His findings may be further corroborated by the roentgenologist, who by means of fluoroscopic and x-ray examination has an opportunity, as it were, to look through the chest. But it remains for the bronchoscopist, by means of direct visual inspection, to look inside the bronchial tree and observe the underlying pathologic condition. It becomes evident, then, that when there exists such a close association between the internist, the roentgenologist, and the bronchoscopist, the diagnosis of bronchial pulmonary conditions is greatly facilitated.

Aside from the removal of aspirated foreign bodies in lungs, for which no procedure other than bronchoscopy is worthy of a moment's consideration, the profession at large has not realized that the greatest value of the bronchoscope is in diagnosis. A recent statistical survey of the patients admitted to several of the large bronchosopic clinics in this country revealed that only 2 percent of the admissions were for the removal of foreign bodies. As Dr. Howard Lilienthal, one of the great teachers of thoracic diseases, has aptly said: "The scope of bronchoscopy has rapidly widened from the mere extraction of foreign bodies to the diagnosis and treatment of many pulmonary lesions, such as abscess of the lung, tumor of the lung, bronchial narrowing, bronchial ulceration, postoperative atelectasis, and as an aid to the thoracic surgeon in the localization of thoracic diseases . . ."

ARTHUR Q. PENTA, MD
(*NY State J Med* 1943; 43:953-960)

Arthur Donaldson Smith, MD

Physician, explorer, naturalist, and diplomat—Part IV*

PASCAL JAMES IMPERATO, MD

In 1897 Arthur Donaldson Smith, MD, was 31 years of age. As a handsome and wealthy young physician he was viewed by the ladies of Philadelphia's high society as a very eligible bachelor. His sister's nephew Henry Pleasants comments on this in some detail:

> Uncle Donnie was a great favorite with the ladies. At least they were always discussing him.... They were always wondering why he did not "settle down".... They seemed to think that it just wasn't right for a good looking young bachelor to be on the loose like that when he was really rich.... Uncle Donnie didn't seem to feel that way at all. He was a man's man every inch of the way. If he wanted to go to a prize-fight he just went. If he wanted to go down south for the quail shooting in the winter, his valet packed his bags and attended to all the little details for him. Nobody bothered him about coming home.... Aunt Agnes [Smith's sister, Agnes Spencer] was the one who did the most fussing. She insisted on dragging poor Uncle Donnie around to teas and balls; and having young ladies out to the big house at Kaolin for weekend parties when he was there. My young cousins told me that he spent most of his time with them, instead of around the house with the guests on these occasions. Of course he was perfectly nice and polite to company.[1]

Smith clearly had no intentions at this point in his life of settling down to domestic life and a medical practice. As Pleasants says: "... With all that medical education, he could have had things pretty much his own way so far as practice was concerned." [1] Pleasants' description of Smith at this time shows that he was a complex man whose surface appearances gave no hint of his determination, perseverance, and courage and his ability to withstand incredible physical hardships in remote, far off corners of the world. Pleasants describes Smith as follows:

> His appearance was not suggestive of heroics. When I just met him, I thought he was insipid. He was well below medium height; of slight physique, and so painfully diffident that he seldom raised his voice above a low monotone. His handshake gave you the creeps, for his fingers were so long and delicate you were afraid you would break something if you gave an honest squeeze. His face was really handsome, however, high, intelligent forehead, slightly hooked thin nose, deep-set luminous blue eyes under shaggy eyebrows, and a firm, square jaw. Besides that, he was pleasant and friendly. When he smiled, his whole face lit up and warmed you like a birch-log on a cold night. The fact that his suits were tailor made, his shoes and luggage of the finest leather obtainable, and that he always smelled of really good cigars, stamped him as a real gentleman in spite of his lack of masculine ruggedness.[1]

Boyhood memories are apt to capture surface qualities that are unleavened by mature insights into the true nature of a man. It is not surprising, therefore, that many of Smith's relatives who read Pleasants' 1946 article were not pleased with it.[2]

The man whom Pleasants recalls as lacking masculine ruggedness set sail from San Francisco for China in March of 1897, a year after his return from his expedition to Lake Rudolf. It was his plan to travel through the Khingan Mountains of Manchuria and Inner Mongolia, then one of the most remote regions of the Far East.

JOURNEY THROUGH THE KHINGAN MOUNTAINS

Smith made this trip in the company of two other Philadelphians, George L. Farnum and J. Edward Farnum. They left Peking on March 19, 1897, and headed north toward Dolon Nor (Tollunnoerh), which lay on the western side of the Khingan Mountains. Their trip through the mountains lasted over two months, they arriving at the northern Manchurian town of Tsitsihar (now known as Lungkiang) on August 9, 1897.[3] From there they went on to Vladavostok in Russia. Smith, however, does not describe this last leg of the journey, saying: "I will pass over the rest of our journey, since we had now reached a town where there are telegraph stations." [3] This journey differed from Smith's expedition to Lake Rudolf in that it was not undertaken primarily for the purpose of geographic exploration. Referring to it in a letter to J. Scott Keltie of the Royal Geographical Society, Smith said: "... the Corea trip [he refers to it this way in his early correspondence on the subject] will be very cheap and will be for sport and adventure. . . ." [4] Yet, being the naturalist he was, Smith collected over 100 different moths and butterflies on this trip and many birds and other natural history specimens.[3] He and the Farnums seem to have kept an open mind about their ultimate destination, for he wrote to J. Scott Keltie on May 20, 1897, from San Ho, some 15 miles out of Peking, saying that: "... [we] will probably come out at Vladavostok or else make a loop and return to some point on the Yellow Sea ..." [5] Not surprisingly, Smith often refers in his correspondence to his journey as the Corea trip.

---

* Parts I, II, and III of this article were published in the February, March, and April 1987 issues, respectively; part V will appear in a subsequent issue. Author's acknowledgments will appear at the end of the last installment.

Address correspondence to Dr Imperato, Editor, *New York State Journal of Medicine*, 420 Lakeville Rd. Lake Success, NY 11042.

Even though he was out in China enroute to Manchuria, Smith's mind was on Lake Rudolf and the Omo. In this same letter he said to Keltie:

> I shall be most anxious to hear the result of the English mission to Abyssinia; the result of Bottego's journey and whether any explorers are doing or are trying to do the Omo district, the country to the west of Rudolf, and the southern Borana plains. You would do me a great favor if you write me on these points about August first and I will find the letter when I come out in September.[5]

Smith was referring to the Italian expedition of Vittorio Bottego which, unknown to him, had in fact settled the issue of the Omo River's course and the source of Lake Rudolf.[6] He was clearly aware of the churning political events in the area and the contest between the French and the British for Ethiopian sympathies because he refers to James Rennell Rodd's mission to Addis Ababa in April 1897.[7(pp112-191),8(p258)] Rodd (Fig 1), accompanied by Reginald Wingate, was sent to Ethiopia by the British to counteract French influence there and, as Sanderson succinctly observes, "to allay suspicions that Kichener's operations might ultimately be directed against Ethiopia." [8(p258)] General Sir Horatio Herbert Kichener was at the time beginning Britain's campaign against the Mahdist state in the Sudan in his capacity as Sirdar of the Egyptian army. This early effort was ostensibly undertaken to divert the Mahdist military forces who were pressing Britain's ally, Italy, in the eastern Sudan.[8(p245)] Ultimately, in 1898, British troops formally joined him to reoccupy the Sudan. The main impetus for this 1898 military effort was the threat of French occupation of the Upper Nile, the lifeline of Britain's strategic protectorate, Egypt. A French pincer movement on the Nile from Ethiopia in the east and from equatorial Africa in the west was already underway since 1896 and the British knew it. The last thing the British wanted was for Emperor Menelik of Ethiopia, with his sizable military force which had defeated an Italian army at Adowa on March 1, 1896, to enter the picture as an ally of either the French or the Mahdists.[9]

FIGURE 1. Sir James Rennell Rodd, who led a British mission to Ethiopia in 1897 (reproduced from *Social and Diplomatic Memories 1884–1893*, London, Edward Arnold & Co, 1922) (courtesy of Lord Rennell of Rodd).

In his May 20, 1897, letter to Keltie, Smith inquired if there were any areas in the Far East worth exploring and "... if the town of Lassa has been visited by a European and whether there is any country near Lassa worth mapping." [5] He told Keltie that he planned to spend most of the winter of 1897–1898 in the Far East traveling for pleasure to Borneo, Java, and India. Exhibiting keen insights into the French-British contest for the Nile, he said, "... Africa will hold till the spring and the conditions will not have changed much ..." [5] However, he asked Keltie to keep him informed about the situation and expressed the hope that he could return to Lake Rudolf by the spring of 1898.[5]

During the first phase of the trip to the Khingan Mountains, Smith and the Farnums passed through well-populated areas until they reached Dolon Nor, a large trading center between Mongolia and China, roughly 250 miles north of Peking.[3] Enroute to Dolon Nor, they had to be on the alert for brigands who were armed with Mauser rifles.[3] They were, according to Smith, "a source of constant dread to the inhabitants and to caravans...." [3] However, they rarely attacked Europeans, especially those in well-armed caravans. Before reaching Dolon Nor, Smith and the Farnums met Dr Eugen Von Cholnoky, professor of geography at the University of Budapest.[3] On May 31, Smith and the Farnums trotted on their ponies across the high Mongolian plateau and entered Dolon Nor, where they spent ten days.[3] Smith provides no description of Dolon Nor in his account of the trip. However, Dolon Nor lies on the western flank of the Khingan range, roughly at their mid-point. Smith and the Farnums crossed the range to get to Dolon Nor and then traveled up the northern half of the range for the next two months, from June 10 to August 9, 1897.

In his account of the trip in the *Geographical Journal* Smith provides many details about the topography, the people, and the political situation as well as the fauna and flora. At Long Shan, where there was a hot sulphur spring, they met a Mongol prince, Nau Hang Wang, "... a slender man, forty years of age, with very bright, intelligent eyes and aquiline features." [3] Wang ruled over 11 Mongolian provinces and was, according to Smith, "... held in high esteem at the court of Peking." [3] Smith and the Farnums exchanged visits with the prince and the latter asked Smith for advice on increasing the efficiency of his troops in keeping down the robbers.[3] Smith advised that efficiency could be improved if the soldiers were paid enough so "that their existence should not depend upon bribery." [3] The prince listened but then changed the subject!

Moving in a northeasterly direction between peaks that rose up to 7,000 feet, Smith and the Farnums passed small camps of Mongol nomads and came across remnants of the Great Wall, built chiefly of mud.[3] Smith and the Farnums kept busy collecting natural history specimens along the way.

> Until we arrived in the eastern slope of the mountain range we saw few butterflies, but now we were kept very busy with our hand-nets till we reached the end of our journey. It was different with the birds, for, although we shot about forty different varieties between Peking and Dolon Nor, we were only able to increase our collection by a dozen or more species before reaching the Amur. Of flowering plants there was an infinite variety throughout the entire journey, but these were especially beautiful on the high plateaus, where they made a display such as I have never before witnessed.[3]

No record has been uncovered as to when Smith and the Farnums arrived in Vladavostok. However, by October 8, 1897, Smith was in Shanghai at the Astor House, from where he wrote J. Scott Keltie at the Royal Geographical Society. Smith and the Farnums had covered some 1,200 miles between Peking and Vladavostok via the Khingan range. Smith summarized the purpose of the trip when he wrote:

> The trip was undertaken for sport and the pleasure inherent upon a journey through a country unknown to oneself and therefore I did not take instruments for any proper geographical work. Together with this fact our rate of speed was too great to allow more than the roughest sort of work to be done even if instruments were at hand.[10]

Yet Smith did have a pocket compass and an aneroid with him and made accurate enough readings to draw up a good map of the part of the Khingan range he traversed. Smith and the Farnums were among the few Europeans to have traveled the entire length of the middle Khingan range up to that time. Smith makes a cogent point when he says: ". . . the map . . . would be of value to the next man . . . I am not aware of the existence of any map of the Khingan range that is at all correct. . . ." [10] On November 10, 1897, Keltie responded to Smith's letter and told him he would be pleased to publish the map and the report of the trip which he did the next year.[11,3]

PLANS FOR RETURNING TO AFRICA

In his letter of October 8, 1897, to J. Scott Keltie from Shanghai, Smith leaves no ambiguity about his being aware of the exploratory activity that had been going on around Lake Rudolf:

> I read that Prince Henry of Orleans is going to try for it and that Major Macdonald has gone up the Juba and that Captain Bottego has been killed in "Gobir" but still I believe that I could unravel much of the secrets to the west of Rudolf.[10]

Although he does not explicitly say it, Smith was aware that the issue of the Omo had been resolved. That then left only the area between the lake and the Nile to be explored. He wrote at great length about the need for financial assistance from the Royal Geographical Society to cover a two-year trip which by his estimates would cost £4,500. He asked Keltie to give him some unofficial idea if the council of the society were prepared to cover two-thirds of the cost. In the event they were, Smith was prepared to go to Aden, cross

> . . . to Berbera for a week, find out all about the cost of camels, etc. inquire if I could get most of my old boys (a very important item) and make arrangements so that in case I am going into the interior after making a formal application to the Council of the R.G.S., I shall be able to write ahead for all necessary supplies, such as camels.[10]

Keltie was in no position to speak for the council. Not surprisingly, given the complex political webs that now encompassed the Upper Nile, and his unwillingness to be the bearer of bad news, he in effect advised Smith to be his own judge of the situation.

> I think you might when at Aden, go to Berbera, and from there see what's being done and what you could do best. You might then come here . . . consult about the matter. I am quite sure the Council are still disposed to help you, and if you found it would not be available to attempt the country west of Lake Rudolf, probably some other sphere could be found where you could do your work.[11]

Keltie was obviously guarded as to the council's supporting Smith's trip between Lake Rudolf and the Nile. He goes so far as to suggest that the council might even find some other place for Smith to explore.

British attempts to destroy the Mahdist state in the Sudan were rapidly moving forward to late 1897 at the time Keltie wrote his letter. For Britain had to topple this indigenous state in order to gain access to the Upper Nile before the French moved in from Ethiopia in the east and equatorial Africa in the west. British diplomatic efforts in Ethiopia were aimed at both neutralizing French influence and discouraging a Mahdist-Ethiopian military alliance. A scientific expedition moving along the southern fringes of Ethiopia to Lake Rudolf and over to the Nile was seen by the British government as jeopardizing these delicate plans. Emperor Menelik (Fig 2), like many indigenous African rulers, had previous experience with so-called scientific expeditions which were often political missions in disguise. Despite the purity of Smith's endeavor, there was a good possibility that it would be misinterpreted by the Ethiopians as a political move along their country's southern fringes. This in turn might rouse them into a self-defensive alliance with the Mahdist state, stall British advances up the Nile, and leave the upper reaches of the river open for seizure by the French. The timing of Smith's plans could not have been worse. There was simply no possibility that the Royal Geographical Society would now sponsor and support Smith's expedition to the west of Lake Rudolf while the British government was involved in a high-stakes effort to militarily and politically secure the course of the Nile.

Thus political and military events had moved ahead so rapidly in this part of Africa between early 1897 and early 1898 as to preclude the Society's support of Smith's planned expedition. In early 1897 the council of the Soci-

FIGURE 2. Menelik II, Emperor of Ethiopia (reproduced from *Une Visite à l'Empereur Ménélick*, by Prince Henri d'Orléans, Paris, Librairie Dentu, 1898).

ety had agreed to give Smith £2,000 for an expedition between Lake Rudolf and the Nile. In a letter of July 29, 1898, to Sir Clements Markham (1830–1916), president of the Royal Geographical Society, Smith said:

> I will remind you that a year and a half ago the Council of the Royal Geographical Society promised me £2,000 towards fitting out an expedition to the country west of Lake Rudolf. The offer came too late; only a day before I started for Mongolia, and it would have been a breach of faith on my part to have deserted my two companions on the Mongolian trip."[12]

However, by 1898 the situation had obviously changed, precluding such support. Smith, who was clearly aware of most of these issues, seems to have been blinded by his determination and did not see why the Society could not and would not support his attempts to journey from the lake to the Upper Nile.

However, even before Keltie's diplomatic letter of November 10, 1897, reached him, he wrote back on November 18, 1897, from Bombay, India, saying that he planned to spend the winter in India "... unless I get a favourable reply to the letter I wrote you about Africa."[13] Thus Smith was aware that an immediate trip was out of the question. However, he did not see the trip as foreclosed for the foreseeable future, as did many others.

In this same letter, Smith told Keltie he was sending him 32 photographs from his Mongolian trip together with a paper and three original maps. Lieutenant George Steer, with whom Smith had traveled from Shanghai to Bombay, hand-carried the materials to London.[13] Smith marked some of the photographs "do not publish" as "... I wish them for an article I expect to write later on for a popular magazine."[13] If Smith did write such an article it has not yet been found after extensive search. George L. Farnum was scheduled to speak about the Mongolian trip before the Philadelphia Geographical Society in March 1898 and Smith was anxious that his paper appear before then. He told Keltie, "Mr. Farnum has agreed to wait until March before publishing anything on our journey."[13]

Smith and the Farnums gave a small collection of reptiles and batrachia from their Mongolian trip to the Academy of Natural Sciences of Philadelphia.[14] Smith also donated a collection of moths and butterflies to the Academy. Two tins of these were "... almost destroyed by a little white insect which had a good time of it while the tins were lying at the Bombay Customs House."[15] In his September 1, 1898, letter to the Academy of Natural Sciences of Philadelphia he goes on to say that "... I had to throw away very many specimens and these that are left are in an amazing condition for the specialist who works this up." Smith concluded his letter of donation by saying: "The butterflies (or what is left of them), are given to the Academy in the name of Mr. J. E. Farnum, and Mr. G. L. Farnum and myself."[15] Smith had been elected to life membership in the Academy in April 1896 following his return from the Lake Rudolf expedition.[16,17]

MEDICAL ACTIVITIES IN INDIA AND TIRAH

Smith remained in India for some seven months, from November 1897 to June 1898. During this period he spent much time with Percy Z. Cox (1864–1937), who at the time was assistant to the governor general's agent at Baroda, a position he had assumed three years earlier in 1895.[18,19(p50)] Cox and Smith knew each other well from the time when Cox was the assistant resident at Berbera in Somaliland.[19(p51)] According to Philip Graves, Cox's biographer, Smith and Cox were good friends. Smith spent the 1897 Christmas holidays with Cox and his wife at Baroda.[19(p51)]

Smith put his medical training to good use in India, serving as a physician to the military, for which he was given a stipend.[20] It was at this time that Afridi tribesmen from Afghanistan launched a religious war against the British, captured all the posts in the Khyber Pass, attacked British forts near Peshwar, and overran Tirah, which is a mountainous tract southwest of the Khyber Pass in what is now northwestern Pakistan.[21] British and Indian troops were sent into Tirah and in a series of military encounters in 1897–1898 known as the Tirah campaign, drove the invaders out and retook the Khyber Pass.[21]

Smith participated in the Tirah campaign as both a physician and a foreign news correspondent in 1897–1898.[20,22-24] Most of his obituaries, such as the one that appeared in the *New York Times*, state that he was "... a war correspondent in 1897 during the Tirah Campaign in India."[24] They did not indicate, however, the newspaper for which he served as a war correspondent. In a letter written to J. Scott Keltie from Jubbulpore on February 8, 1898, Smith says: "I have recently left the N.W. Frontier where I have been acting as war correspondent for the *New York Sun* with the Tirah forces. It was very interesting work for me as I saw considerable fighting, first in the Baria Valley and afterwards at Lundi-Kotal."[25] Given the fact that Smith was with the Coxes in Baroda for Christmas 1897 and arrived in Jubbulpore on January 18, 1898, it is likely that he was in Tirah during the last week of December 1897 and the first weeks of January 1898.[26] He says nothing of his medical activities during the Tirah campaign in these letters but he did speak of them later to his family.[20] Smith was superb at surgical techniques and enjoyed performing surgery. During the Tirah campaign he treated a number of the casualties.[20] A search of the *New York Sun* for possible dispatches from Smith has yet to be undertaken. Smith continued to provide medical care to the military until the spring of 1898 when he went on a hunting trip with Cox to the east central state of Bastr.[19(pp51–52)]

HUNTING TRIP TO BASTR, INDIA

Graves cogently notes that Cox had two reasons for making Bastr State in central India the object of his leave and a shooting trip.

> Both were connected with the arrival in India of Dr. A. Donaldson-Smith, whom he had liked and helped in Somaliland . . . he [Smith] and his host [Cox] agreed to meet in Bastr and attempt to obtain heads of that splendid beast, the wild-Indian buffalo . . . But this was not all. Donaldson-Smith was planning another journey with an armed caravan to the then almost unknown region between Lake Rudolf and the Nile. He suggested that Cox should join him, and Cox, to whom two years' furlough would be due in a year's time greeted the idea with enthusiasm.[19(p51)]

Smith preceded Cox to Bastr and wrote to him from Muchnee on April 2, 1898, that he "got a 9 ft 10 inch tiger with fine coat."[27] Cox reached Smith in Bastr after a week-long trip from Baroda, two days of which were spent on a train and four days of which required marching over difficult roads for 120 miles. Cox said of this leg of the journey:

> I did it in 4 days, going hard day and night, changing bullocks at each village and eating and sleeping in the cart on top of my kit. I reached D.S. on the 7th morning after leaving Baroda, and it will take me the same [time] to get back . . . 14 days traveling for 7 days shooting . . .[19(p52)]

Graves says: "He liked Donaldson-Smith, an unselfish sportsman."[19(p52)] In a letter to his brother Edward, Cox said of Smith, "When we were together, he . . . always wanted me to take up the first lot of tracks found."[19(p52)]

Smith had by this time gotten Cox extremely enthusiastic about joining him on the expedition to Lake Rudolf and the Nile. Cox, who had spent several years in Somaliland, planned to take a two-year furlough for the purpose of making the trip. All of Smith's letters to Cox during 1898 deal with the expedition and the various options and schemes Smith had worked out for financing the trip. By late 1897 Smith, sensing the lack of support for the idea by the Royal Geographical Society, came up with the idea of inducing the Gaekwar, the ruler of Baroda, to support the expedition financially. On December 29, 1897, Cox laid this proposal before his superior, Colonel Martelli, in a detailed letter.[28] After describing Smith's reputation and qualifications, Cox related that the expedition would last two years and cost £6,000, of which Smith was prepared to put up £1,000.[28] He proposed that the Gaekwar, who had set up the Baroda State Museum in 1894, be asked to contribute £5,000. In return the expedition would be formally under his auspices and all specimens collected would go to the Baroda Museum.[28] Allowing for the possibility that the Gaekwar might find the expedition too ambitious, Cox proposed a more modest alternative in the form of an expedition to Somaliland for the purpose of collecting natural history specimens.[28] Sensitive to political issues, Cox said:

> I had better point out that Dr. Donaldson Smith would go in a purely private capacity as an American subject, for it would be fatal to the success of the expedition (in case we fall in with Abyssinians as we should be sure to do) for Menelik to imagine that we had any political mission from or were sent by the British Government . . .[28]

He also told Martelli:

> Dr. Donaldson Smith is well known to the Intelligence Branch and I doubt not, to the Foreign Office, but if you consider any reference to Gov't necessary, I beg that the matter may be treated as strictly confidential. It would be suicidal to our interests for anything to get into the newspapers on the subject.[28]

Cox then cited the instance of a ". . . mention in the *Pioneer* of his [Smith's] readiness to organize an expedition to Uganda a short while ago, which was not accurate and was calculated to prejudice his plans."[28]

Martelli was favorably disposed to the proposal and the Gaekwar expressed interest.[19(p52)] However, Cox, because of his official position, could not discuss financial arrangements with the Gaekwar. This Smith had to do. A major reason for Cox going to Bastr to meet Smith was to lay out the approach to the Gaekwar.[19(p52)] Still both men had time for shooting. In describing a buffalo hunt to his brother, Cox said of their experience with a wounded animal:

> . . . in spite of her bad hock she came down on us like a steam engine. There was a sapling near D.S. and he just got there in time . . . all I could do was to get behind him . . . ready to fire if the beast got past the tree, but D.S. got her . . . and she rolled over at our feet.[19(p52)]

ATTEMPTS TO OBTAIN OFFICIAL APPROVAL AND FINANCIAL BACKING

On May 29, 1898, a letter was sent from the office of Gaekwar of Baroda to Cox informing him that £1,200 had been granted ". . . for a Scientific Expedition to Somaliland for the purpose of collecting Natural History specimens for the Baroda State Museum. . . ."[29] The Gaekwar had opted for a modest specimen-collecting expedition more in keeping with the interests of his museum. He understandably had no interest in becoming entangled in the political risks of a far-off corner of Africa, nor was he interested in sponsoring geographic exploration in Africa. No doubt the British authorities in India would not have allowed him to finance the Lake Rudolf-Nile expedition, and there is a hint of this in the letter Cox received. "I beg it to be clearly understood that no further amount will be paid by His Highness' Government over and above that now sanctioned in connection with the Expedition, nor will His Highness' Government undertake any other responsibility in regard thereto." [29]

Thus far Cox and Smith had obtained part of what they needed. This was better than what they had before, which was nothing. Cox drew up a detailed agreement for the Scientific Expedition into Somali Land of His Highness the Gaekwar. The agreement clearly stated that Smith was to lead the expedition, which was to be "confined to the British Somali Protectorate and to those districts which the leader may consider safe." [30] This phrase in article 3 of the scheme was the opening Smith wanted; he was confident he could get to Lake Rudolf and the Nile through districts he considered safe, provided additional financing could be found.

Smith felt some urgency about getting to the country between Lake Rudolf and the Nile because he feared someone else would get there before he did. Keltie had written him from London on March 2, 1898, telling him that Cavendish was planning a return expedition to Lake Rudolf with the intent of exploring the country between Lake Rudolf and the Nile.[31] Cavendish like many others was denied permission by the British government to enter the area[32] and instead planned on going on an expedition for Cecil Rhodes from Lake Tanganyika to the Nile.[33] This should have alerted Smith to the fact that the likelihood of another expedition's going into the region was remote.

Cox wired Smith the good news about the Gaekwar's £1,200 grant. Smith was then in Bombay, where on May 31, 1898, he wrote Cox telling him he was laid up with an attack of dysentery.[34] He told Cox that "the big scheme [the expedition to Lake Rudolf and the Nile] is very doubtful." However, he discussed approaches to solving the problem: ". . . if Menelik's consent be given — H.H. [the Gaekwar] would be willing to go in with the R.G.S. for say not over £4,000 . . . I will talk it over with you in Baroda." [34]

Smith arrived back in Baroda in early June where he spent two days with Cox prior to leaving India for Aden.[34] From the Smith-Cox correspondence over the next several months it is clear that they elaborated a fairly sophisticated plan of action for obtaining British and Ethiopian government approval for the Lake Rudolf-Nile expedition, financial backing from the Royal Geographical Society, and additional funding from the Gaekwar of Baroda. Cox was unwilling to trust the Somali as guards for the larger expedition and was instead inclined to recruit ex-soldiers of the Indian army.[19(p53)] This required bringing the Indian government into the picture as well.

Smith did not leave India for Aden on June 11 as planned but rather a week later on June 18 on the *S.S. Oriental*.[35] He wrote to Cox from Aden on July 3, 1898, saying that he was ". . . a little doubtful about Abyssinians letting us go on. . . ." [33] Because the Somaliland Protectorate officials reported to Aden, Smith was able to get a good idea of what was going on in Somaliland and provided Cox with a detailed update on the price of camels, the monthly wage for Somali men, and a description of conditions in the interior.[33] While in Aden, Smith also learned of Lord Delamere's trip from Somaliland to Lake Rudolf. One of Smith's former headmen even came over from Berbera to Aden to see him.[33] In Baroda Cox wrote to George Curzon (1859–1925) on June 21, 1898. Curzon was then a member of Parliament who, six weeks later, on August 10, 1898, was named viceroy of India.[36,37] Cox, however, wrote him in his capacity as a vice-president of the Royal Geographical Society, saying: ". . . any help you can give to Dr. Donaldson Smith will be given to me." [36] Cox had assisted Curzon on his visit to Somaliland several years before.[19(p34)]

Smith left no stones unturned in drumming up political and financial support for the "big trip," as he referred to it. In Aden he got the support of the resident, General Creagh, who advised him not to mention exploring between Lake Rudolf and the Nile at the Foreign Office.[38] Creagh offered to help Smith and Cox obtain Gurkhas for the trip.[38]

SMITH ENCOUNTERS OPPOSITION TO HIS PLANNED EXPEDITION

Back in London in late July 1898, Smith engaged in an intense effort to get the Royal Geographical Society to provide him with funding and the Foreign Office to intercede with the Ethiopian Emperor Menelik to let him pass unhindered. Both efforts were to fail. On July 29, 1898, he wrote to Sir Clements Markham, the president of the Royal Geographical Society, reminding him of his Society's earlier promise of help.[12] In his letter he outlined his plans, said that the officials in Aden were willing to help him, promised to avoid the Abyssinians, and said that if the council of the Society did not provide him with £2,000 he would approach the Italian Geographical Society of which he was an honorary member.[12] This veiled threat was not apt to intimidate anyone in London since the Italians were beholden to the British for relieving the Mahdist military pressures on them by moving into the northern Sudan. In this same letter he referred to ". . . the insinuations of Mr. Cavendish and Captain Lugard that I did not treat the Boran properly" [12] (see part III).

On August 6, 1898, Smith wrote to Cox, briefing him on what had transpired so far. He said, "The Government has refused permission to half a dozen Englishmen who had strong backing and who wanted to go into the country west of Rudolf. The fact that I am an American gives me an advantage as Curzon hinted." [39] However, Curzon was noncommittal, as was Keltie, whom Smith characterized as ". . . humble as pie on account of the Cavendish affair." [39] Smith was referring to the negative comments made by Cavendish and Lugard about his encounter with the Boran when the former addressed the Royal Geographical Society about his own Lake Rudolf expedition in 1897 (see part III).

Determined to get to Lake Rudolf and the Nile, Smith seems to have been undeterred by the initial benign and vague responses his request elicited. He believed that:

> The R.G.S. would like me to get Gov't permission through them so that they would have a hold over me & need not grant me the £2,000, thinking that I should spend my own money & they would get the credit & the results. I know the sly dogs Keltie & Markham. They know they acted like ——— when Cavendish read his paper & I have given them to understand that I will have

> none of their moral support . . . and if they do not accede to my demands I shall go on my own resources & what the Gaekwar gives me.[39]

These rather acid comments provide some insights into the frustration Smith must have been feeling, heightened by the fact that he would not or could not accept the fact that an expedition of the kind he was proposing was seen by the British government as too risky for both him and Britain. They also reflect that he felt hurt that Keltie and Markham had not defended him against the comments made during Cavendish's presentation before the Society. He was convinced that Cavendish's request to return was denied because of the negative reports about his conduct in Africa.[39] Even if such reports had not reached London, Cavendish would have been refused permission anyway. Yet Smith did not seem to understand this.

Smith did a bit of grandstanding with the Royal Geographical Society by threatening to have the Italian Geographical Society sponsor him or else pay for the trip himself, cutting them out in either case. Referring to the former he told Cox:

> They would hate this like poison . . . Keltie fears my accomplishing the journey & cutting the R.G.S. out & he has expressed himself thoroughly in favor of the £2,000. Markham will be brought to his bearings . . . when he becomes thoroughly aware that I have money enough to beat him & that I stand on a very firm footing with all the other Geographic societies & scientific men the world over.[39]

During this time Smith tried to get the Gaekwar of Baroda to put up more money to support the larger trip, estimating the total amount needed as high as £4,000 to £6,000. This indicates in part that he recognized the need for a larger and more expensive military escort in case they encountered the Ethiopians.

On September 1, 1898, Smith received "... a polite but flat refusal from the British Government. I am feeling very blue."[41] Writing to Cox from Philadelphia, he said:

> Must let Gurkhas remain in India . . . There is the Italian Government to approach but I do not know if they will give me permission if the British Government actively interferes. Lord Salisbury [Prime Minister] compliments me on my qualifications to undertake the expedition—only political conditions are such at the present time . . .[41]

Thus the decision not to allow Smith to proceed was made at the highest echelon of the British government, reflecting the level and degree of influence he must have brought to bear in pursuing his request. Still he persisted, working through the American Geographical Society "to get the U.S. Government to request the English as an act of courtesy to let me conduct an expedition from Berbera to Rudolf and the Nile."[42] He goes on to tell Cox, "The expedition in that case would be under the American flag. Will you go in under the American flag?" Then showing that he was aware to some extent of the political and military turmoil in this part of Africa he said, "This business at Fashoda may arouse Abyssinia [Ethiopia] & in case of war between England & the former our chances may be altered."[42] What Smith did not say but which he must have known was that Britain and France were almost dragged into a war over the latter's occupation of Fashoda on the Upper Nile.[8(pp337-362)]

While in Philadelphia Smith accepted membership in the American Philosophical Society on September 16.[43] He was back in London in early October, writing to Cox that "the U.S. Government have requested English Government to let me pass via Kisimayu or Berbera . . ."[44] Always aware of competitors, he said:

> . . . Wellby was refused among others so he hit upon the plan of going with Harrington [British Agent in Abyssinia] to Menelik's capital with the view of crossing the Nile north of Fashoda. I do not think he will succeed. Harrington told me that Menelik would not give permission to anyone to go through southern districts.[44]

Wellby did succeed in crossing through the region west of Lake Rudolf in 1899, but in a south-to-north direction.[45,46]

Despite all the odds, Smith was still hopeful he would get the Foreign Office to approve the trip to Lake Rudolf and the Nile. "I expect answer any day from Foreign Office. I will wire you if *not* successful Cox Baroda, this will mean short trip. If successful I will wire Cox Baroda, good — & in this latter case pick up fifteen Gurkhas."[44] Towards the end of his letter to Cox he said: "... If we only go on a short trip which is most probable bring your own tent and personal kit, but plenty of boots and clothes for yourself just in case we make a run for it."[44] In other words Smith did not rule out defying the British authorities and making a run for the lake and the Nile.

A week later, however, reality seems to have taken hold because although Smith had not yet had a reply from the British government he told Cox in a letter dated November 3, 1898, "... I think there is very little chance for big trip . . . They may be waiting to settle French question."[47] However, it was not only the French-British confrontation at Fashoda that was involved but Britain's position vis-a-vis Ethiopia. In his last letter to Cox from London before he left for Aden he said: "Government would not let me go to Rudolf since they had refused so many applications from officers & others. I have secured Carlysle Frazer, a Scotchman, as taxidermist. He is an old hand in African work, civil engineer and good taxidermist."[48]

At the beginning of December, Cox left Bombay for Aden where he met Smith. There, on December 11, 1898, Smith drew up the final plans for the Gaekwar's Expedition. Mrs Cox, who had also come to Aden, left for Bombay on December 18 and Cox and Smith then sailed to Berbera on December 19, arriving there the next day.[49(pp3-4)] They would remain in Somaliland until April 9, 1899, collecting specimens for the Baroda State Museum and hunting for sport. Smith had not given up on the idea of going to Lake Rudolf and the region between it and the Nile. However, some 400 miles down the river from where he hoped to meet it, the French and the British were facing each other off at a place called Fashoda, which would come to symbolize the struggle of the British and French for control of the Nile. Shrill rhetoric emerged in both London and Paris and there was serious talk of war.[8(pp332-362)] Yet it was not only the incident at Fashoda that blocked Smith's plans but also the intricate web of military and political events involving Britain, France, Ethiopia, and the Mahdist state that were played out in the regions of the Upper Nile. Smith would eventually reach Lake Rudolf on December 9, 1899, and the Nile on March 10, 1900, a little more than a year after he was denied permission to go there. However, this seemed a dim possibility as he started for the interior of Somaliland with Percy Cox on December 24, 1898.[49(p5)]

THE UPPER NILE IN THE 1880s

On September 13, 1882, Great Britain occupied Egypt after defeating the mutinous Egyptian army under Ahmed Arabi at Tel-el-Kebier.[50(p204)] Twelve days later, the Khedive (ruler) Tewfik was able to return to Cairo. Arabi's anti-Christian and anti-foreign nationalist movement was not without popular support and its causes ran deep. There was legitimate Egyptian resentment against foreign European domination of national life that partially had its origins in the spendthrift habits of the previous Khedive, Ismail, who brought the country to the brink of bankruptcy and French-British financial receivership in 1875. However, European/Christian domination of affairs in Egypt went further back in time, to the 1798 invasion by Napoleon. On June 26, 1879, Turkey, still the suzerain power, deposed Ismail at the request of France and Britain and replaced him with Tewfik. Arabi's popularity rapidly grew after his revolt, as did anti-European sentiment, forcing the flight of some 14,000 Europeans by June 1882. On June 11, 1882, a serious riot broke out in Alexandria in which several hundred people were killed, including 50 Europeans.[50(p203)]

William Gladstone, Britain's prime minister, was averse to invading Egypt in order to restore order and the Khedive. However, public pressure in favor of an invasion mounted and this, coupled with all of the historic circumstances that tied Britain to the Nile, forced his hand.[50(p203)] Gladstone unsuccessfully tried to get France and Italy to join Britain in the invasion. However, they refused, the French later regretting the decision, seeing Britain installed as the master of Egypt. Arabi was sent into exile in Ceylon and Britain was more closely drawn into her involvement with the Nile.

Sanderson cogently observes that:

> So long as Egypt herself controlled the Sudan, no European power was interested in the fate of the Upper Nile basin . . . British subjects were often the agents of Egyptian expansion towards territories where some of their fellow subjects — missionaries and merchants — had interests or aspirations.[8(p12)]

However, by 1887:

> . . . the basin of the Upper Nile was rapidly becoming an object of serious political concern to Britain and within the next three years British concern was greatly increased by the growing interest of other European powers. This rapid and surprising transformation had its roots in the British occupation of Egypt in September 1882 and in the Mahdist insurrection which, combined with heavy British pressure, forced Egypt into withdrawal from the whole of the Sudan.[8(p13)]

Nineteenth century Egyptian efforts to conquer the Sudan had begun in 1820 when the Ottoman Viceroy Mohammed Ali sent his son, Ismail, with an army to conquer the area. Egyptian efforts at consolidating control in the Sudan went into high gear under Ismail, who became the Ottoman pasha of Egypt in 1863 (he was later given the title of Khedive in 1867). He employed Europeans like Sir Samuel Baker, the noted British explorer, who remained in the Sudan from 1869 to 1873, establishing the Equatorial Province of the Egyptian Sudan in the process.[51(pp185-202)] Baker was followed in Equatoria by Charles George Gordon, who had won fame in China. Gordon's task, like Baker's, was to consolidate Egyptian authority in the region of the great lakes and to stamp out the slave trade. In 1877 Gordon was appointed governor general of the Sudan and he took it upon himself to make stamping out the slave trade his highest priority. He surrounded himself with American and British Christians who shared his zeal, but who like him were oblivious to the dislocation their sudden actions had on the Sudan's economic life. Gordon's crusade obviously alienated the powerful slave trading interests and the ordinary people saw the concurrent economic depression as due to the actions of a Christian foreigner. In 1879 Ismail, the Khedive, was no longer able to pay the interest on his loans and was forced to resign and go into exile. Gordon also resigned, leaving behind in the Sudan a tide of rising discontent and economic depression.[52] Yet to his credit, Gordon had accomplished much for the people of the Sudan.[50(pp179-196)]

Two years after Gordon's departure, the son of a Dunqulah boatbuilder, Mohammed Ahmad ibn Abd Allah, declared himself to be the Mahdi ("the divinely guided one").[52] By all accounts the Mahdi was a magnetic personality who was able to galvanize diverse sectors of discontent with Egyptian rule into a broad-based revolt.[53-55] This included the powerful slave traders, the holymen (fakirs) who despised the ". . . legalistic and unappealing orthodoxity of the Egyptians . . . and the Baggarah cattle nomads of Darfur and Kordofan who had had enough of taxes and unbearable Egyptian behavior." [52] As Moorehead notes, "Taxes were gathered with extreme harshness, and every Egyptian official was known to be corrupt . . . There were about 28,000 of them stationed in the various garrisons throughout the country and their behaviour towards the Sudanese had become unbearable." [50(p206)] The Mahdi's military successes were startling. By September 1882 he was in control of all of Kordofan and on November 5, 1883, annihilated an Egyptian army under the command of Colonel William Hicks of the Bombay Army at El Obeid.[50(pp211-212),52] Gordon was sent back to assure an orderly evacuation of Khartoum, the Sudan's capital. However, others saw in his return a chance for forcing the British government into action against the Mahdi. However, Gladstone wanted nothing to do with a military invasion of the Sudan.[50(p212)] The Mahdists eventually captured Khartoum and killed Gordon and most of his men on January 26, 1885.[50(pp233-275)] With the fall of Khartoum, the Mahdi's control of the northern Sudan was virtually complete. The Mahdists had invaded the southern Sudan in 1884 and would do so again in 1888.[56]

The Mahdi moved across the river from Khartoum to Omdurman where he died on June 22, 1885, only five months after Gordon was killed.[50(p279)] Although there is much speculation about the cause of his death—smallpox, poison, or typhus—the last seems the most probable based on Slatin's description.[55(p369)] The Mahdi was succeeded by the Khalifa who ruled the Sudan as an autocrat until it was reconquered by the British in 1898. The Khalifa was a man of limited knowledge of the outside world, but he possessed sufficient ability and charisma to hold the Mahdist state together.

COMPETITION FOR THE UPPER NILE

As Sanderson notes, "Since the Mahdist State neither sought nor obtained the recognition of other powers, the Sudan could be plausibly—and temptingly—regarded as *res nullis* . . ." 8(p13) The occupation of Egypt gave Britain a new strategic position in the Mediterranean. However, that strategic position was dependent on the Nile, whose annual flood gave Egypt its life. The late Victorians firmly believed that European technology could divert the course of the Nile. Thus an unfriendly European power in control of the Nile headwaters could hold Egypt for ransom or worse. These fears on the part of the British were heightened by an address made before the Egyptian Institute by a French hydrologist, Victor Prompt, in 1893.57(p107) He speculated on the consequences to Egypt if an unfriendly power were to control the Upper Nile. Prompt was able to convince President Sadi Carnot of France of the feasibility of damming the Nile at Fashoda just below where the Sobat joins it.58(p19-20) Carnot, himself an engineer by training, quickly endorsed Prompt's idea.58(p19) French politicians saw this possibility as a means of forcing Britain to negotiate a withdrawal of Egypt. Thus Fashoda, an otherwise insignificant Egyptian post first set up in 1855, became the focus of French military expeditions to the Nile.58(p19) However, this was not the only reason why the French wanted to get to Fashoda. They felt justified in extending their central African territories to the left bank of the Nile and having a share in the spoils of the Sudan when the Mahdist state broke up.

The French were not the only ones who posed a threat to Britain's interests in the Nile. King Leopold of the Belgians, who ruled the Congo Free State as an independent ruler, was another, as were the Italians who signed the Treaty of Ucciali on May 2, 1889, with Menelik.8(p69) By this treaty the Italians thought they had gained a protectorate over Ethiopia. Menelik, who at the time needed support in his struggle for supremacy in Ethiopia against Mangasha of Tigre, seems to have agreed to this but later conveniently repudiated it, saying his version of the treaty did not stipulate a protectorate.7(p112) Lord Salisbury's government was anxious to maintain Italy's good will on behalf of Germany, which was fearful of Italy's joining up with France.8(p67) However, Francesco Crispi, Italy's prime minister, was bent on obtaining a foothold in the Nile basin. Britain resolutely opposed this and with a protocol of March 24, 1891, was able to get the Italians to lay aside any claims to the Nile basin in return for recognition of Italy's sphere of influence in East Africa.7(p230),8(p78) Salisbury's diplomacy also effectively removed the Germans as a threat to the Nile. However, there was still the French and King Leopold II of the Belgians. Through much diplomatic effort on the part of Britain the Anglo-Congolese Agreement was signed on May 12, 1894.8(p170) This treaty did not live up to British expectations that it would neutralize King Leopold, nor did it serve to keep the French out of the reaches of the Upper Nile. It gave Leopold an enclave at Lado on the Upper Nile, leased to him for life, and claims to the Bahr el Ghazal which would later prove troublesome to British interests.8(pp162-187)

The first French attempt to reach the Upper Nile from the west was the Mission Monteil (1893–1894).8(pp140-161) Commandant Parfait-Louis Monteil was named Commander of the posts in Upper Ubangi in June 1893.58(p20) On August 24, 1894, he arrived in Loango on the Central African coast but was ordered to West Africa to deal with an urgent military problem posed by the iman-warrior Samory Toure.58(p20) Thus the first French strike at the Upper Nile did not materialize. The second one would, under Commandant Jean-Baptiste Marchand (Fig 3), a French soldier with much African experience—this despite the famous comment from Sir Edward Grey, the Foreign Office under-secretary, who told the House of Commons on March 28, 1895, that a French move into the Upper Nile would be considered an "unfriendly act." 8(p214),57(p11) The Grey Declaration had no real effect even though it was a veiled threat of war.58(p27) Salisbury, who returned to office in 1895, placed his hopes of securing the Nile on pushing through the construction of the railway from Mombasa to Lake Victoria.56(p11) While £3 million were appropriated for this, the project would not be completed until 1901.59(p205)

On July 24, 1896, Marchand arrived at Loango at the head of the Congo-Nile expedition. Because of civil disorders in the Congo which he had to put down, he and his column of Senegalese soldiers and staff of Europeans could not leave for the interior until January 1897. They

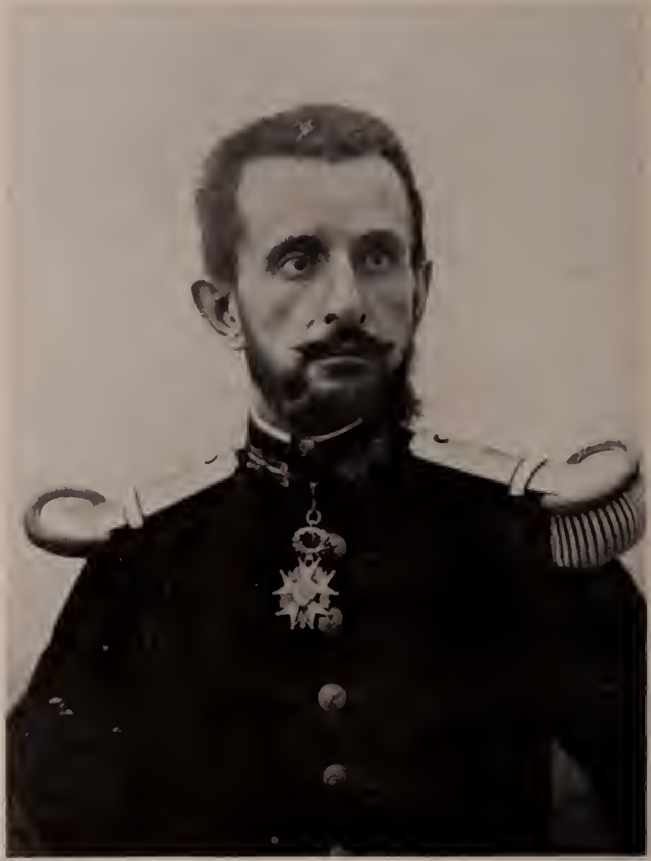


FIGURE 3. Commandant Jean-Baptiste Marchand (courtesy of Archives Nationales, Paris).

traveled up the Ubangi River to Bangui and beyond.[58(pp81-212)] Marchand followed the M'Bomu River which by a stroke of luck was navigable for a distance of almost 300 miles. Taking the *Faidherbe*, the Upper Ubangi post-boat, he sailed to the cataracts on the M'Bomu. Beyond lay 125 miles of unnavigable river. He dismantled the *Faidherbe* even though it was not a *bateau démontable* and portered it to the Sueh river, an affluent of the Bar el Ghazal. He reached Fashoda on July 10, 1898, where he raised the French tricolor two days later.[8(p287),58(p201)] By the standards of the day, Marchand had accomplished a remarkable feat, traversing 2,000 miles of jungles and swamps in a year and a half. This extraordinary journey across the center of Africa, much of it unexplored, has been described in detail by a number of writers, some of whom were participants.[60-64]

Unfortunately for Marchand and France, a combined Anglo-Egyptian force of 25,000 men, under the command of General Sir Horatio Herbert Kichener, was steadily but cautiously moving up the Nile toward Khartoum. Once the Khalifa was defeated and the Mahdist state destroyed, the inevitable and long-feared Anglo-French confrontation over the Upper Nile would unfold at this relatively insignificant spot, Fashoda. While Marchand was making his heroic journey against all odds through the swamps and jungles of Central Africa and Kichener was moving up the Nile, Arthur Donaldson Smith was unsuccessfully trying to get British support for his expedition to Lake Rudolf and the Nile. The British knew of Marchand's expedition and Kichener would soon learn that it had arrived on the Nile. However, before the Anglo-French confrontation could take place, the Khalifa and the Mahdist army would be defeated at Omdurman on September 2, 1898, the day after Smith received "a polite but flat refusal from the British government"[41] to journey to the lake and the Nile.

ETHIOPIAN OBSTACLES TO SMITH'S EXPEDITION

On March 1, 1896, an Italian army of some 17,700 men advancing on Ethiopia from the Italian colony of Eritrea was defeated by the Ethiopians at Adowa.[9(pp315-346),65(v2),66] At the time Arthur Donaldson Smith was in Philadelphia, having returned to the United States a few weeks before from his first expedition to Lake Rudolf. The Ethiopian victory at Adowa was significant in that it represented the defeat of a major European colonial power by an indigenous African state. The reasons for Italy's defeat are many, and a discussion of them is beyond the scope of this narrative. However, the size of the army, 17,700, was clearly insufficient when pitted against Menelik's 100,000 well-armed men. The Italian general, Oreste Baratieri, who was also governor of Eritrea, and his army were ill prepared for the confrontation but were ". . . goaded into action by Crispi's insulting telegrams . . ." as Sanderson aptly says.[8(p243)] The defeat caused widespread street rioting in Italy and the fall of Crispi's government four days later.[8(p243)]

Menelik was shrewd enough to realize that Ethiopia's independence could not be achieved through one military victory alone. Italy could if it wished send a large, well-trained army against him. Although that was unlikely given the political and financial situation in Italy, Menelik was anxious to conclude a peace treaty with Italy for as he saw it the real threat to his independence was the British.[67] Italy and Ethiopia signed a peace treaty on October 26, 1896, at Addis Ababa in which Italy acknowledged Ethiopia's independence. Nicholas Leontiev, a Russian nobleman who as a buccaneer and freebooter played a colorful role in Ethiopia's political and military life in the late 1890s, unsuccessfully tried to involve himself in the peace negotiations.[65(v2,pp237-312)]

Menelik's victory at Adowa and peace treaty with the Italians strengthened his position with other European powers and enabled him to foster Ethiopia's role as an indigenous colonial and imperial power whose focus was primarily in the south in the lands around Lake Rudolf. Ethiopian imperial designs in the southern border lands had been underway for some time, as evidenced by Smith's observations during his 1894–1895 trip (see part III). However, after 1896 these intensified, bringing Ethiopia and Britain, the rival colonial powers in the area, into competition.

One of the casualties of the Italo-Ethiopian conflict of 1896 was Vittorio Bottego (1860–1897), who led a scientific expedition to the Omo from Brava in Italian Somaliland in 1895–1897. Concerning the expeditions that had so far visited the Omo region (Teleki, Smith, and Neumann), Bottego's was, in the words of Tornay, ". . . the most important and the most systematic."[68] Bottego and his companions traversed the entire course of the lower Omo, went down the western shore of Lake Rudolf, explored Lake Stephanie, and then went up the Kibish River into what is now Ethiopia, heading for the Sobat River. As they were making their way north toward the Sobat River, H.S.H. Cavendish arrived at Lake Rudolf on March 3, 1897 (see part III). Bottego was killed in an Ethiopian ambush on March 17, 1897, at Jellem.[6(pp413-421),69] His surviving companions were taken prisoner, but eventually reached the coast at Zeila in British Somaliland on July 23, 1897.[6(p479)] At this time Smith was leaving the US for China and Mongolia. The city of Parma in Italy eventually erected a bronze monument to Bottego, whose great geographic accomplishment was the discovery that the Omo was the principal source of Lake Rudolf.[69]

Menelik's major concern between mid-1896 and 1898 was British aggression against Ethiopia. As Sanderson notes, he feared "that the subjugation of Ethiopia was the ultimate objective of British action in north-east Africa."[67] Thus a Mahdist victory against Kichener on the Nile was his best insurance for forestalling European aggression against Ethiopia.[67] As part of his plan, Menelik solicited diplomatic support from the French, but, as Sanderson notes, gave them little in return.[67] The French were interested in using Ethiopia as an eastern springboard for joining hands with Marchand's expedition coming from the west. With this in mind they sent Leonce Lagarde to Addis Ababa. He was the governor of the Somali Coast Colony. He negotiated the *Convention pour le Nil Blanc* which the Ethiopians and French concluded on March 20, 1897.[67] This treaty recognized Ethiopian rights to the right bank of the White Nile provided Ethiopia assisted any French expedition coming from the west.[67] Menelik, however, had no serious intentions of fostering French advances on the Nile. The French could offer him little in the way of military support and if he allied with them along the Nile he would be brought into conflict with the Mahdist state. To insure Ethiopia's independence he played a double game. On the one hand he offered the French assistance in their quest to secure the Upper Nile (and gave virtually none). On the other hand he entered into an understanding with the Khalifa, seeing in a Mahdist victory over the British his "best insurance against a renewal of European aggression in Ethiopia."[67]

It was against this background that a number of politically motivated expeditions sponsored by the British, the French, and the Ethiopians now moved into the regions near and around Lake Rudolf. On the surface a number of them purported to be of a scientific nature, but they were anything but that. Thus a purely scientific expedition such as Smith was proposing would have been seen by the French and the Ethiopians as probably political. And the British, who had no desire to provoke Menelik, were not about to do so by sponsoring what Menelik would have seen as a surrogate, namely Smith.

The British were of course deeply concerned about Menelik's suspicions of them and French and Russian influence at his court. To deal with these issues they sent James Rennell Rodd to Addis Ababa in 1897.[7(pp111-191)] On May 14 Rodd got Menelik to sign a treaty that in essence declared the Mahdists to be his enemies.[8(p259)] Unknown to Rodd and the British, Menelik had just concluded a friendly entente with the Mahdists. Rodd's visit did not convince Menelik that Britain's ultimate goal was not to subjugate Ethiopia, and the Russian and French representatives in Ethiopia did nothing to disabuse him of this view.

THE FRENCH EXPEDITIONS FROM ETHIOPIA

In order to join forces with Marchand from the east, the French sent out two separate expeditions from Addis Ababa in 1897.[8(p293)] One of these, under Captain Clochette, who was already in Addis Ababa, came to nothing as Clochette died on August 27, 1897, while in Ethiopia.[70(pp204-205)] The other, under the explorers Christian de Bonchamps and Gabriel de Bonvalot, was sent out under the auspices of the French Ministry of Public Instruction "disguised as a scientific expedition responsible for ethnographic and natural history research."[58(p144)] In order to insure the camouflage of this political mission, an entomologist, Charles Michel; a mining engineer, Leon Bartholin; and a landscape painter, Maurice Potter, were assigned to it.[58(p144),70] The expedition was plagued from the start by less than enthusiastic support from Lagarde, who was resentful because he had no direct control over it, and by logistical support problems exacerbated by sabotage on the part of the Ethiopians who feigned support of the enterprise. Menelik had no wish to see the mission succeed, for if it did it would drag him into an unwanted conflict with the Mahdist state, his bulwark against the British. The territories the French wished to give him on the right bank of the Nile were claimed by the Khalifa.

Lagarde was so miffed over his having no control over the Bonchamps-Bonvalot mission that he left Addis Ababa before Bonvalot arrived, meeting the latter on his way up. The two men quarreled, Lagarde upset over the lack of control of the expedition and Bonvalot aware that without Lagarde's support his mission stood little chance of success.[70(pp93-94),8(p294)] Bonvalot had so little success with Menelik that he returned to France, leaving Bonchamps in charge.[58(p145)] The latter was successful in obtaining guides from Menelik and a letter of introduction to the *Dejazmach* Tessama, the governor of Gore, one of Ethiopia's western provinces. The ending of Menelik's letter was telling, in that it said, "But you, come quickly, we will talk of that when you will be here."[70(p137)] In other words, the subtle message from Menelik to Tessama was to feign help but to obstruct the mission by any means possible. Bonchamps found Clochette dying at Gore and Tessama not merely unhelpful but obstructive.[70(pp113-144)] After four months in the field, Bonchamps and one other member of the expedition returned to Addis Ababa to solicit the intervention of Menelik. Lagarde, prodded by the government in Paris, intervened with the emperor, but in retrospect this intervention did not change matters much. Menelik sent Bonchamps a copy of a letter he sent to the Dejazmach [governor] Tessama dated October 12, 1897, authorizing the French expedition to move on to the west toward the Nile. Lagarde sent Bonchamps a letter dated October 10, 1897, authorizing him to plant the French flag on the western bank of the Nile and the Ethiopian one on the eastern. Contact with the Mahdists to the north was to be avoided.[70(pp253-255)] As usual, Menelik had an ace up his sleeve. While Tessama became more cooperative the expedition was instructed by Menelik to march along the left (south) bank of the Sobat River, the bank furthest removed from Fashoda and covered by swamps and traversed by several tributaries.[70(p254),58(p146)] This decision proved fatal to the small, poorly equipped expedition, as Menelik hoped it would. Bonchamps and his men managed to get to the confluence of the Baro and Adjouba rivers, which marks the beginning of the Sobat, in December 1897. They were some 90 miles from Fashoda but, afflicted by disease, hunger, and the seasonal flooding of the area, had to turn back on January 1, 1898.[70(pp276-334)] Menelik had won the day.

In March 1898 Tessama was instructed by Menelik to lead a reconnaissance party down the Sobat, because the latter was worried about possible British moves into the area from the south. While Bonchamps returned to Addis Ababa seeking instructions, a boat that had never materialized, and financing, two members of his expedition, Faivre and Potter, went along as Tessama's guests. Faivre and Potter persistently urged Tessama to move on to the Nile instead of following a southern route towards the region north of Lake Rudolf.[70(pp431-444)] They were finally successful in inducing him to allow a flying column of armed men to go down the Sobat to the Nile.[70(p445)] While Tessama remained behind with the bulk of his army, one of his lieutenants, Haile, led 800 men down the Sobat. Faivre and Potter accompanied the group, as did the Russian Colonel Artamanoff and several cossacks. They left Tessama on May 18, 1898[70(p445)] and reached the Nile on June 22, 17 days before Marchand passed the same point on July 9 on his way to Fashoda.[70(p455)] Colonel Artamanoff and two of his cossacks, in a display of Russo-French solidarity, swam to the left bank of the Nile to plant the French tricolor since there were no available boats. Meanwhile, the Ethiopians hoisted their flag on the right bank.[70(pp454-455)] France's attempt to lay claim to the Upper Nile now consisted of a makeshift tricolor fluttering above a lonely stretch of the river. Marchand would reinforce this claim a short time later, installing himself at Fashoda, some 60 miles downstream from the Sobat-Nile confluence.

The highland Ethiopians refused to remain in these swampy lowlands and despite repeated pleas by Faivre and Potter, left the flags and returned home toward Ethiopia.[70(pp455-460)] The small expedition of Ethiopians, Frenchmen, and Russians rejoined Tessama on August 5, 1898, after almost three months since they had left him.[70(p457)]

In terms of scientific accomplishments the Bonchamps expedition was a success. It had mapped a large, unexplored area to the west of Ethiopia, collected many natural history specimens, and produced detailed descriptions of regions largely unknown to outsiders.[70,71] Faivre and Potter along with their companions had traversed a stretch of country all the way to the Nile from a point (Gore) some 210 miles north of Lake Rudolf. This was relevant to Smith's plans, because he proposed to follow a parallel route between the lake and the Nile, arriving on the river some 165 miles above the point reached by the French. While Faivre and Potter were looking out over the Nile in late June 1898, Marchand was slowly making his way toward the river from the west. Smith was enroute to Aden from India, in the midst of his frantic efforts to obtain financing for his expedition to the river. In late 1897 Menelik had become aware of a British attempt to move on the Nile from the south (the Macdonald expedition). This posed an obvious threat to his designs on the lands south of Ethiopia around Lake Rudolf, and he made military plans to oppose the British there.[58(p147)] In fact, in mid-1898 Menelik sent Ras Wolda Giorgis down into the Omo region at the head of an army to assert Ethiopian sovereignty.[70(pp442-458)] Lake Rudolf and the region that lay between it and the Nile, where Smith so desperately wanted to go, were no longer merely objects of geographical and scientific research, but prizes in a complex and dangerous political game involving two major European powers, France and Britain, and two powerful indigenous states, Ethiopia and the Mahdia of the Sudan.

THE BRITISH APPROACH FROM THE SOUTH

Following Cavendish's expedition to Lake Rudolf in 1896–1897, only one other British expedition had visited the area by 1897. This was led by Hugh Chomondeley (1870–1931), Third Lord Delamere, who in 1891 at the age of 21 had gone on his first hunting trip to Somaliland.[72(p8)] Delamere made three return hunting trips to Somaliland. During his third trip in 1894 he contracted typhoid fever at Aden, and his leg was later severely mauled by a lion, which left him with a permanent limp.[72(p16)] Despite an attack of peritonitis he returned to Somaliland the following year, and in 1896 organized his most ambitious expedition, one to Lake Rudolf.[72(p23)] At the insistence of his mother he took along a physician, Dr A. E. Atkinson, who later settled in British East Africa along with Delamere.[59(p23-28)] The expedition left Berbera in December 1896 and by March of 1897 reached the Italian post of Lugh on the Juba River. Following a route similar to Smith's 1894–1895 one, Delamere passed along the line of wells that fringe the Mega escarpment. Then, departing from Smith's route, he went down to Marsabit, where he arrived in August 1897.[72(p45)] Cavendish's expedition to Lake Rudolf, which preceded Delamere's by a short time, arrived at Fort Smith in the East Africa Protectorate in August 1897 while Delamere was at Marsabit.

Delamere's expedition was the second European one to see Marsabit, Smith having "discovered" the forest-covered mountain in 1895 (see part III). The mountain swarmed with elephants and Delamere stayed a while to permit Dr Atkinson to accumulate some ivory. He shot 21 elephants in as many days and sold the ivory for £1,100 at Mombasa.[72(p47),59(pp23-28)] The Marsabit that Smith and Delamere saw has significantly changed because of encroaching human populations.[73,74]

Delamere moved south to Laisamis and then across to Lake Rudolf, which he reached in September 1897.[72(p47-49)] They arrived on the eastern shore of the lake, went south along it, and up the western shore for a distance before heading for Lake Baringo.[72(p49)] Delamere later settled in East Africa where he became the leader of the white settlers.[59,72] His expedition was modest in terms of its geographic accomplishments, but he took along a taxidermist and a photographer and collected natural history specimens.[72(p26)] Delamere, who had followed in Cavendish's footsteps by a few months, complained to the Foreign Office about the former's harsh treatment of the Boran.[75(intro, pxv)] Delamere's voyage was without political motive. However, the next one to arrive at Lake Rudolf was part of the British push to secure the Upper Nile.

THE JUBA EXPEDITION

In March 1897 Lord Salisbury, increasingly concerned by reports of Marchand's expedition to the Nile, was unsuccessful in getting the cabinet to approve a mission from Uganda to the Upper Nile.[8(p256)] The man chosen to lead this expedition was Major James Ronald Macdonald (1862–1927) of the Royal Engineers. Macdonald (Fig 4), who was 35 years old at the time, had already had a brilliant military career in India and much African experience.[76,77(intro)] He had served as the chief engineer for the preliminary survey for the Uganda Railway in 1891–1892 and had served as acting commissioner for the Uganda Protectorate in 1893.[76]

Macdonald was asked to submit plans for an expedition to the Upper Nile in April 1897 to Lord Salisbury, who kept both the Treasury and most of the cabinet in the dark about the true purpose of the expedition.[8(p256)] On the surface, the expedition was to explore the source of the Juba River. In reality Macdonald was instructed to move up the eastern banks of the Nile to just below the tenth parallel, which would take him into the area of Fashoda.[8(p257)] Sanderson sums up the expedition's purpose by quoting Lord Salisbury's note to Macdonald: ". . . secure the allegiance of the Chiefs by presents and the grant of the British flag . . . effectively to secure the territories in question against other powers . . ." [8(p257)] In other words Macdonald was to "stake claims by means of treaties." [8(p257)]

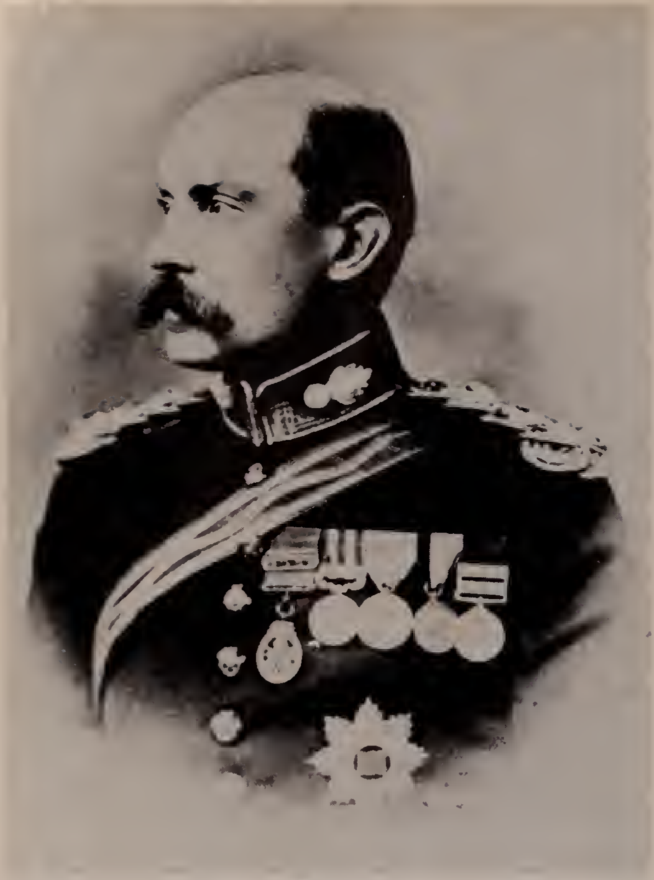


FIGURE 4. Major James R. L. Macdonald, who led the Juba expedition (reproduced from *With Macdonald in Uganda*, London, Edward Arnold & Co, 1903).

Although Arthur Donaldson Smith was in India at the time, he became aware of the Juba Expedition at least by November 1897, but not of its true political purpose. J. Scott Keltie wrote to him from London saying that the Macdonald expedition might be surveying Lake Rudolf, "... but we are not ... certain of this."[78] It was in this same letter that Keltie told Smith that Bottego had determined that the Omo flowed into Lake Rudolf.[78] As Sanderson notes, the timing of Macdonald's expedition could not have been worse.[8(p257)] Nubian soldiers, remnants of Emin Pasha's force, who had been settled in northern Uganda by Lugard in 1891, mutinied over poor and late pay and the rumor that Macdonald was going to force them to fight their Moslem brothers in the Sudan. In short order, other discontented elements in Uganda rebelled.[75(pp36-54,90-114),79(pp72-77)] Macdonald had to put down the rebellion, which required several months, and was not able to begin his mission until April 1898.[8(p257),75]

Menelik had reasons to be concerned about an expedition to the Juba River headwaters since he considered them to be in Ethiopian territory. Not surprisingly, Menelik, who was now gearing up to exert Ethiopian sovereignty over the regions around Lake Rudolf, was prepared to meet Macdonald with force.[58(p147),67] With Kichener moving down from the north and Macdonald coming up from the south it was plausible to him that a pincer movement against Ethiopia was underway.[67] One of Menelik's obvious reasons for sending out Dejazmach Tessama in March 1898 was to keep himself informed of European expeditions in the area, particularly Macdonald's, whose goal was the Sobat-Nile Confluence.[67] This was the journey that enabled the Frenchmen Faivre and Potter, who were part of the Bonchamps mission, to plant the French flag on the Nile.

Macdonald decided to split his force, sending his second in command, Captain Herbert Austin (1868–1937), with a party up to the Lake Rudolf region while he made for the Fashoda area.[75] Lord Salisbury was highly displeased when he learned of Austin's expedition to Lake Rudolf, because it constituted an unnecessary provocation to Menelik at the time when Kichener was rapidly moving to conquer the Mahdist state in the Sudan. The Austin trip held the potential for precipitating "the dreaded clash with the Ethiopians."[67] Menelik had remarked concerning Macdonald's encroachments on Ethiopian-claimed territory, "I'll send out Ras Wolda Giorgis with 20,000 men—they can check his passport."[67]

Austin started from the Mount Elgon area in July 1898, went up the western shore of the Lake to the Omo delta which he reached on September 12, and arrived back at his base, Save, on November 12, 1898.[75(p220)] He was gone three and a half months and surveyed 850 miles of territory.[75(p220)] Macdonald's column got as far north as Torit in what is now the southern Sudan but in November 1898 was forced to turn back because of a lack of provisions.[67(p258),80] Austin signed some 13 treaties on his trip to Lake Rudolf, although he makes no mention of them in either his book or his article describing the expedition.[75,81] His article accompanied Macdonald's. Matson cogently observes in the introduction to the 1973 reprint of Austin's book: "There seems little doubt that Austin knew the government would not welcome a discussion of them [the treaties] in his book ..."[75(intro ppvii-viii)] Any mention of the treaties arranged by either Macdonald or Austin was censored out of their reports and writings. Macdonald in fact was refused permission to publish a true account of the expedition and its purposes called *Uganda in Revolt*, and Austin's book had to pass through careful government scrutiny and censorship before he was able to sign a contract with the publisher on May 22, 1902.[75(intro pviii)] Salisbury, who was an arch-imperialist, was anxious about "increasing Parliamentary and press criticism of his East African policies, which were providing the opponents of further costly and provocative colonial commitments with grounds for attacking the government."[75(intro pvii)] Austin's book was finally published in 1903, the delay being due to both his subsequent preoccupation with a second expedition to Lake Rudolf undertaken in 1899–1900, when he reached the lake coming down from Omdurman in the Sudan,[82-85] and to British government censorship activities. Ironically, his book describing the second expedition appeared in 1902, a year before the one describing the first.[82]

In addition to his article in the *Geographical Journal*, Macdonald wrote one for the Royal Anthropological Institute and four official reports on the Juba Expedition which contained no material that "would lead to much bitter controversy and some inconvenient Parliamentary discussion."[75(intro pvi),86-90]

Arthur Donaldson Smith had no way of knowing of the true purpose of the Juba Expedition and it is reasonable to assume that Lord Salisbury for his part had no wish to allow him to go into the area and find out. Thus there was one more, but "secret," reason for the British Government's objections to Smith's proposal to visit Lake Rudolf and the Nile. The treaties made by Austin and Macdonald were not published and possibly "not circulated to even Salisbury's cabinet colleagues." [75(intro pvii)] A number of years would pass before their existence would be made public.

THE FASHODA CRISIS

On September 2, 1898, Kichener entered Omdurman, where the Mahdist army under the Khalifa had made its last stand. The Sudan, which Britain had so long desired, was now under the Union Jack. Yet there was disquieting news. Marchand and his force of roughly 80 Senegalese troops and seven French officers were installed at Fashoda on the Upper Nile.[91(p101)] After the loss of so much blood in conquering the Sudan, Britain was not about to sit idly by and leave the Upper Nile in the hands of the French. Kichener was instructed by the Foreign Office to proceed to Fashoda, which he did on September 10, 1898.[91(p101)] In a sense Kichener's war against the Khalifa had saved Marchand from annihilation since the Mahdist military forces were concentrated at Omdurman. Yet the Khalifa had sent two steamers up river to dislodge the intruders. Both had engagements with the French and had been damaged.[8(p333)] One of them, the *Tawfiqia*, arrived at Omdurman, its captain reporting an encounter with whites at Fashoda whose identity he did not know. However, Kichener had no doubts about who they were.

Kichener went up the Nile in force on the steamer *Dal*, escorted by four gunboats.[8(p333)] Enroute they encountered the second Mahdist steamer, the *Safia*, whose commander offered resistance which was overcome. The *Safia* was also badly damaged from its encounter with the whites at Fashoda.[8(p333)] Anchoring 12 miles downstream from Fashoda, Kichener sent Marchand a letter informing him of the Khalifa's defeat. Marchand in turn replied, congratulating Kichener on his victory, and informing him that he (Marchand) had signed a treaty on September 3 with the *mudir* of the region, placing the area under French protection, and that the treaty was subject to ratification by the French government.[91(p102)] By beginning their contacts in this courteous manner Kichener and Marchand had at least avoided gunfire. Kichener moved up river, noticing that the French flag was flying from the buildings erected by the Egyptians and abandoned 15 years before. He invited Marchand aboard the *Dal*. Fortunately both men took an instant liking to one another. Kichener's fluency in French was clearly an important asset in the delicate discussions that followed.[91(p102)]

Marchand stood his ground, much to Kichener's surprise. Kichener and Marchand exchanged gifts and the former set up a post a little further down the river. It was now up to the diplomats and politicians in Paris and London to solve the problem. The British press, rising on the wave of popular good feelings over the stunning victory at Omdurman, became almost hysterical about the French presence on the Upper Nile. The French press was no less restrained, claiming that the British had abandoned the Sudan in 1885 and that now the area around Fashoda and the Upper Nile belonged to France by right of first occupation. Matters reached a fever pitch during the first two weeks of October, and for a time it seemed that war would be declared between France and Britain. Marchand left Fashoda for Cairo on October 25, 1898, so that he could communicate directly with Paris.[58(p226)] Kichener left Cairo late in the same month, arriving in London to a hero's welcome.

Lord Salisbury viewed the French occupation as illegal and ineffective and put forth the right-of-conquest argument. Essentially, this argument held that Britain had conquered the Mahdist state and captured the capital, Omdurman, and thus by right the whole country was British.[8(p345)] The French did not see matters that way at all, claiming that Britain had abandoned the area for many years and, according to the rules of the game of colonial acquisition, the French now could stand on the right of first occupation. There were obviously merits to both arguments, and Africans living in the affected areas today would cogently argue that neither the British nor the French had any right being there at all. Determining who was in the right and who was in the wrong at Fashoda is a matter of perspective, insuring that the matter will be perpetually debated by successive generations of scholars, which in fact has been the case.[8,58,92]

British public opinion was unanimous that the French must withdraw. French public opinion on the other hand was deeply divided. In addition, the French army was at the time seriously split over the Dreyfus affair and the navy was in deplorable condition.[58(p222)] The French government gradually came to the conclusion that it would be better to put pride aside and come to an honorable compromise.

On December 11, 1898, Marchand, who had returned to Fashoda, lowered the French tricolor according to the instructions he had received from Paris.[58(pp233-234)] As British and French negotiators worked toward an agreement, Marchand stood firm that he did not wish to return via the Nile to Cairo through British territory as in so doing he would appear to be little more than a British prisoner. Instead he proposed returning via the Baro and Sobat Rivers and through Ethiopia. Lagarde, who was still in Addis Ababa as French minister, asked for and obtained Menelik's permission for this, and on December 13, 1898, Marchand and his men started on the long journey to Addis Ababa, where they arrived on March 8, 1899.[58(pp233-237)] After a month in the Ethiopian capital Marchand left for Djibouti on the coast, arriving there on May 16. Two weeks later he and his men disembarked at Toulon and were received in Paris as heroes. Although Marchand's mission to the Nile represented a French political failure, it nonetheless was one of the most remarkable and courageous journeys ever undertaken across Africa.

The French and British finally worked out a settlement which became an additional Declaration to the West African Convention of June 14, 1898. It was signed on March 21, 1899.[8(p371),58(p247)] By the terms of this treaty France was effectively pushed out of the Nile Valley, but given territorial concessions in regions to the west of the river. The Nile was now British from its source all the way to the Mediterranean.

SMITH AND COX IN SOMALILAND

As the Fashoda crisis was coming to an end, Smith and Cox were starting on what would be a three-and-a-half-month expedition to Somaliland. The principal purpose of the expedition was to collect natural history specimens on behalf of the Gaekwar for the Baroda State Museum. However, both men had obviously kept open the option of making a run for Lake Rudolf and the Nile.

Enroute from Aden to Berbera, the Goanese butler of the ship, the *S.S. Woodcock*, found that "... a Somali boy was concealed in our four kit bags. I went down to the baggage room," recounts Cox in his diary, "and found that a boy of about thirteen or fourteen had concealed himself in a dirty clothes bag of Donaldson-Smith's—on interrogating him I found he was a relation of D. S. Somali servant—Mohamed, whom he had taken on in Aden and it was evident that the latter has brought the boy over like this to evade paying his fare." [49(p4)] Other than the stowaway and a ship's mate who "was in his cups" and rambled about Satan being a female and the mother of the human race, they had "a beautiful passage." [49(p3)]

Smith and Cox arrived in Berbera on December 20, 1898, and on Christmas Eve set off with 30 camels, three ponies, and two riding camels for their collecting trip. The Gaekwar's Expedition to Somaliland was underway.

Cox kept a daily diary of the trip, written in a very legible hand.[49] It recounts in detail their stalks, hunting experiences, and natural history observations, and as such is a valuable historical document for conservationists. Cox also took along a camera and made numerous photographs during the trip. Both men were in contact with the outside world while in Somaliland, receiving and sending letters and reading slightly dated newspapers.[49(p10)]

On Thursday, December 29, Cox received a letter from John Lane Harrington (1865–1927), the British agent in Addis Ababa. Cox recorded in his diary that Harrington had written that "Wellby had got permission to go on to the Sobat and that we could also probably get permission if we wanted it, but might have to come up to Addis Ababa." [49(p10)] Smith was already aware of Welby's expedition to Lake Rudolf, referring to it in a letter to Cox on October 26, 1898.[44] Cox replied to Harrington the same day, saying: "... it would be too much a business and expense for us to come up to Addis Ababa, obliged him to try and arrange for Menelik to let us go on from where we are." [49(p11)] With the French-British confrontation on the Nile coming to an end Menelik could no longer exploit the rivalry between the two powers and saw it in his interests to be friendly to Britain, the dominant European power that now encircled his kingdom.

Cox and Smith continued their hunting, bringing down gerenuk, lesser kudu, oryx, Speke's gazelle, and wild Somali asses.[49(p13–25)] On January 8, 1899, they emerged on the coast at Bulhar where Cox met some of his old Somali acquaintances, including Jlmadar Musa, his old comrade from the Rer Hared campaign.[49(p25)] Two days later Smith had a close call with a wounded lion. Following the hunting practice of the time, they had tethered a donkey near a *zareba* (thorn bush enclosure) in which Cox stayed throughout the night. A lion and lioness were attracted to the donkey, killed it, and in turn were shot but not killed by Cox. At daybreak the lion was found dead nearby and bloody tracks through the elephant grass led Cox and Smith to the probable location of the wounded lioness.[49(pp29–33)] The men set fire to the grass behind the wounded lioness in an effort to drive her forward toward Cox and Smith.[49(p32)] Cox related in a matter-of-fact way what happened next:

> Sure enough she suddenly did break in front of D.S. and charged straight at him. He was on a little rising ground and fired when the lioness was at the bottom and his shot did not stop her. She was right onto D.S. and his boy when D.S. gave her his 2nd barrel between the ears and dropped her at his feet. She was only a foot or two away at the time and the powder singed her forehead—it was a narrow squeak for him and a sporting kill.[49(p32)]

And this was the man whom Henry Pleasants had described as "quiet and insignificant." [1]

On January 22, the mail from Berbera and Bombay arrived in camp and Cox noted in his diary, "... [I] see in the paper ... that Macdonald is pushing on N. from Uganda to Rudolf etc." [49(p42)] He also noted that Lieutenant Colonel James Hayes Sadler (1851–1922), the resident in Berbera, "... writes he hopes we have got what specimens we want now!!" [49(p40)] Sadler, who would later prove to be somewhat unaccommodating in helping them with their plans to go to the Nile, seems to have wanted to speed them on their way. On February 5, Cox recorded that Smith, who was at another camp site, sent him two papers, the *Spectator* and the *Weekly Times*. The *Times* contained a letter "... from Wellby giving account of trip with Menelik." [49(p52)] Lake Rudolf and the Nile were very much on their minds. Their hunting continued: "... D.S. went after some Klipspringer[49(p53)] and ... was actively collecting natural history specimens ... D.S. went out in the morning to look for more of the sunbird ..." [49(p57)]

By mid-February Cox was seriously ill with malaria. He records, "... fever came on heavily, rising to 104°. However was better in the evening but vomiting a good deal. Took a lot of quinine etc and got a little sleep." The next day, February 19, he had a single entry in his diary, "Lay low all day with fever." [49(p60)] Unbelievably, the next day he and Smith were off, "... feeling very shaky but well enough to march ... did 5 hours on the Darror road." [49(p50)] Five days later Smith became ill. Cox wrote:

> Unfortunate chap—D.S. is confined tonight—urethral catarrh! nice prospect of being laid up for 2 or 3 weeks—however it is a good place for him to be quiet in. If necessary I can make excursions for a day either here or there. Perhaps it is a false alarm—I hope so devoutly.[49(p64)]

The next day, on February 26, he recorded: "Symptoms have developed, so it is no false alarm. So D.S. lay low." [49(p64)] By March 2, Cox, who was hunting from a distant camp, said: "... note from D.S. saying his ailment was going on in a desultory way and that he would march tomorrow to Robosso." [49(p67)] Smith appears to have been suffering from gonorrhea but Cox provides no information on how he was treated, if at all.

The most exciting day of the expedition for Cox was on March 9, when he encountered a rhinoceros in high grass. Smith apparently had no interest in shooting rhino for Cox recorded on February 24 that "D.S. did not want to shoot rhino . . ." 49(p62) He fired both barrels of his gun at her as the animal "burst out of the grass," and turned to his gun bearer Abidi, who was carrying Smith's .577. However, Abidi had fled, leaving Cox no choice but "to scout for the nearest tree." 49(p75) His running scared the wounded rhino, which he then had to track. Wounded a second time, the rhino chased him around a tree and fled, driving a Midgan Somali into some thorn bush in the process.49(pp75-77) Cox finally tracked the rhino down again and killed it as it made a final charge the following afternoon. Using Smith's .577 and following his advice he recorded: ". . . taking DS's advice and aiming for the belly and not the shoulder." 49(p78) Of his rhino hunt Cox said, "Have had the most exciting day in my experience." 49(p77)

March 26, 1899, was a "red letter day" for Cox.49(p87) He received ". . . an enormous mail . . . 6 letters . . . best of all a letter from Harrington saying Menelik's permission to travel in Abyssinia granted. Hurrah! Now we only have Sadler i.e. Br. authorities' scruples to overcome. Cracked a bottle . . . in honor of the occasion." 49(p88) The next day Cox stayed at camp to:

> discuss matter with D.S.—now that long trip seems possible. Warned the boys that we had now got the permission to go on. They gave us a lot of trouble all day, incessantly changing their minds, first they would go then they wouldn't, one wanted one thing, one another—we had no peace . . . It was difficult for DS to decide what to do, but the final decision was—Fraser [the taxidermist] to go on to Aden on 29th with collections and dispatch them to England—then return to Berbera where we will rendezvous with him staying here meanwhile—once arrived at Berbera deal with Sadler and see finally whether our boys or other boys are game to go if the long trip is feasible.49(p88)

Menelik had given his approval but two other obstacles remained, the local British authorities in Somaliland and the recruitment of good men. It was clear to Cox and Smith that many of the men who had gone out on their hunting trip were not suitable for the trip to Lake Rudolf and the Nile. However, they had a solution to this—the recruitment of Gurkhas in India.49(p91) Colonel Sadler, the British resident in Berbera, who was host to Smith and Cox, proved uncooperative. Cox wrote in his diary on April 6:

> I produced Harrington's letters granting Menelik's permission to us to travel in Abyssinia, but he said it was not sufficient for him. He must refer to the Foreign Office. I argued that I did not see what concern or responsibility he had in the matter, all he had to do was forward our official report of our intentions to the Consular Agent at Harrar, as directed by Harrington. Conversation got a little heated. He said his instructions were not to let armed parties go at any rate beyond the Webbe [river] and that he must inform the Foreign Office. Asked if he could wire at our expense, he said no he must write—asked if he had any personal objection to offer he said no—but that he doubted Foreign Office attitude regarding Ghoorkas. Tried to reassure him that they were not sepoys [native Indian soldiers under British command]. Mr S. asked us to dine but we begged to be excused.49(pp92-93)

Cox tried to find out from the assistant resident in Berbera, Cordiaux, what Sadler's objection was and learned that it was the "idea of taking Ghoorkas 'the best fighting race in India' that put him off." 49(p93) Cox was determined and saw Sadler again the next day:

> I went over to see Sadler and again urged that he should throw the matter on to Harrington shoulders but he insisted that he must write and refused to wire. And also refused to show me what he wrote, but he swore he had no objection to offer and thought there was no likelihood of gov't objecting & said he thought we were perfectly safe in going on to India and making our preparations. . . . Cannot see why, if he has no objection to offer, he won't show us what he writes . . . but he won't show me . . . he said . . . that he had said all he could in our favor but he mentioned having said that we were accompanied by 50 men 30 Esc sepoys and 20 natives of India. The Esc sepoys is just the thing to scare them. Begged him to change it.49(p93)

The following day, April 8, Cox saw Sadler again and

> . . . repeated request to change Esc sepoys and he agreed to put partly ex sepoys and partly porters! He also showed me the letter he had written and I found he had put 'Rudolfland' as our objective! This is quite enough to put them off, & quite inaccurate. In my official notice for the Harrar authorities I said 'Lake Abaya and the Omo'—quite a different pair of shoes. He however agreed to change it and put as in my official.49(p93)

Sadler of course had the destination correct but Cox and Smith had misstated it to Harrington so as to disarm any opposition to the trip. Sadler was an experienced soldier and diplomatist having served in India and as consul at Muscat (1892–1896) before taking up his post at Berbera. He would later serve as commissioner in Uganda (1901–1905), and governor of the British East Africa Protectorate (1905–1909) and of the Windward Island (1909–1914). He could not be fooled. Lake Abaya and the Omo were viewed as Ethiopian. The Lake Rudolf area was still an open question and Lord Salisbury was concerned about possible armed clashes with the Ethiopians in the region. Menelik had in effect approved a trip through Ethiopian territory, not one to Lake Rudolf and the Nile.

Smith and Cox left for Aden on April 9, 1899.49(p93) As Graves notes, Cox was in India for the next two months helping Smith to recruit ex-sepoys for the Lake Rudolf-Nile expedition.19(p54) However Cox was never to go on the trip. On May 2, 1899, Lord Curzon, the new viceroy of India, appointed Cox resident at Muscat where the French were challenging British supremacy at the entrance to the Persian Gulf.19(p54) The news reached Cox in Bombay where as Graves notes, ". . . he was helping Donaldson Smith." 19(p54) Mrs Cox,

> . . . who made it a rule never to touch his official letters, broke it on this occasion in his absence, opened the letter and felt an immense relief when she read its contents. He accepted the appointment without hesitation. He was ambitious.19(p55)

Curzon made the offer very appealing by promising to raise the allowance at Muscat and by arranging a permanent appointment for Cox under the Foreign Department.19(p55) Smith was now on his own as far as the trip to the Nile was concerned.

The Gaekwar's Scientific Expedition to Somaliland was a success, and descriptions of the birds and batrachians collected were published in the *Proceedings of the Geographical Society of London*.93,94 Some of the 103 bird specimens collected were presented by the Gaekwar to the British Museum.93 Sixteen specimens of batrachians were also collected.[94] These collections amplified those which Smith had given to the British Museum following his 1894–1895 trip. Among the latter was an excellent butterfly collection.[95]

In August 1901, more than two years after the Gaekwar's Scientific Expedition to Somaliland had ended, Cox received a letter in Muscat from Baroda complaining that "Dr. Donaldson has not sent us any information regarding the expedition and all that we have heard on the subject is that a small collection of specimens of ethnology of Somaliland has been received per the enclosed list." [96] The letter went on to say:

> . . . [we] request the favour of your instructing Dr. Donaldson Smith to give an account of the expedition and to state what collections were made for the Baroda State Museum, how many objects collected have been forwarded to His Highness's Government and how many still remain to be supplied.[96]

On the surface these appeared to be serious allegations. However, Cox responded a month later, pointing out that:

> . . . the enclosure contains a long list of zoological specimens . . . representing a fine collection . . . Dr. Donaldson Smith complained very much to me of his inability to obtain any replies or acknowledgements to his letters to your address and after he left again on his expedition to Central Africa (for which the Royal Geographical Society awarded him their Gold Medal) the authorities of the British Museum . . . made complaints to me of a similar nature . . . I wrote to you on one occasion in this connection. . . . On my own part I may add that I addressed a long personal letter to His Highness the Maharajah giving him an interesting account of our doings; but I never had the pleasure of receiving any acknowledgement . . . when Doctor Donaldson Smith made up his account of the expenses of the expedition, he found he was out of pocket by it . . . You may be interested to know that 2 new species of batrachia collected by us, one of them named *Hemidactylus Barodanus*, are described and figured in the *Proceedings of the Zoological Society* Vol I Part I 1901.[97]

The list of specimens and ethnographic objects received by the Baroda State Museum was not enormous. It consisted of 28 mammals, 14 specimens of birds (103 specimens were collected and given to the British Museum), 150 entomological species termed "very ordinary," ten specimens of shells also termed "very ordinary," and four fossils called "insignificant." [98] There were 31 ethnographic objects including 14 spears and four wooden throwing clubs.[98] It is clear that the director of the Baroda State Museum, A.M. Masani, was unimpressed with both the quantity and quality of the collection. The Gaekwar, however, seems to have given a good deal of what Cox and Smith collected to the British Museum, for Sharpe remarks in her description of the birds collected that "H.H. the Gaekwar has very kindly presented to the British Museum such specimens as were desiderata to that institution." [93] It may well be that the Baroda State Museum staff was not fully aware of this.

Smith continued his preparations in India, spending most of May and June 1899 recruiting Sikhs and Gurkhas in the Punjab. By July he was back in Berbera, ready to leave for Lake Rudolf and the Nile.[99-101]

REFERENCES

1. Pleasants H Jr: Some horses are lucky. Biographical sketch of Arthur Donaldson Smith, MD. *The General Magazine and Historical Chronicle*, University of Pennsylvania General Alumni Society, XLVIII, 1946; 4:254-261 (courtesy of H. Spencer Potter, PhD).
2. Personal communication, H. Spencer Potter, PhD, Manhasset, NY, July 7, 1986.
3. Smith AD: A journey through the Khingan Mountains. *Geographical J* 1898; 11:498-509.
4. Letter from A. Donaldson Smith, MD, to J. Scott Keltie, Rittenhouse Club, Philadelphia, Pa, January 3, 1897. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
5. Letter from A. Donaldson Smith, MD, to J. Scott Keltie, San Ho, China, May 20, 1897. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
6. Vannutelli L, Citerni C: *Seconda Spedizione Bottego, L'Omo: Viaggio d'Esplorazione nell'Africa Orientale*, Milano, Ulrico Hoepli, 1899.
7. Rodd JR: *Social and Diplomatic Memories 1894–1901, Egypt and Abyssinia*. London, Edward Arnold, 1923, vol 2.
8. Sanderson GN: *England, Europe and the Upper Nile 1882–1899*. Edinburgh, University Press, 1965.
9. Berkeley G F-H: *The Campaign of Adowa and the Rise of Menelik*. London, Archibald Constable & Co, Ltd, 1902, reprinted, New York, Negro Universities Press, 1969.
10. Letter from A. Donaldson Smith, MD, to J. Scott Keltie, Shanghai, China, October 8, 1897. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
11. Letter from J. Scott Keltie to Dr Donaldson Smith, London, November 10, 1897. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
12. Letter from A. Donaldson Smith, MD, to Sir Clements Markham, London, July 29, 1898. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
13. Letter from A. Donaldson Smith, MD, to J. Scott Keltie, Bombay, India, November 18, 1897. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
14. Letter from Carol R. Spawn, Manuscript/Archives Librarian, the Academy of National Sciences, Philadelphia, Pa, to Pascal James Imperato, MD, May 8, 1979.
15. Letter from A. Donaldson Smith, MD, to the Academy of Natural Sciences, Philadelphia, Rittenhouse Club, Philadelphia, Pa, September 1, 1898 (Mss Col 150). Correspondence of the American Entomological Society, Academy of Natural Sciences of Philadelphia Archives, Philadelphia, Pa.
16 Letter from Edward J. Nolan, Recording Secretary, the Academy of Natural Sciences of Philadelphia, Pa, to A. Donaldson Smith, MD, April 1896 (Mss Coll 567). Academy of Natural Sciences of Philadelphia Archives, Philadelphia, Pa.
17. Letter from A. Donaldson Smith, MD, to J. Edward Nolan, Esq, 2227 Locust St, Philadelphia, Pa, April 7, 1896 (Mss Coll 567). Academy of Natural Sciences of Philadelphia Archives, Philadelphia, Pa.
18. Wickham Legg LG (ed): Sir Percy Zacariah Cox (1864–1937), in *The Dictionary of National Biography 1931–1940*. London, Oxford University Press, 1949, pp 196–199.
19. Graves P: *The Life of Sir Percy Cox*. London, Hutchinson and Co, 1941.
20. Personal communication, H. Spencer Potter, PhD, East Northport, NY, September 30, 1986.
21. Tirah Campaign, in *The Encyclopaedia Britannica*. Chicago and London, Encyclopaedia Britannica, 1972, vol 22, pp 12–15.
22. A. D. Smith Buried: African Explorer. *Philadelphia Record*, February 20, 1939.
23. Dr A. D. Smith, 74; African Explorer. *Philadelphia Evening Ledger*, February 21, 1939.
24. Dr Arthur D. Smith, Explorer of Africa. *New York Times*, February 21, 1939.
25. Letter from A. Donaldson Smith, MD, to J. Scott Keltie, Jubbulpore, India, February 8, 1898. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
26. Letter from A. Donaldson Smith, MD, to Percy Z. Cox, Jubbulpore, India, January 18, 1898. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
27. Letter from A. Donaldson Smith, MD, to Percy Z. Cox, Muchnee, India, April 2, 1898. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
28. Letter from Percy Z. Cox to Colonel Martelli, Baroda, India, December 29, 1897. Sir Percy Z. Cox Papers, Royal Geographical Society Archives, London.
29. Letter from the Office of the Gaekwar of Baroda to Perry Z. Cox, Baroda, May 29, 1898. Sir Percy Z. Cox Papers, Royal Geographical Society Archives, London.
30. Scheme for a Scientific Expedition to Somali Land on Behalf of His Highness the Gaekwar, undated. Sir Percy Z. Cox Papers, Royal Geographical Society Archives, London.
31. Letter from J. Scott Keltie to A. Donaldson Smith, MD, London, March 2, 1898. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
32. Letter from A. Donaldson Smith, MD, to Percy Z. Cox, London, October 26, 1898. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
33. Letter from A. Donaldson Smith, MD, to Percy Z. Cox, Aden, July 3, 1898. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
34. Letter from A. Donaldson Smith, MD, to Percy Z. Cox, Bombay, India, May 31, 1898. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.

35. Letter from A. Donaldson Smith, MD, to Percy Z. Cox, Bombay, India, June 8, 1898. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.

36. Letter from Percy Z. Cox to George Curzon, Baroda, India, June 21, 1898. Percy Z. Cox Papers, Royal Geographical Society Archives, London.

37. Weaver JRH (ed): George Nathaniel Curzon (1859–1925), in *The Dictionary of National Biography 1922–1930*. London, Oxford University Press, 1937, pp 221–234.

38. Letter from A. Donaldson Smith, MD, to Percy Z. Cox, aboard the *S.S. Carthage* bound for London, July 11, 1898. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.

39. Letter from A. Donaldson Smith, MD, to Percy Z. Cox, London, August 6, 1898. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.

40. Cavendish HSH: Through Somaliland and around and south of Lake Rudolf. *Geographical J* 1898; 11:372–396.

41. Letter from A. Donaldson Smith, MD, to Percy Z. Cox, Rittenhouse Club, Philadelphia, Pa, September 2, 1898. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.

42. Letter from A. Donaldson Smith, MD, to Percy Z. Cox, Rittenhouse Club, Philadelphia, Pa, September 16, 1898. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.

43. Letter from A. Donaldson Smith, MD, to the American Philosophical Society, Rittenhouse Club, Philadelphia, Pa, September 16, 1898. Letters acknowledging election series, American Philosophical Society Archives, Philadelphia.

44. Letter from A. Donaldson Smith, MD, to Percy Z. Cox, London, October 26, 1898. A Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.

45. Wellby MS: King Menelik's dominions and the country between Lake Gallop (Rudolf) and the Nile Valley. *Geographical J* 1900; 16:292–306.

46. Wellby MS: *'Twixt Sirdar and Menelik: An Account of a Year's Expedition from Zeila to Cairo Through Unknown Abyssinia*. London and New York, Harper & Brothers, 1901, reprinted, New York, Negro Universities Press, 1969.

47. Letter from A. Donaldson Smith, MD, to Percy Z. Cox, London, November 3, 1898. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.

48. Letter from A. Donaldson Smith, MD, to Percy Z. Cox, London, November 17, 1898. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.

49. Cox PZ: *Diary of an Expedition to Somaliland, December 1898*. Sir Percy Z. Cox Papers, Royal Geographical Society Archives, London.

50. Moorehead A: *The White Nile*. New York, Harper and Row, 1960.

51. Hall R: *Lovers on the Nile: The Incredible African Journeys of Sam and Florence Baker*. New York, Random House, 1980.

52. Collins RO: Nilotic Sudan, History of, in *Encyclopaedia Britannica*. Chicago, Encyclopaedia Britannica, 1973, vol 12, pp 108–116.

53. Wingate R: *Mahdiism and the Egyptian Sudan*. London, Macmillan, 1891.

54. Ohrwalder J: *Ten Years' Captivity in the Mahdi's Camp*, Sir R. Wingate (trans). London, Sampson, Low, and Martson, 1892.

55. Slatin RC: *Fire and Sword in the Sudan 1879–1895*. London, Edward Arnold, 1896.

56. Collins RO: *The Southern Sudan, 1883–1898: A Struggle for Control*. New Haven and London, Yale University Press, 1962.

57. Collins RO: *Land Beyond the Rivers: The Southern Sudan, 1898–1918*. New Haven and London, Yale University Press, 1971.

58. Michel M: *La Mission Marchand 1895–1899*. Paris, Mouton & Co, 1972.

59. Trzebinski E: *The Kenya Pioneers*. New York, WW Norton Co, 1986.

60. Baratier A: *Vers le Nil. Souvenirs de la Mission Marchand*. Paris, Arthème Fayard & Co, 1912, 1923.

61. Castellani C: *Vers le Nil Francais Avec La Mission Marchand*. Paris, Ernest Flammarion, 1898.

62. Baratier A: *Souvenirs de la Mission Marchand. Fachoda*. Paris, Editions Bernard Grasset, 1941.

63. Emily JM: *Fachoda. Mission Marchand 1896–1899*. Paris, Librairie Hachette, 1935.

64. Delebeque J: *Vie du General Marchand*. Paris, Librairie Hachette, 1936.

65. Zaghi C: *I Russi in Etiopia. Il protettorato italiano sull'Etiopia*, vol I, *Menelik e la battaglia di Adua*, vol II, Napoli, Guida Editori, 1972.

66. Combes P: *L'Abyssinie en 1896. Le Pays. Les Habitants. La Lutte Italo-Abyssine*. Paris, Librairie Africaine & Coloniale, Joseph Andre & Co, 1896.

67. Sanderson GN: The foreign policy of the Negus Menelik, 1896–1898, *J African History* 1964; 5:87–97.

68. Tornay S: The Omo Murle enigma, in Bender ML (ed): *Peoples and Cultures of the Ethio-Sudan Borderlands*. East Lansing, Mich, African Studies Center, Michigan State University, 1981, pp 33–60.

69. Vittorio Bottego, in *Enciclopedia Italiana*, Rome, Enciclopedia Italiana, 1930, vol 7, pp585–586.

70. Michel C: *Mission de Bonchamps Vers Fachoda. A la Rencontre de la Mission Marchand à Travers d'Etiopie*. Paris, Librairie Plon, 1900.

71. Michel C: Resultats géographiques de la mission de Bonchamps. *La Géographie* 1900; 2:25–34.

72. Huxley E: *White Man's Country, 1870–1914*. London, Chatto and Windus, 1935, 1953, vol 1.

73. Imperato PJ, Imperato EM: A short history of Marsabit. Paradise lost. *Africana* 1978; 6:28–32.

74. Imperato PJ, Imperato EM: Kenya's Lake Paradise, *Explorers J* 1980; 58:22–27.

75. Austin HH: *With Macdonald in Uganda*. Folkestone and London, Dawsons, 1973.

76. Weaver JRH (ed): James Ronald Leslie Macdonald (1862–1927), in *The Dictionary of National Biography 1922–1930*. London, Oxford University Press, 1937, pp 528–530.

77. Macdonald JRL: *Soldiering and Surveying in British East Africa 1891–1894*. Folkestone and London, Dawsons, 1973.

78. Letter from J. Scott Keltie to Dr Arthur Donaldson Smith, London, November 10, 1897. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.

79. Harlow V, Chilver EM, Smith A: *A History of East Africa*. Oxford, Oxford University Press, vol 2, 1965.

80. Macdonald JRL: Journeys to the North of Uganda. *Geographical J* 1899; 14:129–148.

81. Austin HH: Lake Rudolf. *Geographical J* 1899; 14:148–155.

82. Austin HH: *Among Giants and Swamps in Equatorial Africa. An Account of Surveys and Adventures in the Southern Sudan and British East Africa*. London, C. Arthur Pearson Ltd, 1902.

83. Austin HH: Survey of the Sobat Region. *Geographical J* 1901; 17:495–512.

84. Austin HH: A journey from Omdurman to Mombasa via Lake Rudolf. *Geographical J* 1902; 19:669–690.

85. Austin HH: Through the Sudan to Mombasa via Lake Rudolf. *Scottish Geographical Magazine* 1902; 18:281–302.

86. Macdonald JRL: Notes on the ethnology of tribes met with during the progress of the Juba Expedition. *J Anthropological Institute*, 1899, p 226.

87. Report by Lt-Col Macdonald, RE, of His Expedition from the Uganda Protectorate, 3 May 1898 to 5 March 1899, *Africa 9 (1899)*; C. 9503; presented to Parliament in August 1899.

88. Report of the Juba (Macdonald) Expedition, Part I: Operations of the Expedition by Lt-Col. J. R. L. MacDonald, RE; Foreign Office Confidential Prints 7167.

89. Military Report on Operations of the Juba Expedition after leaving Mumia's in June 1898 till the close of the Expedition; Foreign Office Confidential Prints 7232.

90. General Report of the Juba (Macdonald) Expedition, Part II; Foreign Office Confidential Prints 7593, October, 1901.

91. Warner P: *Kichener. The Man Behind the Legend*. Atheneum, New York, 1986.

92. De Caix R: *Fachoda. La France et l'Angleterre*. Paris, Librairie Africaine et Coloniale, J. Andre Editeur, 1899.

93. Bowdler Sharpe R: On a collection of birds made by Dr Donaldson Smith in Northern Somaliland. *Proc Zoolog Soc London* 1901; 69 (2):298–316.

94. Boulenger GA: A list of batrachians and reptiles obtained by Dr Donaldson Smith in Somaliland in 1899. *Proc Zoolog Soc London* 1901; 69 (2):47–49.

95. Sharpe EM: List of lepidoptera obtained by Dr. A. Donaldson Smith during his recent expedition to Lake Rudolf. *Proc Zoolog Soc London* 1896, 64:530–537.

96. Letter from Shrinivas Raghava Aieyanger to Percy Z. Cox, Baroda, India, August 26, 1901. Sir Percy Z. Cox Papers, Royal Geographical Society Archives, London.

97. Letter from Percy Z. Cox to Shrinivas Raghava Aieyanger, Muscat, September 20, 1901. Sir Percy Z. Cox Papers, Royal Geographical Society Archives, London.

98. Masani AM: List of specimens received from the Gaekwar Somaliland Expedition, Baroda Museum, Baroda, India. Sir Percy Z. Cox Papers, Royal Geographical Society Archives, London.

99. Smith AD: An expedition between Lake Rudolf and the Nile. *Geographical J* 1900; 16:600–625.

100. Smith AD: An expedition between Lake Rudolf and the Nile, in *The University of Pennsylvania Alumni Register*, February 1901, pp 148–155.

101. Smith AD: An expedition between Lake Rudolf and the Nile. *Bulletin of the Geographical Society of Philadelphia* 1900; 2:115–138.

Etiology of infectious mononucleosis

Solving the riddle

CARL POCHEDLY, MD

Infectious mononucleosis has long been an enigma and, to a certain extent, it still is. It has broad clinical manifestations.[1,2] Nearly every organ and system in the body can be involved, and the condition varies in severity from a trivial or inapparent illness to a fulminant, fatal disease. A chronic form is associated with bizarre psychological disturbances.

The evolution of our understanding of infectious mononucleosis can be traced from a vague clinical notion when the disease was first proposed in the 1880s, to a fairly clear-cut disease with specific serologic findings 80 years later. Landmarks in solving the riddle of infectious mononucleosis include description of the clinical syndrome, recognition of the hematologic changes, discovery of the heterophil antibodies, and identification of Epstein-Barr virus (EBV) as the etiologic agent.[2,3]

GLANDULAR FEVER AND ATYPICAL LYMPHOCYTES

The first descriptions of a disease with features vaguely reminiscent of infectious mononucleosis were reported by two pediatricians. In 1885, Nicoli Filatov, a Russian, described an illness in children characterized by malaise, fever, sore throat, hepatosplenomegaly, and lymphadenopathy.[4] Four years later, Emil Pfeiffer in Wiesbaden, Germany, noted a similar clinical entity in children, which he called glandular fever, or *Drusenfieber*.[5] However, on careful review of the original clinical descriptions, the diseases that Filatov and Pfeiffer described were not infectious mononucleosis. The cases occurred in epidemic form, affected mostly young children, and the incubation period was short—none of which are properties of infectious mononucleosis (W. Henle, personal communication, 1986). In any case, without specific laboratory findings on which to establish the diagnosis, the notion of glandular fever as a clinical entity was forgotten.[6]

The concept of glandular fever was revived about 30 years later, in 1920. Sprunt and Evans,[7] in Baltimore, reported the occurrence of fever, lymphadenopathy, and fatigue occurring in six medical students at the Johns Hopkins University. They also noted increased numbers of lymphocytes and the presence of atypical mononuclear cells on the blood smear. They put the various clinical and blood findings together and named the disease infectious mononucleosis.

Three years later, Downey,[8] in Minneapolis, described additional cases of infectious mononucleosis and provided an elegant morphologic description of the atypical lymphocytes. The blood cells he described became known as Downey's cells.[8] The recognition of atypical lymphocytosis as a hematologic marker led to more accurate descriptions of the clinical manifestations of this disease. But with these limited criteria for diagnosis, infectious mononucleosis was still a poorly defined clinical entity.

SERENDIPITY 1

The next important discovery occurred by accident. In the early 1930s, sheep red cell antibodies were found to occur in serum sickness. Investigators were exploring the distribution of these antibodies in various populations among both healthy and affected individuals. Paul and Bunnell,[9] at Yale, were studying rheumatic fever, and because of the similarities between the clinical manifestations of rheumatic fever and serum sickness, they examined sera of patients with rheumatic fever with the hope that they would find heterophil antibodies. As controls, specimens from patients with a variety of other acute diseases were also tested. Results for the patients with rheumatic fever were all negative, but among the controls, there was one serum sample with an unusually high titer of sheep red cell antibodies. The donor of the serum was quickly identified as a medical student who had signs, symptoms, and blood cell findings of infectious mononucleosis.[2] From this accidental finding in 1932, Paul and Bunnell went on to establish the association between infectious mononucleosis and heterophil antibodies.[9]

A few years later, Davidsohn and Walker[10] modified the original heterophil test by the addition of differential absorptions with guinea pig kidney and beef red cells to detect the specific heterophil antibody associated with infectious mononucleosis. This modification of the test enhanced the specificity for detection of heterophil antibody.

Dr Pochedly is a member of the Hematology/Oncology Department at Santa Rosa Children's Hospital, and Professor of Clinical Pediatrics in the Health Sciences Center of the University of Texas, San Antonio.

Address correspondence to Dr Pochedly, Department of Pediatrics, Health Sciences Center, University of Texas at San Antonio, San Antonio, TX 78284.

QUEST FOR THE ETIOLOGIC AGENT

During the 1940s and 1950s, strenuous but fruitless efforts were made to detect an etiologic agent for infectious mononucleosis. Attempts to culture bacteria and viruses from patients with infectious mononucleosis were unsuccessful, and the disease could not be transmitted to animals. Interpretation of experimental attempts to transmit the disease to humans was hindered by the failure to appreciate the widespread occurrence of asymptomatic infection in preadolescent children, as well as by the fact that there was as yet no specific serologic marker of immunity.[11]

In 1942, Wising, in Sweden, tried to transmit infectious mononucleosis to volunteer medical students by inoculating lymph node material and whole blood from acute cases. Only one of these efforts was successful. A student given 250 mL of whole blood from a patient with the acute disease exhibited characteristic clinical, hematologic, serologic, and biopsy evidence of infectious mononucleosis 18 days later.[2] Subsequent attempts to transmit the disease to humans by blood, serum, throat washings, and stool were unsuccessful. One wonders whether a modern institutional review board or human research committee would give approval for performing such experiments today.

The negative results were largely due to the fact that most of the volunteers were already immune prior to inoculation. This was shown when serologic studies were later performed on serum samples of previously inoculated volunteers, after tests for EBV antibodies were developed. These studies showed that EBV antibodies were already present in the sera of these volunteers at the time when they were inoculated with infected materials from patients with the disease.

In 1958, Denis Burkitt[12] described a peculiar lumpy tumor of the jaw among children living in certain regions of equatorial East Africa. Because the disease had a distribution pattern that coincided with the mosquito belt, Burkitt postulated that a mosquito-borne virus might be the cause of the tumor. Although no virus could be immediately isolated, efforts were made to grow the tumor tissue in culture.[2]

In 1964, Epstein and Barr[13] detected the presence of virus particles in cultured Burkitt tumor cells when examined under the electron microscope. First attempts to propagate the virus using conventional tissue culture techniques were unsuccessful, but it was later found that EBV could be propagated by infecting lymphoid cells. The infected cells became transformed into a continuous lymphoblastoid cell line. These chronically infected cell lines provided a source of viral antigen for testing clinical serum samples.

In 1966, Gertrude and Werner Henle,[14] of Philadelphia, introduced an indirect immunofluorescence antibody test to detect antibody to Epstein-Barr virus. The immunofluorescent test had two functions: (1) to detect the virus in virus-producing Burkitt's lymphoma cells or in other lymphoblasts and to identify it as a "new" virus (distinct from other herpes viruses); and (2) to detect antibodies to the virus in human sera. The Henles named the virus EBV after the EB-1 line of Burkitt's lymphoma cells in which it was first detected. This name was intended to be temporary, to be used only until more was learned about the virus. But by the time the virus' relation to infectious mononucleosis was generally accepted, it was too late to rename it infectious mononucleosis virus, which would have been most logical (W. Henle, personal communication, 1986).

By using the immunofluorescence antibody test for EBV in sero-epidemiologic studies, the Henles found EBV antibody to be present not only in sera from patients with Burkitt's lymphoma, but also in sera from healthy African children as well as healthy American children and adults.[13-15] The frequency and age distribution of the antibody suggested a common infectious disease. But which one? Neither the virus itself nor an antibody response to EBV could be associated with a specific infectious disease.[16]

SERENDIPITY 2

The fact that Epstein-Barr virus is the etiologic agent of infectious mononucleosis was discovered by accident, another credit to serendipity.

In their research, the Henles were trying to associate the presence of EBV antibodies with an array of febrile illnesses, as part of their extensive studies of "viruses in search of diseases." However, EBV antibodies were often found in all the populations studied, and their presence could not be linked with a specific disease. Confusion was compounded by the fact that it was not then known that infectious mononucleosis often occurs in inapparent or subclinical form in young children.[15] But research efforts expended in these fruitless studies were not in vain.

The crucial revelation in the search for an illness caused by EBV occurred in 1967. A young research technician named Elaine Hutkin working in the Henles' virology laboratory at the Children's Hospital of Philadelphia was using her own serum as a negative control in EBV antibody studies. Then Ms Hutkin developed a sore throat, fever, and enlarged cervical lymph nodes. Before long, the diagnosis of infectious mononucleosis was made on the basis of atypical lymphocytes in the blood smear, an increased lymphocyte count, and a positive test for heterophil antibodies. When Ms Hutkin returned to work in the laboratory after being out sick for a few days, it was discovered that her serum was now positive for the presence of antibodies to EBV. Her serum could no longer be used as a negative control. Thus, a young woman with proven infectious mononucleosis, who lacked antibody to EBV prior to having the disease, developed antibody to EBV after the appearance of typical symptoms and laboratory findings associated with the disease. Also, Ms Hutkin's lymphocytes soon showed growth of EBV in tissue culture.[17]

The Henles, of course, were impressed and delighted by Ms Hutkin's unexpected seroconversion. At once they suspected that there was a causal link between the occurrence of infectious mononucleosis and the sudden appearance of EBV antibodies.

But how could they prove that Elaine Hutkin's seroconversion meant that EBV was the etiologic agent of infectious mononucleosis? As luck would have it, researchers at Yale University had a collection of frozen and stored serum specimens that they had accumulated from ongoing, unsuccessful studies to determine the etiology of infectious mononucleosis. The Yale serum collection had been assembled in preparation for the time when candidate viruses for the cause of infectious mononucleosis would sooner or later be found, which could then be rapidly proven or disproven. These studies confirmed the Henle's suspicions about EBV (W. Henle, personal communication, 1986).

Sera had routinely been collected from students when they entered as freshmen and again when they developed infectious mononucleosis, as a part of a long-term survey of the disease and its transmission. These serum samples were exactly what the Henles needed to prove their suspicion that EBV causes infectious mononucleosis. This was rapidly followed by confirmatory findings on sera from college students elsewhere.[2] Thus, for the Henles in this study, serendipity was followed by extreme good luck.

The studies that were carried out on sera from Yale University students revealed that all students who had had infectious mononucleosis also had antibodies against the Epstein-Barr virus. These antibodies were absent in serum specimens collected from students before they developed symptoms of the disease. Among those students who did not have a positive history of having had infectious mononucleosis, the majority did not have antibodies to EBV.[16]

In a later study, serum samples were taken from students on entry into college at Yale, with the expectation that some of them would eventually develop infectious mononucleosis. The students were followed for several consecutive years. Among the students who had initially been free from antibodies to EBV, those in whom infectious mononucleosis later developed showed seroconversion and developed high titers of Epstein-Barr antibodies.[17] Antibodies to EBV appeared early in the disease, rose to peak levels within a few weeks, remained at relatively high levels during convalescence, and then persisted for many years. These antibodies were clearly distinct from the heterophil (anti-sheep red cell) antibodies.

Thus, large-scale epidemiologic studies demonstrated that heterophil-positive infectious mononucleosis occurred in persons who did not have preexisting EBV antibody. And, conversely, heterophil-positive infectious mononucleosis was always accompanied by acquisition of EBV antibodies. The presence of EBV antibody indicated immunity to infectious mononucleosis, and its absence indicated susceptibility. EBV antibody invariably appeared during heterophil-positive infectious mononucleosis as well as in some heterophil-negative cases.[2] When immunoglobulin M (IgM) antibodies to EBV appeared, they were clearly a sign of primary or recent EBV infection. An EBV seroconversion rate of nearly 15% per year was observed in susceptible college students.[2] These epidemiologic studies also showed that subclinical EBV infections occurred.

Furthermore, with the use of these specific antibody tests for EBV, it was found that 10–20% of cases of mononucleosis syndrome, of which most were heterophil-negative, were caused by other agents, such as cytomegalovirus, *Toxoplasma gondii*, hepatitis virus, or rubella virus.[17] Thus, EBV-specific serodiagnostic tests were found to be helpful in the diagnosis of heterophil-negative cases and in patients with unusual complications.

TRANSMISSION BY KISSING

The long persistence of shedding of EBV in the throat after the occurrence of infectious mononucleosis suggested that the virus is transmitted, at least in young adults, by contact with oral secretions. But is this disease really transmitted by kissing?

Colonel Robert Hoagland,[18,19] as a physician at the US Military Academy at West Point, New York, examined and interviewed all cadets who developed infectious mononucleosis. He was most impressed by a case that involved a cadet who admitted to having had a liaison with a female student. "During their brief but eventful 12 hours together," Hoagland noted, "the two kissed frequently and also drank from the same bottles." The girl became ill three days after the time she and the cadet were together, and the cadet became ill seven weeks later. From clinical and laboratory findings, both cases were diagnosed as infectious mononucleosis. The incubation period between the index case (the female student) and the secondary case (the cadet) was 47 days.[20]

The Henles studied a similar case with Swedish colleagues. A Swedish boy kissed a girl once at a party. The girl showed signs of infectious mononucleosis two days later, and the boy developed the disease 35 days later. Since he was seronegative, he was studied twice during the incubation period and EBV-carrying lymphoblast lines were established at 13 and 25 days after the kiss (W. Henle, personal communication, 1986).

Hoagland questioned 73 male cadets in whom infectious mononucleosis developed and discovered that 71 of the 73 confessed to having had intimate oral contact with girls 31 to 49 days before the onset of symptoms of infectious mononucleosis. It seemed that transmission by kissing was confirmed. But the majority of cadets who did not have infectious mononucleosis also had had intimate oral contact sometime during the seven weeks before being asked the question. There are two obvious explanations: not all cadets were susceptible; and not all girls excreted virus when kissed. Also, the testimony of cadets on this delicate issue is subject to bias: Many may have considered it unmanly to answer no, to admit that they had kissed no one for the past seven weeks.

Of course, direct mouth-to-mouth spread of infectious mononucleosis does undoubtedly occur. But the disease may also be transmitted by inhalation of droplets containing EBV, as well as by sharing drinking cups, toys, etc.[21]

EBV seems to multiply first in oropharyngeal epithelial cells; sooner or later it infects and transforms B lymphocytes, which proliferate wherever they settle out. Activated T cells then attack the virus-altered B cells, leading to the signs of infectious mononucleosis: namely, sore throat, lymphadenopathy, hepatosplenomegaly, etc (W. Henle, personal communication, 1986). Thus, EBV is present first in saliva, from which it enters the oral cavity and causes a localized infection in the salivary gland or in adenotonsillar tissue. After about two weeks, the virus circulates in B lymphocytes throughout the body. During the initial two to three weeks of this virocytemia, the patient remains asymptomatic. Therefore, a prodrome is followed shortly by symptomatic disease, including presence of the atypical T lymphocytes. The virocytemic period probably includes multiple cycles of viral replication, which are finally terminated by the emergence of the characteristic EBV antibodies.[20]

The Epstein-Barr virus is associated with other human diseases. The X-linked lymphoproliferative syndrome is a genetically determined disease in which EBV infection can result in a lethal outcome. Susceptible persons have overwhelming EBV infections, with fatal hepatitis, lymphoma, or agammaglobulinemia. The Epstein-Barr virus is causally linked to Burkitt's tumor, which occurs endemically in equatorial Africa. Persons who have had infectious mononucleosis have a two-fold to three-fold increased risk of Hodgkin's disease. EBV infection has also been implicated in undifferentiated vasopharyngeal carcinoma, which commonly occurs in Southern China, East Africa, and Tunisia, and in Alaskan Eskimos.

Acknowledgment. The author thanks Dr Werner Henle, who kindly reviewed the manuscript, confirmed its accuracy, and offered many suggestions for revision.

REFERENCES

1. Betts RF: Mononucleosis syndromes, in Williams WF, Beutler E, Erslev AJ, et al (eds): *Hematology*, ed 3. New York, McGraw-Hill Book Co, 1983, pp 943–950.
2. Evans AS: The history of infectious mononucleosis. *Am J Med Sci* 1974; 267:189–195.
3. Pochedly C: Laboratory testing for infectious mononucleosis: Cautions to observe in interpreting results. *Postgrad Med* 1987; 81:335–342.
4. Filatov NF: *Lektuse ob ostrikh infektsion Nikh Lolieznyak* (Lectures on acute infectious diseases of children). Moscow, U Dietel, 1885.
5. Pfeiffer E: Drusenfieber. *Jahrb f Kinderheilk* 1889; 29:257–264.
6. Tennican PO: Epstein-Barr virus infection, in Kelly VC (ed): *Practice of Pediatrics*. New York, Harper and Row, 1985, vol 4, pp 1–13.
7. Sprunt TP, Evans FA: Mononuclear leukocytosis in reaction to acute infections ("infectious mononucleosis"). *Bull Johns Hopkins Hosp* 1920; 31:409–417.
8. Downey H, McKinlay CA: Acute lymphadenosis compared with acute lymphatic leukemia. *Arch Intern Med* 1923; 32:82–112.
9. Paul JR, Bunnell WW: Presence of heterophile antibodies in infectious mononucleosis. *Am J Med Sci* 1932; 183:90–104.
10. Davidsohn I, Walker PH: Nature of heterophilic antibodies in infectious mononucleosis. *Am J Clin Path* 1935; 5:455–465.
11. Schooley RT, Dolin R: Epstein-Barr virus (infectious mononucleosis), in Wardell GL, Douglas RG, Bennett JE (eds): *Principles and Practice of Infectious Diseases*. New York, John Wiley and Sons, 1979, pp 1324–1336.
12. Burkitt DP, Davies JNP: Lymphoma syndrome in Uganda and tropical Africa. *Med Press* 1961; 245:367.
13. Epstein MA, Achong BG, Barr YM: Virus particles in cultured lymphoblasts from Burkitt's lymphoma. *Lancet* 1964; 1:702–703.
14. Henle G, Henle W: Immunofluorescence in cells derived from Burkitt's lymphoma. *J Bact* 1966; 91:1248–1256.
15. Kaplan AS (ed): *The Herpes Viruses*. New York, Academic Press, 1973, p 530.
16. Gross L: *Oncologic Viruses*, ed 3. New York, Pergamon Press, 1983, pp 934–936.
17. Henle G, Henle W, Diehl V: Relation of Burkitt's tumor-associated herpes-type virus to infectious mononucleosis. *Proc Nat Acad Sci USA* 1968; 59:94–101.
18. Hoagland RJ: Transmission of infectious mononucleosis. *Am J Med Sci* 1955; 229:262–272.
19. Hoagland RJ: in Glade PR (ed): *Infectious Mononucleosis*. Philadelphia, JB Lippincott Co, 1973.
20. Grose C: The many faces of infectious mononucleosis; the spectrum of Epstein-Barr virus infection in children. *Pediatrics in Review* 1985; 7:35–44.
21. Volpi A, Rocchi G: Natural history, diagnosis, and management of Epstein-Barr virus infection. *Ala J Med Sci* 1985; 22:157–159.

FROM THE LIBRARY

INFECTIOUS MONONUCLEOSIS

From the Point of View of the General Practitioner

In the days of Emil Pfeiffer, it was a simple matter to make a diagnosis of infectious mononucleosis. It was enough to keep in mind the clinical picture of the disease as described by Pfeiffer, with its special emphasis on the enlargement of the cervical lymph nodes, the fever, and the benign character of the disease....

There are, however, cases of infectious mononucleosis which clinically have little in common with the type originally described. Infectious mononucleosis is looked upon today as a generalized infectious disease, although the causative agent has not yet been isolated with certainty.

As is the case with many infectious diseases, several types of infectious mononucleosis have been described, depending on the predominant clinical symptoms appearing during the illness. The glandular enlargement may be of such small degree that it is barely distinguishable from the normal, especially in children. Contrary to the statement by Pfeiffer, skin exanthemata are not rare. The rash may resemble that of German measles or any of the acute exanthematic infectious diseases.

Reports have been made of an abdominal type of the disease simulating acute appendicitis and of a cerebral type with a predominance of meningeal symptoms....

Recently an asymptomatic type of the disease has been reported, in which, except for a high mononucleated cell count, no abnormalities could be found. The heterophile antibody agglutinative reactions were negative in every instance.

From the above remarks one can clearly see how rich and variable the symptomatology of infectious mononucleosis can be. Indeed, sometimes the disease may develop rapidly, with sudden chills, high fever, severe headaches, vomiting, skin rashes, jaundice, and all the common signs of an acute and severe infection. At other times the symptoms may be so mild that the patient feels only a little "indisposed," and continues with his work. These cases may easily be overlooked if no blood examination is made. The term "abortive" infectious mononucleosis would be appropriate in cases of this kind.

Like the symptoms, the duration of the disease also varies greatly. It may last from a few days to three or four weeks. The prognosis is said to be always favorable....

HENRY B. SOKAL, MD
(*NY State J Med* 1943; 43:848–852)

Death of a dentist

NAOMI R. BLUESTONE, MD, MPH

No one thought to inform me about the funeral, nor was there any reason why they should have. I was, after all, only a patient. I did not discover that my dentist of almost a quarter-century had died until six months after the office was closed. His sister, who began to cry when she heard my voice, called to tell me that my records were now over in the Medical Arts building on Joralemon Street. (This was an advisement that proved to be more good intent than fact; actually, they were lost and are probably still moldering away in a basement somewhere). I suppose it did not occur to any member of the family that upon receiving this startling news, I would be sufficiently moved to sit down and cry. I did, however, shedding those salty drops that leave eyes and nose red and puffy, and render the mourner unfit for duty until major cosmetic restorations have been performed. I loved Jack. I depended on him, I trusted him; I felt that he cared about me and that we shared many good memories. What more could be said about a good doctor?

Like all mourners, I mentally ran through the years we had known each other. I had found this man in the worst possible way, through the Yellow Pages of the Brooklyn phone book. I'm ashamed to admit it. But I had lost an inlay, I needed a dentist, I was new in town, and the address was only a block away. I took a chance and made an appointment. It proved that while a fool and his money are soon parted, a fool and her teeth may not be.

I found a friendly, large person who, with a persistent and nosy curiosity, wanted to know everything about me. Where I came from. Who had done all that beautiful gold work in my mouth. Why a nice Jewish girl like me wasn't married yet. From anyone else, I would have run a mile. But from him, I took it.

His office was like that of an old GP. There was but one chair, looking as if its immediate antecedents were probably reposing in museums, attics, or junkyards. His dental cabinet, with its emaciated, vertically lined drawers, was of some grand old wood but badly chewed through years of banging and neglect. Behind the tiny office in which the chair loomed so large, there was an antiquated lavatory, and a small antechamber dominated by a cluttered desk of the same "Art Wrecko" style. The waiting room, predictably close, was furnished in growing-boy maple. The loveseat and armchairs, with their very wide armrests, seemed to have come from a tourist cabin in the Adirondacks, circa 1930. It was all old and worn and comfy, like grandmothers used to be before they became polyester and peroxide. Only the high ceilings of this modest first-floor suite bespoke the former grandeur of the apartment house on Clinton Street. This was, after all, still Brooklyn Heights, and his rent, even then, was out of sight.

If the office would have made the squeamish flee, his manner and style would have made many of them take off even faster. He was, as I have said, a busybody and an overt snoop. With the greatest glee, he would inform me of the foibles of his other patients, making me despair of ever keeping my private life from their ears, too. He was a nonstop talker, and mercilessly treated his captive audience to a running account of his troubles with his wife, who, he grumbled good-naturedly, did not understand him; or his retarded son, whose long-term welfare remained his principal preoccupation. As his fingers plied their busy machinations, he would recount his adventures on his junkets to the gambling casinos, the track, and, as he enjoyed hinting, to places of feminine solace. And, when his drill was a micron's distance from the root canal, he would produce a joke, usually ribald, frequently unrepeatable, and generally so funny that all dental work would cease while I heaved and roared.

Unlike many young dentists who treat the tooth, the whole tooth, and nothing but the tooth, Jack treated the whole tooth, the whole mouth, and the whole person. He took his time. He was a lonely man, isolated in that office with no assistant for company, and his patients were his amusement. He knew their needs. He made appointments available to me at ridiculous hours because he knew I was so busy. He scaled his fees to a raw minimum because I was first a resident, then an academic, next a public servant, and finally a resident again in mid-life. He took pride in my accomplishments. He read my articles, brought them home to his family, boasted of them to other patients in his waiting room. The night the lights went out in New York, a now historic occasion, his dental drill went dead in mid-cavity. He walked me home and waited for me to climb ten flights of stairs and wave to him that I was securely back in my nest. Now, how's that for a shared memory?

I believe he was a good dentist, although what non-dentist in this world can ever be sure of that?! He certainly

---

Address correspondence to Dr Bluestone, Consulting Editor, *New York State Journal of Medicine*, 420 Lakeview Rd, Lake Success, NY 11042.

took great pride in his work, never tried to con me about his abilities, shared the results of his new discoveries with me. He never used novocaine and he never hurt me. Not a bad record for a man working on a basically terrorized, inwardly screaming coward.

Jack was no dope. He knew that all the new crop, his other son among them, were making big bucks fresh from dental school, far more than he would ever command. He griped about it plenty. But he was an old dog who believed in his old tricks, and his patients were grateful.

I was aware that my dentist was chronically ill, for he griped about that, too. He was hospitalized for diabetes and dropped so much weight he looked like a gaunt miniature of his former self. He complained about all the things he couldn't do, couldn't eat, couldn't have. He griped because his wife had dragged him to suburbia for a forced semi-retirement, and he hated every minute of it. He said he wanted to die in harness, because he was terrified of death.

I shared his sense of fleeting time. There came a period when each time I left, I wondered if I would ever see him again. When I, too, moved to another suburbia, on the opposite side of the city, I kept telling myself that it was sheer madness to drive back into the cauldron and put up with the parking hassles, just to see a sick man who should be retiring himself. But I went. So did others. We knew someday it would have to end, that soon the long arm of the modern dental chair would reach out and wrap itself around our lower mandibles. I, for one, indulged in an orgy of anticipatory dread about the real world of girl dental hygienists, computerized billing, plastic waiting rooms, cherry-flavored novocaine, and polyester dentists.

So, when Rose told me that Jack had died, I wept. It has been over a year since that relic of an office has been dismantled, painted, and turned into a condo, or some other form of prime real estate. And my eyes are still wet, as I write this sentimental prose elegy to a dentist who served his patients faithfully, and left them with indelible memories.

It is rare that a physician is privileged to become aware, suddenly and sharply, of some human distress about which he or she has never thought much before. I would say that such an awareness has come now. How fallacious it is when we grow old, magically believing that our doctor will remain a constant for us indefinitely! What a terrible thing it must be to be preceded in death by one's physician of many years! How confusing and frightening to have to consult some young kid, no older than a grandchild and equally weird, who knows us only by our historical representations and our diagnostic returns!

If I were still actively teaching medical students, I would instruct them to ask patients over 55: "Who is (or was) your personal physician? How long did he (or she) attend you? What was the nature of your relationship?" Only then could a sensitive young person be able to say something like: "That must have been a great loss for you. You must feel that no one else could possibly pick up the story of your life, or be as skillful or caring. I am trying to understand how you must feel, and I will do all that I can to help carry on. You can help me by . . ." (fill in the blanks). Certainly such an approach to the older patient is not free of hubris, since the nature of "understanding" enriches only with age and experience. But the intent would be clear. It would acknowledge that there are still a few fortunate souls left alive who have had an enduring relationship with one human being. Not too common in this age of the throwaway toothbrush, the disposable marriage, and the dispensable doctor.

The old-time physician, solo, ever-available, intensely valued, and exclusively endowed with faith, has given way to the group, a corporation who in the aggregate are probably less available now than their professional grandfathers, certainly less valued, and endowed only with the faith of the desperate, who have no other choice and so console themselves with the realities of efficiency. As time goes on, fewer and fewer individuals will have been able to claim both a private relationship and its loss.

Not all transitions to new relationships and new treatment situations have to be traumatic. In my own case, after dealing with my resistance to starting up again with someone new, I ended up calling my husband's dentist, whom I had never met before. He was a pleasant man, courteous, appropriately respectful of both my mourning and the remains of my old gold inlays (now a shadow of their former selves). He was amused that despite my undisputed middle age, he was only the third dentist to have incised my choppers. He informed me in no uncertain terms that *he* used novocaine because it made him feel more secure in his work, and I submitted before this inarguable logic. He worked efficiently and silently, aided by an earnest young woman who looked very upwardly mobile. His work-up and management seemed excellent. His air conditioning was silent, his walls had been painted, and his chair obediently refrained from grabbing my mandibles. He was even close by, and offered wide open streets for my parking convenience. A month after he made up my new chart, he notified me that, at the age of 55, he was giving up dentistry to become a high school science and biology teacher in Florida.

Time moves along. So, dear Jack, you are now put to rest in my mind. I hope you are now in dentist heaven, where the horses are swift, the odds favorable, the casinos full, and the drills at work on interesting restorations.

Serum creatinine increase associated with amiodarone therapy

MICHAEL B. JACOBS, MD

Amiodarone is an effective therapeutic agent for both ventricular and supraventricular arrhythmias.[1] Its use, however, is accompanied by a host of adverse effects.[2-4] Reported here is a case of an amiodarone-associated elevation in serum creatinine—an uncommon toxicity associated with this drug.

CASE REPORT

An 86-year-old man had been receiving treatment for systolic hypertension since 1975, for paroxysmal atrial fibrillation since 1980, and for atypical chest pain with a borderline abnormal exercise electrocardiogram since 1981. His arrhythmia was poorly controlled with digoxin, quinidine, and propranolol. During episodes of atrial fibrillation, ventricular rates ranged from 44 to 160 beats per minute.

Amiodarone therapy (200 mg/day) was begun in January 1984, with subsequent maintenance of normal sinus rhythm. Six months later, the patient's serum creatinine level rose from 1.8 mg/dL to 2.8 mg/dL, with a concomitant rise in the blood urea nitrogen (BUN) level from 24 mg/dL to 36 mg/dL. Because of this unexplained azotemia, amiodarone was stopped in July 1984, and the patient was referred to the nephrology service.

He was taking no drugs known to be nephrotoxic or to interfere with creatinine determination. There was no evidence of hypotension, congestive heart failure, or intravascular volume depletion. Urinalysis disclosed 1+ protein by both dipstick and sulfasalicylic acid tests. The specific gravity of the urine was 1.020. Microscopic examination of a urine sample disclosed no abnormalities. Results of a 24-hour urine creatinine clearance study and a 24-hour urine protein excretion study were 35 cc/min and 1,096 mg, respectively. A renal ultrasound examination was performed, and when compared to similar studies from March 1984 and December 1982, showed that the kidneys were slightly small, stable in size, and otherwise normal. When the amiodarone therapy was discontinued, the patient's serum creatinine level fell from a high of 2.8 mg/dL in June 1984 to 2.0 mg/dL in January 1985. His BUN level fell from 36 mg/dL to 18 mg/dL. The results of urinalysis remained unchanged, except that a dipstick protein test was now trace positive. Amiodarone therapy was reinstituted in April 1985, and by June 1985, the serum creatinine level had increased to 2.7 mg/dL and the BUN level to 35 mg/dL (Fig 1).

DISCUSSION

Amiodarone is a benzofuran derivative that is structurally similar to thyroxine and contains 37.5% iodine (75 mg iodine per 200 mg tablet).[5] It has been successfully used in the treatment of both atrial and ventricular arrhythmias.[1,6] However, multiple adverse effects have been reported, including corneal microdeposits and photosensitivity.[2,3] Thyroid dysfunction, hepatic abnormalities, interstitial pulmonary disease, and neuromuscular symptoms are also well documented.[1-9] The hepatic, neuromuscular, and ocular side effects appear to depend on the serum concentration of amiodarone, whereas the pulmonary and cutaneous effects may be seen even at low serum drug concentrations.[1] The thyroid abnormalities are explicable on the basis of the drug's biological activity.[5]

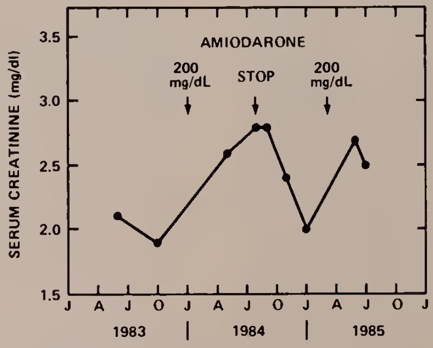


FIGURE 1. Effect of amiodarone therapy on serum creatinine level.

Amiodarone-related changes in renal function were not noted by Heger et al[6] in 45 patients followed for an average of 12.7 ± 8.8 months. Neither Romensch et al[1] (127 patients) nor Harris et al[2] (140 patients) described renal dysfunction in patients who received amiodarone. However, these authors apparently did not routinely measure either creatinine or blood urea nitrogen levels. Leak and Eyalt[10] followed 130 patients on chronic amiodarone therapy over a ten-year period, with renal function tests done at least twice yearly. No mention was made of any change in either serum creatinine or BUN levels. Fogoros et al[3] documented serum creatinine elevation in eight of 96 patients treated with amiodarone. The average increase was 0.81 ± 0.33 mg/dL (range, 0.5-1.4 mg/dL). The creatinine increase was sustained and could only be explained by the medication. One of the patients had to discontinue this drug due to mild but progressively worsening renal dysfunction. This patient's serum creatinine level returned to normal over the ensuing two weeks. McGovern et al[4] described azotemia and hypercalcemia in one of 80 patients treated with amiodarone. Within two weeks of stopping the drug, both the serum calcium and the creatinine levels returned to normal. A repeat challenge and subsequent withdrawal of treatment reproduced this sequence of events.

No histopathologic information is available to explain the cause of amiodarone-induced renal dysfunction. Maggioni et al[11] studied the distribu-

---

Address correspondence to Dr Jacobs, Clinical Associate Professor of Medicine, Department of Medicine, Stanford University Medical Center, 300 Pasteur Dr, A309, Stanford, CA 94305.

tion of amiodarone in the tissues of a 60-year-old man who died suddenly while on the drug. The authors found the expected preferential concentration in lipid-rich tissues, namely (in decreasing order of concentration) adipose tissue, lung, bile, pancreas, liver, heart, kidney, aorta, thyroid, muscle, and blood. Microscopic and ultrastructural information is available for amiodarone-related hepatic[7,8] and pulmonary[9] toxicity. There are striking similarities, with both tissues showing osmophilic, granular membranous structures within lysosomes. These inclusions morphologically resemble those described in several primary and secondary phospholipidoses.[8] Similar findings have been reported in the lungs, liver, and kidneys of patients receiving a variety of amphophilic drugs that have some structural features in common with amiodarone (a nonpolar ring system and a hydrophilic cationic side chain).[8] Amiodarone may form a drug-lipid complex within lysosomes, causing cellular injury either through a consequent metabolic derangement or as an epiphenomenon of this interaction.[8,9]

An alternate explanation is that amiodarone causes a rise in serum creatinine levels by competing for proximal tubular secretion (as is seen with cimetidine[12]) or by interfering with the laboratory assay of creatinine. The absence of an active urinary sediment and the rapid reversal of the laboratory findings suggest that an acute inflammatory process was not present in the kidney. Whatever the exact pathogenic mechanism, clinicians should be alert to nephrotoxicity that may occur in association with amiodarone use.

REFERENCES

1. Rotmensch HH, Belhassen B, Swanson BN, et al: Steady-state serum amiodarone concentrations: Relationships with antiarrhythmic efficacy and toxicity. Ann Intern Med 1984; 101:462–469.
2. Harris L, McKenna WJ, Rowland E, et al: Side effects and possible contraindications of amiodarone use. Am Heart J 1983; 106:916–923.
3. Fogoros RN, Anderson KP, Winkle RA, et al: Amiodarone: Clinical efficacy and toxicity in 96 patients with recurrent, drug-refractory arrhythmias. Circulation 1983; 68:88–94.
4. McGovern B, Garan H, Kelly E, et al: A erse reactions during treatment with amiodarone h drochloride. Br Med J 1983; 287:175–180.
5. Martino E, Safran M, Aghini-Lombardi F, al: Environmental iodine intake and thyroid dysfun tion during chronic amiodarone therapy. Ann Inte Med 1984; 101:28–34.
6. Heger JJ, Prystowsky EN, Jackman WM, al: Clinical efficacy and electrophysiology dur long-term therapy for recurrent ventricular tachyca dia or ventricular fibrillation. N Engl J M 1981; 305:539–545.
7. Simon JB, Manley PN, Brien JF, et al: Ami darone hepatotoxicity simulating alcoholic liver d ease. N Engl J Med 1984; 311:167–172.
8. Poucell S, Ireton J, Valencia-Mayoral P, et Amiodarone-associated phospholipidosis and fibro of the liver. Light, immunohistochemical, and electr microscopic studies. Gastroenterology 1984; 86:92 936.
9. Marchlinski FE, Gansler TS, Waxman H et al: Amiodarone pulmonary toxicity. Ann Inte Med 1982; 97:839–845.
10. Leak D, Eydt JN: Amiodarone for refracto cardiac arrhythmias: 10-year study. Can Med Assoc 1986; 134:495–501.
11. Maggioni AP, Maggi A, Volpi A, et al: Ami darone distribution in human tissues after sudd death during Holter recording. Am J Card 1983; 52:217–218.
12. Freston JW: Cimetidine: II. Adverse rea tions and patterns of use. Ann Intern M 1982; 97:728–734.

See also pp 322–324 and 324–325

Fatal *Branhamella catarrhalis* pneumonia

LORRAINE COLI, MD; SUNNY WONG, MD; WILLIAM SCHRAFT, MD; FRANK E. IAQUINTA, MD;
ROBERT D. BRANDSTETTER, MD

Branhamella catarrhalis is a gram-negative diplococcal organism capable of causing pneumonia in patients with underlying illnesses.[1] Although it is believed rarely to cause death,[2] a review of the literature suggests that this organism may be more virulent than initially thought.[3]

We report here a fatal case of *B catarrhalis* pneumonia which occurred in a young man with no known immune disorder. It appears that *B catarrhalis* has the ability to cause fatal pneumonia in otherwise healthy persons.

CASE REPORT

A 34-year-old man was admitted to the hospital after experiencing a witnessed seizure. He was restless and agitated on admission. His medical history was significant for a childhood idiopathic seizure disorder for which phenytoin was prescribed.

On physical examination, the patient's rectal temperature was 38.6°C; blood pressure, 150/90 mm Hg; respirations, 28/min; and pulse rate, 130/min. The neck was supple, and there was no palpable adenopathy. Auscultation revealed coarse breath sounds bilaterally and a regular heart beat. The rest of the examination was normal, except a neurologic evaluation showed that the patient had intermittent periods of lethargy and agitation. He was unable to answer questions and was unaware of his surroundings. The patient responded appropriately to noxious stimuli.

Laboratory studies showed a normal hemoglobin level and a white blood cell count of 3,900/mm^3 with a left shift and 19% ba forms. The platelet count, prothromb time, and partial thromboplastin time we within the normal range. Electrolyte stud revealed a sodium level of [illegible] mEq/L; p tassium, 4.3 mEq/L; chloride, 81 mEq and a carbon dioxide content of 22 mEq The serum osmolality was 270 mosm H$_2$O, and the urine osmolality was [illegible] mosm/kg H$_2$O. Urinalysis showed 2+ k tones and 1–2 white blood cells. Spot ur electrolyte analysis revealed a sodium cou of 11 mEq/L; potassium, 96 mEq/L; a chloride, 5 mEq/L. A chest roentgenogra and electrocardiogram were normal. An a terial blood gas test on 6 L/min of oxygen nasal prongs revealed a respiratory alkalo and a PaO$_2$ of 77 mm Hg. A toxicolo screen of blood, urine, and gastric conten was negative.

The patient's fluid intake was restricte based on a presumed diagnosis of inappr priate secretion of antidiuretic hormone.

From the Department of Medicine (Drs Coli, Wong, Iaquinta, Brandstetter) and Pathology (Dr Schraft), New Rochelle Hospital Medical Center, New Rochelle, NY.

Address correspondence to Dr Brandstetter, Assistant Director of Medicine, New Rochelle Hospital Medical Center, 16 Guion Pl, New Rochelle, NY 10802.

lumbar puncture was performed, and tests for venereal disease, Gram's stain, culture for bacteria, smear for acid-fast bacilli, cytologic studies, and India ink smears were all negative. Approximately eight hours after admission, the patient became lucid and was able to answer questions. He denied drug use or homosexuality, but admitted to drinking alcohol and smoking tobacco. He stated that he had been feeling weak and tired prior to admission, with frequent headaches. He also reported a history of seizures, but could not remember when the last one had occurred.

Nine hours after admission, he became acutely short of breath. An arterial blood gas analysis revealed respiratory alkalosis, with PaO_2 of 44 mm Hg. He was placed on a 60% Venturi mask, and a repeat chest roentgenogram was taken. At the time of the roentgenogram, the patient became apneic. An endotracheal tube was inserted, and he was placed on a ventilator. Breath sounds were equal bilaterally, and suctioning of the endotracheal tube was difficult. The patient's heart rate, which had been at 130 beats/min, dropped to 70 beats/min. Shortly after, he became asystolic and was successfully resuscitated.

Flexible fiberoptic bronchoscopy was performed after the patient's condition had stabilized, and 30 cc of purulent yellow mucoid secretions was aspirated from the right and left main stem bronchi. Gram's stain of aspirated material revealed sheets of polymorphonuclear leukocytes and gram-negative intracellular diplococci which, in pure culture, grew *Branhamella catarrhalis* sensitive to penicillin. Blood cultures were negative. One gram of methylprednisolone sodium succinate was administered for presumed sepsis as well as four million units of aqueous penicillin every four hours intravenously. Hypotension was treated with dopamine and intravenous infusion of normal saline. The chest roentgenogram, taken shortly before the respiratory arrest, showed an infiltrate in the lower lobe of the right lung which was not present on his admission film. On neurologic evaluation after resuscitation, the patient remained unresponsive to noxious stimuli. A computed tomographic (CT) scan of the head, without contrast, was performed and was normal. In view of the irreversible brain damage suffered during cardiac arrest, the patient was formally acknowledged as brain dead based on data from two isoelectric electroencephalograms. With permission from his sister, life support measures were discontinued.

Postmortem examination revealed an acute right lower lobe pneumonia, acute septic splenitis, respiratory brain syndrome, and fatty metamorphosis of the liver. Cultures taken from the right lower lobe at post mortem were sterile. A human immunodeficiency virus antibody analysis was reported as negative; T-cell analysis (T-helper and suppressor cells) was also normal prior to death.

DISCUSSION

Branhamella catarrhalis is a member of the normal flora of the oropharynx and has been called nonpathogenic to distinguish it from disease-producing *Neisseria*, *N meningitidis* and *N gonorrhoeae*. *B catarrhalis* has been cultured from sources other than the oropharynx,[4,5] and it has been responsible for bronchitis and pneumonia in patients with underlying illnesses.[6,7] Infections due to this organism appear to be successfully treated with erythromycin, tetracycline, trimethoprim sulfamethoxazole, cephalosporin, or penicillin (non-beta-lactamase producing organisms).

It is difficult to determine the actual incidence of pneumonia in *B catarrhalis* infections. A literature search disclosed cases of infection and possible infiltrates, with the organism's pathogenic role in causing pneumonia questioned. We believe, however, that we have accounted for most of the cases of pneumonia, and probably all of the deaths caused by pneumonia, due to this organism. At least six reported patients,[6,8-11] including this one, have died as a direct result of this organism's virulence (Table I).

This patient's presentation and outcome are not dissimilar from those of patients listed in Table I. It is curious that six of the patients who died had lower lobe involvement. Four patients (nos. 2, 4, 5, 6) had only lobar disease. A review of reported cases of *B catarrhalis* pneumonia discloses an overwhelming predominance of lower lobe involvement. The organism's presence in the oral cavity and the fact that debilitating disorders may predispose patients to aspiration of oral contents may explain, in part, the lower lobe involvement in these patients.

It was previously thought that *B catarrhalis* infection has a favorable outcome when an underlying debilitating disease is present.[1] The spectrum of illness may be related to circulating bactericidal antibodies, different serotypes—as noted in *N meningitidis*[12] and *S pneumoniae*[13] infection—or to the low levels of the organism in the blood stream. The latter speculation may account for the paucity of positive blood cultures,[2] as was seen in this patient. In addition, the rarity of recognized bacteremia may, in part, be artifactual, since sodium polyanethol sulfonate, the decomplementary substance that is present in most commercially available

TABLE I. Fatal *Branhamella catarrhalis* Pneumonia

Patient	Reference	Age (yr)/Sex	Underlying Condition	Location of Pneumonia	Blood Cultures	Treatment	Postmortem Examination
1	9	64/male	Occult malignancy	Right middle lobe; right lower lobe	Negative	Penicillin and amikacin	No
2	6	64/male	Chronic alcoholism; right pneumonectomy for recurrent lung abscess; chronic obstructive pulmonary disease	Left lower lobe	2 of 3 positive	Penicillin*	No
3	8	64/male	Multiple myeloma	Bilateral lower lobe	Negative	Ampicillin and gentamicin	Yes
4	11	89/female	Muscular dystrophy	Left lower lobe	Negative	Penicillin, cefamandole	No
5	10	64/male	Lymphocytic leukemia	Right lower lobe	Not reported	Not reported	Yes
6	Present	34/male	Seizure	Right lower lobe	Negative	Penicillin†	Yes

* Sensitive to penicillin, ampicillin, erythromycin, cephalosporin.
† Sensitive to penicillin.

culture media for blood, is known to inhibit the growth of related pathogenic *Neisseria*.[14]

For practical purposes, the Gram's stain of the sputum in patients infected with *B catarrhalis* cannot be differentiated from infection with other gram-negative diplococcal organisms, such as *N sicca* or *N meningitidis*. The majority of *B catarrhalis* organisms produce beta-lactamase, and it has thus been reported that some of these organisms are penicillin-resistant.[15] Accordingly, based on Gram's stain of the sputum, it may be prudent to start with second or third generation cephalosporins in treating such patients who present with no neurologic signs, as all three *Neisseria* species are sensitive to this group of antibiotics.[16,17] Adjustment in antibiotic coverage may, therefore, be made based on sputum culture sensitivities.

In conclusion, it appears that *B catarrhalis* can produce fatal pneumonia in an otherwise healthy immunocompetent host. Additional reports are required in healthy as well as previously ill patients to determine the organism's true virulence and whether a need exists for a prophylactic vaccine.[12]

REFERENCES

1. Doern GV, Miller MJ, Winn RE: *Branhamella (Neisseria) catarrhalis* systemic disease in humans. Case reports and review of the literature. *Arch Intern Med* 1981; 141:1690-1692.
2. Nicotra B, Rivera M, Luman JI, et al: *Branhamella catarrhalis* as a lower respiratory tract pathogen in patients with chronic lung disease. *Arch Intern Med* 1986; 146:890-893.
3. Wallace RJ Jr: In honor of Dr Sarah Branham, a star is born. The realization of *Branhamella catarrhalis* as a respiratory pathogen. *Chest* 1986; 90:447-450.
4. Wald ER, Milmoe GL, Bowen A, et al: Acute maxillary sinusitis in children. *N Engl J Med* 1981; 304:749-754.
5. McCague JJ, McCague NJ, Altman CC: *Neisseria* catarrhal urethritis: A case report. *J Urol* 1976; 115:471.
6. Srinivasan G, Raff MJ, Templeton WC, et al: *Branhamella catarrhalis* pneumonia. Report of two cases and review of the literature. *Am Rev Respir Dis* 1981; 123:553-555.
7. Polsky B, Gold JWM, Whimbey E, et al: Bacterial pneumonia in patients with the acquired immunodeficiency syndrome. *Ann Intern Med* 1986; 104:38-41.
8. McNeely DJ, Kitchens CS, Kluge RM: Fatal *Neisseria (Branhamella) catarrhalis* pneumonia in an immunodeficient host. *Am Rev Respir Dis* 1976; 114:399-402.
9. West M, Berk SL, Smith JK: *Branhamella catarrhalis* pneumonia. *South Med J* 1982; 75:1021-1023.
10. Cox PM, Colloff E: *Neisseria catarrhalis* empyema in an immunodeficient host [letter]. *Am Rev Resp Dis* 1979; 120:471-472.
11. Roth RM, Gleckman RA, Hibert DM: *Branhamella catarrhalis:* An unappreciated pulmonary pathogen. *Am Fam Physician* 1984; 30(4):169-173.
12. Centers for Disease Control: Meningococcal polysaccharide vaccines. Recommendation of the Public Health Service Advisory Committee on Immunization Practices. *Ann Intern Med* 1978; 89:949-950.
13. Centers for Disease Control: Recommendations of the Immunization Practices Advisory Committee. Update: Pneumococcal polysaccharide vaccine usage—United States. *Ann Intern Med* 1984; 101:348-350.
14. Eng J, Iveland H: Inhibitory effect in vitro of sodium polyanethol sulfonate on the growth of *Neisseria meningitidis*. *J Clin Microbiol* 1975; 1:444-447.
15. Nash DR, Wallace RJ, Steingbe VA, et al: Isoelectric focusing of β-lactamases from 123 sputum and ear isolates of *Branhamella catarrhalis* recovered in the United States. *Drugs* 1986; 31(supp 3):47-53.
16. Gilrane T, Tracy JD, Greenlee RM, et al: *Neisseria sicca* pneumonia. Report of two cases and review of the literature. *Am J Med* 1985; 78:1038-1040.
17. The choice of antimicrobial drugs. *Med Lett Drugs Ther* 1986; 28(710):33-40.

Bilateral pregnancy in a uterus didelphys

STEPHEN W. GIORLANDO, MD, FAKHRY Y. ALEXANDER, MD

Uterus didelphys bicollis with bilateral intrauterine gestation is estimated to occur once in a million pregnancies.[1] According to Lewenthal et al,[2] and our confirmatory search, only ten such cases have been reported in the world literature.

The case of this patient, who came to us with a history of a termination of a ten-week pregnancy at another institution and no subsequent coitus, is reported to emphasize diagnostic pitfalls and to provide possible insight into the susceptibility of the contralateral myometrium to contractions when one side is evacuated.

CASE REPORT

A markedly obese 17-year-old woman, gravida 1 para 0, was seen for the first time as an outpatient complaining of symptoms of pregnancy, despite a previous dilatation and curettage procedure performed on August 17, 1985 (at another institution) to terminate a pregnancy of ten weeks' duration. Her last menstrual period had been on June 1. Clinically, the size of the uterus was consistent with a pregnancy of about 25 weeks; sonographic measurements of 6.2 cm for the fetal biparietal diameter and 5 cm for fetal femur length were obtained. On vaginal examination, a longitudinal vaginal septum was noted, as well as two cervices, but it was difficult to outline the post-abortal uterus on the right side.

Surgical and pathology reports (based on analysis of products of conception, including villi) from the other institution confirmed that termination of the ten-week gestation had been performed. Post-operative examinations did not disclose the nature of her müllerian anomaly.

Though the prenatal course was uneventful, she went into spontaneous labor at 37 weeks with a breech presentation, and a cesarean section was performed. The female infant, with a 9/10 Apgar score, weighed 6 lb and was in excellent condition. Both halves of the uterus appeared to be contracting vigorously after removal of the placenta. The patient's postpartum course was uneventful.

DISCUSSION

In the seventh to eighth week of embryonic development, the müllerian (paramesonephric) ducts normally fuse (except for the tubal anlage) and extend caudally toward the urogenital sinus, thus forming the uterus, cervix, and upper vagina. Failure of fusion of the ducts at the latter stage causes the condition of uterus didelphys (uterus duplex separatus) with a double upper vagina, as in the present case. The lower third of the vagina is believed to be derived from the urogenital sinus.

Though failure of fusion of the müllerian ducts results in uterine and vaginal abnormalities, renal abnormalities can also occur, including unilateral re-

---

From the Department of Obstetrics and Gynecology, Catholic Medical Center of New York.

Address correspondence to Dr Giorlando, Interboro Medical Group, PC, 2791 Linden Blvd, Brooklyn, NY 11208.

nal agenesis.[3] In some cases, dyspareunia,[4] dysmenorrhea, and leukorrhea are noted, but this patient did not complain of any of these symptoms prior to her pregnancy. Though routine sonography may be of some diagnostic value,[5] its accuracy is considerably enhanced if a vaginal examination has revealed a longitudinal vaginal septum[6] and two cervices.[7]

In a patient with uterus didelphys, pregnancy carries a high risk of complications[8]: premature labor,[9] 23%; breech presentation, 43%; cesarean section, 82%; occasional lower segment torsion;[10] and oxytocin-induced rupture of the uterus.[11] In addition, as illustrated in the present case, formidable diagnostic difficulties are faced by physicians who attempt evacuation of the uterus.

As yet, no urologic anomaly has been found in the patient's postpartum evaluation, which included a negative intravenous pyelogram. The infant is thriving almost a year later. With a fetal survival rate of 67.5% and a perinatal mortality rate of 3.6%, uterus didelphys, even with a unilateral pregnancy, demands careful attention and accurate diagnostic efforts.

REFERENCES

1. Nhan VQ, Huisjes HJ: Double uterus with a pregnancy in each half. *Obstet Gynecol* 1983; 61:115-117.
2. Lewenthal H, Biale Y, Ben-Adereth N: Uterus didelphys with a pregnancy in each horn. Case report. *Br J Obstet Gynaecol* 1977; 84:155-158.
3. Jones TB, Fleischer AC, Daniell JF, et al: Sonographic characteristics of congenital uterine abnormalities and associated pregnancy. *JCU* 1980; 8:435-437.
4. Heinonen PK: Longitudinal vaginal septum. *Eur J Obstet Gynecol Reprod Biol* 1982; 13:253-258.
5. Valdes C, Malini S, Malinak LR: Ultrasound evaluation of female genital tract anomalies. A review of 64 cases. *Am J Obstet Gynecol* 1984; 149:285-292.
6. Heinonen PK: Uterus didelphys. A report of 26 cases. *Eur J Obstet Gynecol Reprod Biol* 1984; 17:345-350.
7. Malini S, Valdes C, Malinak LR: Sonographic diagnosis and classification of anomalies of the female genital tract. *J Ultrasound Med* 1984; 3:397-404.
8. Hochner-Celnikier D, Hurwitz A, Beller U, et al: Uterus didelphys. Ultrasound diagnosis in the case of an adnexal mass as a presenting symptom in early pregnancy. *Eur J Obstet Gynecol Reprod Biol* 1984; 16:339-342.
9. Heinonen PK, Saarikoski S, Pystynen P: Reproductive performance of women with uterine anomalies. An evaluation of 182 cases. *Acta Obstet Gynecol Scand* 1982; 61:157-162.
10. Legarth J, Hansen B: Uterus didelphys. Report of a case with torsion of the uterus occurring during labor. *Acta Obstet Gynecol Scand* 1982: 61:517-518.
11. Duncan ME: Oxytocin-induced rupture of a double uterus in a primigravida. *J R Coll Surg Edinb* 1981; 26:47-49.

The neuroleptic malignant syndrome after open heart surgery: Successful treatment with bromocriptine

ABRAHAM LIEBERMAN, MD; PETER PASTERNACK, MD; STEPHEN COLVIN, MD

Neuroleptic malignant syndrome (NMS) is an idiosyncratic reaction to neuroleptic drugs and is characterized by high fever (to 41°C), unresponsiveness, rigidity, and autonomic dysfunction.[1,2] Associated laboratory abnormalities include elevated serum creatine phosphokinase (CPK) levels and myoglobinuria. The symptoms of NMS may develop from a few days to a few weeks following neuroleptic drug therapy, and they are progressive. NMS occurs in less than 1% of patients who receive neuroleptic drugs and is attributed by some to a primary dopamine receptor blockade in the basal ganglia and hypothalamus, and by others to a disturbance of calcium uptake in skeletal muscle.[3-6] The mortality rate associated with NMS, when not promptly treated, is at least 20%. Treatment consists of the withdrawal of the neuroleptic drug, supportive care, and the use of bromocriptine, a dopamine agonist, or sodium dantrolene. NMS may be difficult to recognize after open heart surgery in a patient in whom high fever, unresponsiveness, and autonomic dysfunction develop. When these abnormalities occur following open heart surgery, the differential diagnosis can encompass infections, ischemia, and metabolic disturbances. In this setting, a drug-induced illness may be easily overlooked.

CASE REPORT

A 67-year-old man was admitted to New York University Hospital for a triple coronary artery bypass and aortic valve replacement. There was no previous history of neurologic or psychiatric disease. Three days after admission, he underwent triple coronary artery bypass surgery and replacement of his aortic valve. The time required to perform the bypass was two hours and 22 minutes, and the aortic cross clamp required 86 minutes. Anesthesia was accomplished with sufentanil citrate, pancuronium bromide, diazapam, oxygen/nitrous oxide inhalation, and droperidol (20 mg).

In the recovery room immediately after surgery, the patient was awake, responded to voice, and spontaneously moved all extremities. However, he was agitated and his behavior was inappropriate. Because of the agitation, he was started on haloperidol, 2 mg, intramuscularly. During the next three days, he received a total of 12 mg of haloperidol. Three days after surgery, he became unresponsive and did not move his extremities. Examination revealed a blood pressure of 130/80 mm Hg; his temperature was 39°C. The patient's face was masked and expressionless. There was rigidity of the neck and of all four limbs, a resting tremor, and bilateral Babinski signs. Within the next few hours, the patient became more rigid, his temperature rose to 40°C, and his blood pressure was 150/90 mm Hg. The serum CPK level was 28,000 units, and CPK MB was absent. Levels of electrolytes, blood urea nitrogen, creatine, calcium, magnesium, and amylase were within normal limits, but there was myoglobinuria. A computed tomographic scan of the head was normal. Lumbar puncture revealed acellular fluid, a protein count of 40 mg%, and a glucose level of 90 mg%. An infectious disease consultant found no evidence of infection.

Because of the history of droperidol and haloperidol use, a tentative diagnosis of NMS was made. The patient was begun on bromocriptine administered through a naso-

---

From the Departments of Neurology (Dr Lieberman), Medicine (Dr Pasternack), and Surgery (Dr Colvin), New York University Medical Center, New York Infirmary-Beekman Downtown Medical Center.

Address correspondence to Dr Lieberman, 650 First Ave, New York, NY 10016.

gastric tube. He received an initial dose of 20 mg, and then received 10 mg four times a day for the next three days, and then 5 mg four times a day for the next four days. Within one day of starting bromocriptine therapy, the patient's temperature dropped to 37°C, and within three days he was afebrile; his CPK level reverted to normal, and he no longer had myoglobinuria. Within one day of starting bromocriptine therapy, the patient was more responsive and spontaneously moved all four limbs. Within three days, he began to talk and his rigidity and tremor disappeared. Within ten days he exhibited no signs of NMS.

DISCUSSION

NMS may result from dopamine receptor blockade in the basal ganglia and hypothalamus.[3-5] This blockade or some other mechanism can then result in inhibition of calcium ion uptake in skeletal muscle, which can then result in rigidity. The rigidity may be so severe as to result in immobility, which may lead to high fever (41°C), dyspnea, cyanosis, decreased oxygen saturation, and death.

Autonomic instability, other than high fever, may include hypertension, tachycardia, and cardiac arrhythmias.[1-2] Other central nervous system symptoms may include tremor and pyramidal tract signs. The rigidity, when present, may lead to large-scale muscle destruction with increased serum CPK levels, myoglobinuria, and even renal failure.[7] Some patients may require hemodialysis, but it should be noted that the hemodialysis has no effect on removing the offending neuroleptics, as these drugs are protein-bound and cannot be removed by dialysis.

Debate has arisen as to whether the high fever and rigidity of NMS originate from a central nervous system effect or a direct effect of neuroleptics on skeletal muscles. Thus, in malignant hyperthermia, which is in many ways similar to NMS, the primary disorder is in the skeletal muscle. In both NMS and malignant hyperthermia there is rapid leakage of calcium from the sarcoplasmic reticulum into the muscle cytoplasm, thereby setting off a chain of events that results in massive muscular contractions, an increased level of CPK, hyperthermia, and myoglobinuria.

Malignant hyperthermia and NMS both respond to sodium dantrolene therapy.[8] Sodium dantrolene impedes the flow of calcium from the sarcoplasmic reticulum into the muscle cytoplasm, thereby counteracting the abnormal muscular contractions. Sodium dantrolene works only on the muscle and only so long as there is calcium left in the sarcoplasmic reticulum; it will not put calcium back into the reticulum.

In NMS, the primary effect is thought to be on the central nervous system, with a secondary effect on skeletal muscles. Not all investigators concur with this hypothesis: Some believe that in NMS, as in malignant hyperthermia, the primary effect is on skeletal muscle. The reason for believing that the primary effect in NMS and in this patient is on the central nervous system is because of the association of NMS with other features of central nervous system dysfunction. This patient had a resting tremor and bilateral Babinski signs. In patients with NMS, a drug such as bromocriptine, which predominantly acts as an agonist at dopamine receptors in the central nervous system, may be the drug of choice, rather than sodium dantrolene.[9] Bromocriptine should be continued for as long as necessary. As the half-life for a drug like haloperidol is between 12 and 24 hours, bromocriptine should probably be continued for at least five days (or five half-lives).

Acknowledgments. The authors thank Drs R. Fox, R. Press, G. Gopinathan, V. Seltzer, and O. Seltzer for their assistance.

REFERENCES

1. Smego RA, Durack DT: The neuroleptic malignant syndrome. *Arch Intern Med* 1982; 142:1183-1185.
2. Mueller PS: Neuroleptic malignant syndrome. *Psychosomatics* 1985; 26:654-662.
3. Henderson VW, Wooten GF: Neuroleptic malignant syndrome: A pathogenetic role for dopamine receptor blockade? *Neurology* 1981; 31:132-137.
4. Geller B, Greydanus DE: Haloperidol induced comatose state with hyperthermia and rigidity in adolescence. *J Clin Psych* 1979; 40:102-103.
5. Burke RE, Fahn S, Mayeux R, et al: Neuroleptic malignant syndrome caused by dopamine-depleting drugs in a patient with Huntington disease. *Neurology* 1981; 31:1022-1025.
6. Toru M, Matsuda O, Makiguchi K, et al: Neuroleptic malignant syndrome-like state following a withdrawal of antiparkinson drugs. *J Nerv Ment Dis* 1981; 169:324-327.
7. Eiser AR, Neft MS, Slifkin RF: Acute myoglobinuric renal failure. A consequence of the neuroleptic malignant syndrome. *Arch Intern Med* 1982; 142:601-603.
8. Gronert GA: Malignant hyperthermia. *Anesthesiology* 1980; 53:359-423.
9. Mueller PS, Vester JW, Fermaglich J: Neuroleptic malignant syndrome. Successful treatment with bromocriptine. *JAMA* 1983; 249:386-388.

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Patient-physician relationship

TO THE EDITOR: I would like to congratulate you on the excellent special issue of the *New York State Journal of Medicine*, Medical Malpractice: An Overview (July 1986).

One of the issues that may have been overlooked in this overview is a statement by Charles Kramer, Esq, in his article entitled, "Medical malpractice: The patient's point of view."[1] The article is written from the point of view of the patient's lawyer, and it repeats one of the myths that trial lawyers like to perpetuate, ie, that the case of *O'Neill v Montefiore Hospital*[2] established in law that a patient-physician relationship may be established merely by a patient or his agent's calling a physician on the telephone. Because of the complexity of this case, such a broad interpretation is not warranted.

The accused, Dr Craig, a member of the Health Insurance Plan (HIP) of New York, was designated by the Plan as the "on-call" physician to attend to HIP emergencies in that area during the night that patient O'Neill had a myocardial infarction and died. Dr Craig had never spoken to nor had he seen the patient before that night, and he knew absolutely nothing about him except that Mr O'Neill was a member of the HIP. Under these circumstances, the only possible previously existing patient-physician relationship between Dr Craig and Mr O'Neill was a presumed contractual agreement between the patient (O'Neill) and his HIP group, of which Dr Craig was a member. The issue here is in what way, if any, did a nurse's telephone call, *per se*, notifying the doctor that the patient was in the hospital emergency room, involve Dr Craig? After noting that the law is settled that a physician who undertakes to examine or treat a patient and then abandons him may be held liable for malpractice,[3] the court in this case opined:

> We are not called upon to decide whether, by contract with HIP Dr Craig assumed to examine and treat the deceased and failed to do so as distinguished from whether he, defendant Craig, independently undertook or assumed to examine and treat and failed to exercise due care in so doing. Perhaps a more complete development of the facts in that regard might have been desirable. Thus whether, as the plaintiff claimed, there was an abandonment of the patient by the doctor, or as the defendant claimed, there was a refusal to accept a proffered examination presents a question of fact which should have been submitted to the jury.[2]

In *O'Neill*, therefore, in granting a retrial the appellate court merely held that it might be determined whether or not a valid patient-physician relationship had been established and consequently whether or not the physician had abandoned the patient. It did not state that a telephone call in itself established that relationship, or even that it might have.

Since physicians at times do give medical advice over the telephone without meaning to undertake to examine or treat a patient, the lesson to those physicians who have had no previous professional relationship with a potential patient is clear. While *O'Neill v Montefiore* did not hold that a telephone call in itself can establish a valid patient-physician relationship, such a conversation may lead to such an allegation. Therefore, without performing a proper history or physical examination, physicians must be extremely wary of providing any medical diagnosis, advice, or treatment over the telephone or implying that they are willing to do so, unless they are also willing to assume the risk of allegation of responsibility for whatever happens to the patient subsequently.

To imply that the judicial opinion in *O'Neill v Montefiore Hospital* held that a valid patient-physician relationship can be established *per se* "merely by a telephone call" between a patient and a physician, however, is patently misleading.

NORMAN S. BLACKMAN, MD
35 Monroe Pl
Brooklyn, NY 11201

[1] Kramer C: Medical malpractice: The patient's point of view. *NY State J Med* 1986; 86:348-351.

[2] *O'Neill v Montefiore Hospital*, 11 AD 2d 132 (1960).

[3] *Meiselman v Crown Heights Hospital*, 285 NY 38999 (see Ann 57 ALR 2d 432-464, 1941).

In reply. Dr Blackman misreads my article if he thinks I said that the *O'Neill v Montefiore*[1] case stands for the proposition that "a patient-physician relationship may be established merely by a patient or his agent's calling a physician on the telephone." That would be an erroneous statement. I was making the point that of the three elements a plaintiff must prove to make a prima facie case of malpractice, the first one is the relationship of physician and patient. I pointed out the various ways in which that relationship could arise. I then said, "Indeed, the relationship *may* [emphasis added] arise merely from a telephone conversation," and went on to recite the facts and holding of the court in the *O'Neill* case.

It is of interest to note that in that case it was the patient's claim that in the telephone conversation, Dr Craig told him to go home and return when the HIP office was open. However, Dr Craig's version was that he offered to come to the emergency room and examine the man but the patient refused, saying he felt better and would prefer to see another physician. The trial judge dismissed the case. The decision was reversed on appeal and a new trial ordered, the court holding that "the jury could have concluded that Dr Craig undertook to diagnose the ailments of the deceased and could have decided whether he abandoned the patient. . . ." [1]

Dr Blackman does indeed make a valid point when he cautions physicians against giving "medical advice over the telephone without meaning to undertake to examine or treat a patient." In fact, he says it better than I did when he writes, "while *O'Neill v Montefiore* did not hold that a telephone call in itself can establish a valid patient-physician relationship, such a conversation may lead to such an allegation."

CHARLES KRAMER, ESQ
233 Broadway
New York, NY 10279-0137

[1] *O'Neill v Montefiore Hospital*, 11 AD 2d 132 (1960).

Medicaid billing

TO THE EDITOR: The Office of General Counsel of the Medical Society of the State of New York is grateful to the Medical Society of the County of Niagara for providing this office with a copy of the decision of the County Court, County of Niagara, in *People of the State of New York v Mehta*, MD (October 1986). The *Mehta* case demonstrates how errors in office billing procedures can lead to claims of overcharges, and possibly a criminal indictment. Fortunately, in *Mehta*, County Court Judge Charles Hannigan had the good sense to dismiss the indictment.

In *Mehta*, the physician was indicted for fraudulently advising the Medicaid system that he had performed comprehensive examinations of patients (at $24.00 or $19.50 per patient) when, in fact, he had only performed routine office visits (at $9.00 per patient). There was no claim that patients were not seen or that medical services were not rendered.

In dismissing the indictment, Judge Hannigan stated that there was no evidence of criminal intent to defraud Medicaid. The evidence, according to Judge Hannigan, only showed that the physician and his secretary had no experience in setting up an office billing system and did not comprehend the codes described in the MMIS Manual. The mistakes had a compounded effect over a period of years, which resulted in a total of $21,000 in overcharges having been paid by Medicaid. Judge Hannigan stated that the defendant should be audited and required to repay the overcharges as a civil remedy.

Finally, Judge Hannigan's decision directed harsh criticism at the Medicaid bureaucracy. He stated:

> We [the State of New York] have created a billion dollar industry. It is apparent from this record that we are attempting to run it by computer. A minimum of personal contact, a modicum of communication would have nipped these problems in the bud.
>
> It is apparent that there is no communication between the Medicaid bureaucracy and the provider of services (the doctor and his staff). I get the impression that our Medicaid program is basically a computer oriented, desk bound bureaucracy. Person to person communication is not built into the system. The result is that the providers of services are confused and misinformed. The mistakes that were found in this office of a sole practitioner are probably to be found in a majority of the offices of small medical practices. It is not uncommon in a small practice to have one secretary, who has little or no experience. We are not dealing with a hospital staff who have their computer department. We are dealing with foreign-born physicians, who are not familiar with our bureaucracies, assisted by secretaries of shallow experience.
>
> The problem is that these people rely on the bureaucracy. The record is replete with the testimony of successive secretarys [sic] who thought that the codes were correct because Medicaid paid them.
>
> I would suggest that a Medicaid representative or auditor visit each physician's office at least twice a year. They should examine the system, which will vary with each small office, audit the billing procedures, and improve or correct the problems. Having made this suggestion, I realize that it will be completely ignored because each segment of the bureaucracy will say that this is not their responsibility. It is far easier to sit at a desk, and program computers than to deal with people and their problems.
>
> It is small wonder that we are wasting millions of dollars in this state on our Medicaid program. This is one example of how $25,000 slipped through the cracks in one office over a two year period. The problem will continue until the bureaucracy that manages the system decides that the providers are not a foreign element and begin to personally communicate with them. Under the present system, they think of each other as the enemy. A true adversarial relationship.

Judge Hannigan's remarks make eminent good sense. Let us hope that the Medicaid bureaucracy is listening.

DONALD R. MOY, JD
General Counsel
Medical Society of the
State of New York
420 Lakeville Rd
Lake Success, NY 11042

Carbon dioxide laser surgery of female genital, perineal, and anal condyloma acuminata

TO THE EDITOR: Within the last decade, the incidence of genital condyloma acuminata has increased at an alarming rate.[1,2] From 1966 to 1978, the number of consultations for condyloma acuminata increased 398% for men and 684% for women.

A variety of treatment regimens have been tried, with limited success, including topical agents such as podophyllum resin, caustic agents such as trichloroacetic acid, chemotherapeutic agents such as bleomycin and 5-fluorouracil, and sensitizing agents such as dinitrochlorobenzene.[1,3,4] Other therapeutic methods include surgical excision—including vulvectomy—cryosurgery, cautery, and immunotherapy using a vaccine. Treatment of genital condyloma acuminata with interferon has been under investigation since the late 1970s. Several investigators have reported success rates of greater than 90% with carbon dioxide (CO_2) laser treatment of condyloma acuminata of the lower genitalia.[5-9]

Case Report. From July 1979 to July 1984, 160 women with condyloma acuminata of the genitalia, perineum, anus, and/or rectal mucosa were treated with CO_2 laser therapy in hospitals affiliated with the State University of New York at Buffalo. Primary condyloma acuminata had been diagnosed in 95 patients. Sixty-five of the 160 women had undergone previous treatment for this disorder: 53 had received podophyllum resin application, four underwent cryotherapy, and eight women had had both these forms of treatment.

The patients' ages ranged from 13 to 55 years. Four of the patients had diabetes: three had juvenile-onset diabetes, and one had class F diabetes. All of the women were sexually active, and eight were pregnant. One of the pregnant patients, who was in the first trimester, opted for termination of the pregnancy with suction curettage at the time of CO_2 laser treatment of the vulva, vagina, and perisacral area. Of the other seven pregnant patients, who were in the second or third trimesters, two underwent CO_2 laser treatment of the cervix only, and five were treated for condyloma acuminata of the cervix, vagina, and vulva.

Ninety-eight patients underwent colposcopic evaluation of the vagina, cervix, and vulva. Condyloma acuminata were detectable only on colposcopy in 16 women: 14 patients had lesions of the cervix, one had a vaginal lesion, and one had a condylomatous lesion of the vulva. Histologic evidence of cervical condyloma acuminata was found on colposcopy in 30 patients with lesions of the vulva, vagina, and perineum. Twenty women had associated cervical intraepithelial neoplasia (CIN): nine with CIN I; nine with CIN II; and two with CIN III. Two patients were found to have mild atypia of the vulva.

Laser treatment was performed using a CO_2 laser system (Cavitron Model AO 33) attached to a colposcope (Carl Zeiss OPML-1), achieving a depth of 2–3 mm, with 800–1,000 watt/cm^2 power density. During the removal of extensive condylomatous lesions, multiple CO_2 laser excisional biopsies were done to rule out underlying invasive or preinvasive neoplasia.

Forty-two patients received general anesthesia, 100 had local anesthesia, and one patient received spinal anesthesia. No anesthesia was required in the treatment of 16 patients with flat condylomatous lesions of the cervix and of one woman with condyloma acuminata of the vagina.

Five patients with extensive condyloma acuminata experienced temperature elevations of 101°F and higher within 24 hours after laser therapy; two of these patients had diabetes. All five patients received empiric intravenous antibiotic therapy; their blood cultures were subsequently found to be negative. In two patients who underwent a total of three laser treatments, temperature elevation occurred after the first and second, but not the third, treatments. Mild vulvar synechia developed in one patient following her first laser treatment; this was transected using laser beams during her second treatment following recurrence (Fig 1).

Thirty patients (18%) had persistence and/or recurrence of lesions within six months of laser treatment. Repeat therapy was effective in 23 patients with only slight recurrences. Only two of seven patients with extensive recurrence or persistence of lesions were successfully treated. Of the five patients for whom repeat therapy failed, four underwent a third laser treatment with complete response, and one patient was subjected to vulvectomy at another institution, but this treatment failed to prevent further recurrence. One patient had a recurrence four years after laser therapy, and her case is presently being followed.

Evaluations of male sexual partners were performed by their urologists. Men without evidence of condylomatous lesions were advised to use condoms during intercourse for a period of six months following CO_2 laser treatment of their female sexual partners.

Discussion. Bellina[8] described persistence of genital condyloma acuminata as appearance of the condition within six months after CO_2 laser treatment, and recurrence as the presence of lesions more than six months after treatment. In an eight-year follow-up of 242 women with condyloma acuminata, Bellina found that 14% of the women required repeat therapy. Following the initial treatment, all patients had at least a one-year disease-free interval, and 65% of patients were disease free for at least two years. Once the male partner had been treated, and the female partner had been retreated, the effective cure rate was 97%.

In three and a half years of experience treating 110 women with condyloma acuminata, Baggish[5] reported a 5.5% (seven patients) recurrence rate. Four recurrences occurred among the 38 patients who had more than one treatment for extensive lesions. Calkins et al[7] reported a 91% overall success rate in 49 women subjected to CO_2 laser therapy.

In the study reported here, the initial cure rate was 82%. After a second laser treatment, which occurred within six months to one year of the initial therapy, the cure rate increased to 96%. Excluding one patient with severe immunosuppression who underwent a vulvectomy, the remaining 4% of patients responded to a third laser treatment; one of these patients suffered a recurrence at the end of the fourth year.

CO_2 laser treatment of the genitalia is not free of complications. The development of pyrexia in five patients is an interesting phenomenon which calls for further investigation. Bellina[8] discussed the possibility of an allergic response to protein absorption. He achieved prompt normalization of temperature with intravenous prednisone therapy, and he recommended administration of glucocorticoids during laser surgery as a preventive measure.

Vigorous and effective treatment of condyloma acuminata is important, since lesions caused by the human papillomavirus have been shown to be associated with cervical and vulvar intraepithelial neoplasia and, occasionally, with invasive squamous cell carcinoma of the lower genitalia. For extensive and disseminated condylomatous lesions of the genitalia, perineum, and/or anus and rectal mucosa, CO_2 laser therapy in single or multiple applications has practical and technical advantages over cryosurgery, cautery, and other surgical methods. Although persistence or recurrence is relatively common within six months of initial CO_2 laser treatment of genital condylomatous lesions, with repeat therapy, the one- to four-and-a-half-year follow-up results are encouraging.

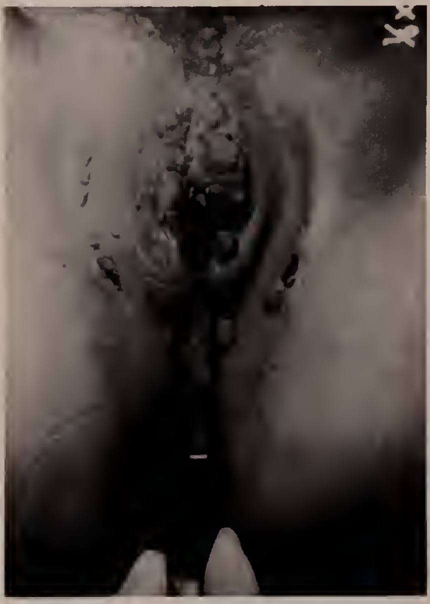


FIGURE 1. Partial fusion of the labia majora following CO_2 laser treatment for persistent condyloma acuminata.

HALUK CAGLAR, MD
MEHMET ERK, MD
MYROSLAW M. HRESHCHYSHYN, MD
Department of Gynecology-Obstetrics
State University of New York
at Buffalo School of Medicine
Buffalo, NY 14209

1. Syrjänen KJ: Current concepts of human papillomavirus infections in the genital tract and their relationship to intraepithelial neoplasia and squamous cell carcinoma. *Obstet Gynecol Surv* 1984; 39:252–265.
2. Condyloma acuminatum—United States, 1966–1981. *MMWR* 1983; 32:306–308.
3. Powell LC Jr: Condyloma acuminatum: Recent advances in development, carcinogenesis, and treatment. *Clin Obstet Gynecol* 1978; 21:1061–1079.
4. Margolis S: Therapy for condyloma acuminatum: A review. *Rev Infect Dis* 1982; 4:S829.
5. Baggish MS: Carbon dioxide laser treatment for condylomata acuminata venereal infections. *Obstet Gynecol* 1980; 55:711–715.
6. Hahn GA: Carbon dioxide laser surgery in treatment of condyloma. *Am J Obstet Gynecol* 1981; 141:1000–1008.
7. Calkins JW, Masterson BJ, Magrina JF, et al: Management of condylomata acuminata with the carbon dioxide laser. *Obstet Gynecol* 1982; 59:105–108.
8. Bellina JH: The use of the carbon dioxide laser in the management of condyloma acuminatum with eight-year follow-up. *Am J Obstet Gynecol* 1983; 147:375–378.
9. Baggish MS: Improved laser techniques for the elimination of genital and extragenital warts. *Am J Obstet Gynecol* 1985; 153:545–550.

Diarrhea caused by group G streptococcus

TO THE EDITOR: Group G streptococci have not been implicated as a cause of bacterial diarrhea. We recently treated a child with prolonged diarrhea whose disease may have been caused by group G beta hemolytic streptococcus.

Case Report. A five-year-old girl was admitted with the chief complaint of intermittent diarrhea of one month's duration. The stools were frequent, small, and contained blood and mucus. She was seen in a clinic two weeks prior to admission and was treated symptomatically without relief. There was no history of contact with any diarrheal illness or pets, and she had not traveled outside the United States.

Physical examination revealed a well-developed, well-nourished child with diminished skin turgor and dry oral mucous membranes. Her temperature was 98°F; pulse, 118/min; respiratory rate, 28/min; and blood pressure, 90/50 mm Hg. Her tonsils and pharynx were normal, and there was no unusual lymph node enlargement. The abdomen was flat, soft, and nontender, with hyperactive bowel sounds. The perineal and anal areas were normal.

Laboratory studies showed a hemoglobin level of 16.1 g/dL and a white blood cell count of 33,300/mm^3 with 67% polymorphonuclear leucocytes and 23% band forms. Blood culture was negative, and urinalysis was normal. Microscopic examination of the stool revealed sheets of polymorphonuclear leucocytes. A stool culture initially produced heavy growth of a beta hemolytic streptococcus. Cultures for *Yersinia* and *Campylobacter* were negative; viral studies were not obtained. The organism was identified as a member of group G by a rapid flocculation method (Streptex, Burroughs Wellcome Co).[1] Three subsequent stool cultures showed heavy growth of group G beta hemolytic streptococci with identical antibiograms (sensitive to all but tetracycline).

The patient was treated with aqueous penicillin G sodium, 50,000 U/kg/day, intravenously for five days, followed by five days of oral medication (penicillin-V). After two days of antimicrobial therapy, stools were becoming normal. A stool culture taken after ten days of treatment was reported negative for group G streptococci and other pathogenic organisms, and the child was well and had normal stools.

Discussion. Group G streptococcus normally inhabits the skin, pharynx, gastrointestinal tract, and vagina,[2,3] and has been implicated as an unusual cause of neonatal sepsis,[4] puerperal infection,[5] pharyngitis,[6] septic arthritis,[7] endocarditis,[8] skin and wound infection, peritonitis, pneumonitis, and empyema.[9] Guss and Larsen[10] recently reported the case of a child with the Down syndrome who had diarrhea apparently caused by a group A beta hemolytic streptococcus. In the absence of stool viral studies in the case reported here, the causal relationship between the child's illness and streptococcal infection is not proven, but the white blood cell count, sheets of polymorphonuclear leucocytes, and heavy growth of group G streptococci from the stool, as well as the prompt response to treatment with penicillin, suggest the possibility of an association. Serologic confirmation of group G streptococcal infection (ASO titres, anti-G carbohydrate response) would be desirable.

GREGORY BARD, MD
ERNEST B. VISCONTI, MD
JOHN P. CURRAN, MD
PASCAL J. DE CAPRARIIS, MD
Department of Pediatrics
Lutheran Medical Center
Brooklyn, NY 11220

1. Castle D, Kessock-Philip S, Easmon CS: Evaluation of an improved Streptex kit for the grouping of beta-haemolytic streptococci by agglutination. *J Clin Pathol* 1982; 35:719–722.
2. Nicholas WC, Steele CP, Bismark BS: Occurrence of groupable beta-hemolytic streptococci. Study among school children in Bismark, ND. *JAMA* 1962; 181:197–205.
3. Norlander IM, Thal E, Tunevall G: Occurrence and significance of hemolytic streptococci groups B-U in human infectious disease. *Scand J Infect Dis* 1975; 7:35–38.
4. Dyson AE, Read SE: Group G streptococcal colonization and sepsis in neonates. *J Pediatr* 1981; 99:944–947.
5. Ramsay AM, Gillespie M: Puerperal infection associated with haemolytic streptococci other than Lancefield's group A. *J Obst Gynaec Brit Emp* 1941; 48:569–585.
6. McCue JD: Group G streptococcal pharyngitis. Analysis of an outbreak at a college. *JAMA* 1982; 238:1333–1336.
7. Nakata NN, Silvers JH, George WL: Group G streptococcal arthritis. *Arch Intern Med* 1983; 143:1328–1330.
8. Lam K, Bayer AS: Serious infections due to group G streptococci. Report of 15 cases with in vitro-in vivo correlations. *Am J Med* 1983; 75:561–570.
9. Armstrong D, Blevins A, Louria DB, et al: Groups B, C, and G streptococcal infections in a cancer hospital. *Ann NY Acad Sci* 1970; 174:511–522.
10. Guss C, Larsen JG: Group A beta-hemolytic streptococcal proctocolitis. *Pediatr Infect Dis* 1984; 3:442–443.

PRIMARY ANAESTHESIA

Edited by Maurice King, MD. 169 pp, illustrated. New York, Oxford University Press, 1986. $13.95 (softcover)

This book is intended to occupy a unique position in the anesthesia literature. The editor, Maurice King, has collaborated with a group of authors who have first-hand experience with organizing and delivering anesthetic care in district hospitals in Africa and India. Their combined experience with the realities of anesthetic care in developing nations is the foundation of a book intended mainly for practitioners who are not anesthesiologists and who must deliver anesthetic care in rural hospitals or in disaster areas. As such this text places a premium on anesthesia methods which are inexpensive, have a large margin of safety, and can be delivered with reusable equipment that is reliable under exacting conditions.

The book is divided into 20 chapters covering topics such as preoperative evaluation of patients, use of local anesthesia, nerve blocks, inhalation anesthesia, intravenous anesthesia, tracheal intubation skills, muscle relaxants, intravenous fluid therapy, and intensive care. The final chapter is a self-evaluation, multiple-choice test with the correct answers provided. There is an appendix covering the preparation of intravenous fluids from tap water and an appendix giving the addresses of various equipment manufacturers. The text is well indexed.

The book is well organized, proceeding from simpler to more complicated concepts and techniques. Essential basic concepts such as "The Ten Golden Rules of Anaesthesia" are emphasized and highlighted. Techniques are described in simple, concise language with ample illustrations of high quality. Common causes for failure of a technique are addressed and corrective measures delineated in a logical progression. Adverse effects, complications, and contraindications to a given technique are treated in the same logical fashion.

Many of the methods described would be considered archaic and less than optimal by anesthesiologists in developed countries. However, they are all proven to be safe, reliable, and capable of being administered with relatively uncomplicated devices at low cost. The text emphasizes that it is not a substitute for individualized instruction by a qualified anesthesiologist. Furthermore, practitioners are urged to refer elsewhere those cases beyond the capabilities of the hospital personnel and facilities.

The book is also of interest to the anesthesiologist in larger hospitals as a source of practical information on techniques infrequently used in everyday practice, such as the inhalation of ether and trichloroethylene. It also provides some perspective for those of us most familiar with "high tech" anesthesia.

Primary Anaesthesia fulfills its stated intention of providing a manual useful to practitioners not trained as anesthesiologists who are called upon to deliver safe anesthesia in a setting with limited resources.

JAMES A. DiNARDO, MD
Beth Israel Hospital
Boston, MA 02215

BASIC SURGERY

By John A. McCredie, MCh, and Gerald P. Burns, MCh. 656 pp, illustrated. New York, Macmillan Publishing Co, 1986. $29.95 (softcover)

Basic Surgery is a 656-page textbook of surgery. According to its preface, it is directed toward students, residents, practicing physicians, and surgical nurses. The editors, Dr McCredie from Ontario and Dr Burns from New York, are well-qualified professors of surgery. Ms Carol Donner is the medical illustrator. The contributor list is impressive, and is composed mostly of distinguished service chiefs and chairmen.

The book is organized into four units covering basic considerations, total care of the surgical patient, principles of general surgery, and principles of specialty surgery. Considerable emphasis is focused upon such basic considerations as management of pain, tissue healing, and immune response. Anatomy and physiology are reviewed in each clinical chapter prior to a generally well-organized discussion of the preoperative, operative, and postoperative implications. The text is concise, yet easily comprehensible. The conclusion of each chapter features not only the usual listing of cited references but also annotated suggestions for further reading that are convenient and practical. The scope is most suitable for medical students and possibly for nurses, but probably not for surgical residents or practicing surgeons.

Both in coverage and in price, this book falls into a category between the 300-page review books and the 2,500-page standard compendia in surgery. This intermediate category is overpopulated with books from just about every medical publishing house, yet *Basic Surgery* surpasses the others. It is the multitude of illustrations that distinguishes this book from its competition. Carol Donner's drawings are attractive and accurate; they clearly and beautifully depict the important anatomic and procedural detail that is described in the text. The price ($29.95) makes it quite a bargain in view of the voluminous figures.

In summary, *Basic Surgery* is a superbly illustrated, well-written, moderate-size surgical textbook that is most appropriate as part of a core curriculum for medical students during an 8-12-week surgical rotation.

RICHARD STILLMAN, MD
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

SHOULDER SURGERY IN THE ATHLETE

Edited by Douglas W. Jackson, MD. 197 pp, illustrated. Rockville, Md, Aspen Systems Corp, 1985. $36.50 (hardcover)

This text is one that should prove useful to orthopedic residents as well as to practicing attendings, not so much as the final word on ailments of the athlete's shoulder, but as a concise, up-to-date source on the highlights of diagnosis, conservative and operative treatment, and proper rehabilitation of the most common shoulder ailments. The contributing authors certainly constituted a "who's who" among shoulder surgeons. The book is well written and easy to read, and the overall quality and clarity of the intra-operative photographs and the illustrations are superb. Perhaps equally important is the fact that the chapters follow a logical progression. The two chapters reviewing the technique and anatomy of shoulder arthroscopy are particularly well done. They identify the various portals, and note what structures are most easily approached, as well as the pitfalls associated with them. In these chapters particularly, the intra-operative photographs, the photographs of cadaver dissections, and the anatomic illustrations are extremely helpful in clarifying a somewhat poorly understood procedure.

The chapter on impingement syndrome, in a few short pages, logically covers the highlights of diagnosis, staging, and treatment. The chapter on rotator cuff tears is particularly well done. The illustrations of the various methods of "closing" the tear according to size, location, and configuration will be of enormous help to the general orthopedist who does such procedures only occasionally. The chapter on rehabilitation simplifies and organizes a complex and generally poorly understood topic. It covers the use of some of the modalities and offers a systematic approach to stretching and strengthening the supportive soft tissues of the shoulder. The brief chapter on the acromioclavicular joint gives a most concise description of the six types of separations as well as a rational approach to determining which injuries should do well with symptomatic treatment and which should be treated more aggressively. Also of interest is the historical review of the changing trends of treatment of this entity.

Perhaps more could be said regarding multidirectional instability of the shoulder. With a separate chapter regarding its diagnosis, treatment, and rehabilitation, in my opinion, the book would be complete.

REGINALD MANNING, MD
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

SPINE. FAILED BACK SURGERY SYNDROME: EVALUATION AND TREATMENT

Edited by Arthur H. White, MD. 179 pp, illustrated. Philadelphia, Hanley & Belfus, Inc, 1986. $30.00 (hardcover)

This thoughtfully written and often thought provoking collection of 12 chapters written by individual authors addresses the challenging topic of the pathophysiology, diagnosis, and treatment of the failed back surgery syndrome. Included are discussions of diagnostic modalities including the techniques, value, and limitations of electrophysiologic tests; diagnostic block procedures; CT and MRI imaging; quantitative devices for physical assessment; and psychological assessment tests and techniques. From a therapeutic standpoint the book discusses inpatient management of chronic pain, medications, and a functional restorative program, and there is a very brief discussion of surgery. The final chapter propounds a series of brief discussions of diagnoses and approaches to therapy of a list of conditions possibly responsible for the failed back surgery syndrome. The authors are orthopedists, radiologists, and other specialists active in the management of lumbar spine and failed back surgery problems; the neurosurgical perspective is not presented.

The emphasis on the limited role of surgery is appropriate, but the discussion of surgical possibilities, especially neurosurgical procedures, is quite sparse. The statement that the first operating surgeon is "the worst person to make the decision whether to re-operate . . ." seems dangerous, since it implies carte blanche to the first surgeon to operate, without responsibility for the consequences or further care of the patient whose surgery fails.

The chapter on needle diagnosis includes a discussion of the little-used thiobarbital examination for pain exaggeration and makes the unsubstantiated claim that epidural block techniques can differentiate anterior-element from posterior-element pain. The value of discography is touted in this and other chapters. While the diagnostic discussion is very good, only oblique reference is made throughout the book to the therapeutic use of needle procedures. This is a major omission.

The concept of "internal disruption," termed "the discogenic pain syndrome" in other texts, is emphasized by several authors. This seems valid despite the controversial nature of this diagnosis, which is not universally accepted. In contrast, there is only passing mention of the phenomenon of aseptic discitis.

The chapter on psychological assessment includes a valuable discussion of available written psychological tests of personality, pain intensity, and social impact as well as important diagnostic factors to be assessed during a patient interview. The book includes a discussion of inpatient psychological therapy but offers little guidance for outpatient therapy or office psychotherapy by the surgeon or nonpsychiatrist.

Overall this book should be extremely valuable to all those who deal with these difficult problems. Its brevity makes it readable, but it per force omits much of the valuable detail found in more extensive texts on back disease (viz Finneson, Mcnab) or the failed back surgery syndrome (Wilkinson).

HAROLD ARTHUR WILKINSON, MD, PhD
University of Massachusetts
Medical Center
Worcester, MA 01605

ADVANCES IN UPPER EXTREMITY SURGERY AND REHABILITATION

Edited by John A. Boswick, Jr, MD. 303 pp, illustrated. Rockville, Md, Aspen Publishers, Inc, 1986. $43.50 (hardcover)

Dr John A. Boswick, Jr, Attending Surgeon at St Joseph's General Rose and Veteran's Hospital and former Professor of Surgery and Chief of Hand Surgery at the University of Colorado, has edited an interesting text based on the annual Rocky Mountain International Symposium held in Colorado in August 1984. Numerous international experts present their views on a variety of subjects ranging from global topics such as rheumatoid arthritis to the more esoteric ones such as surgical management of the arthrogrypotic thumb. The text is arranged to parallel the anatomy under discussion, the initial chapters dealing with rheumatoid disease of the shoulder, rotator cuff tears, and total shoulder replacements, and progressing through arthritis of the elbow to problems in the fingers and hands. The last chapters deal with comprehensive rehabilitation of the extremities, work evaluations, and joint protection in rheumatoid disease. Each of the authors seem to have utilized a somewhat different format for their chapters, and as such the style is somewhat uneven. Some authors take the opportunity to describe their individual approaches to a problem, pointing out technical pearls and specific points of interest. Of necessity, these chapters assume a significant amount of basic knowledge and experience. Others, such as the chapter on extensor tendon injuries, are broader and more general, and might be useful to a less sophisticated reader. The chapter on carpal kinematics is presented in a very distinct and clear fashion, which is unusual for a topic of this complexity. It might, however, have benefited from illustrations.

There is a great deal of extremely valuable information in this text. Many problems that are addressed are seen every day by hand surgeons. Useful information by an experienced international cast of contributors is in abundance. This text would be most useful to a thoroughly experienced hand surgeon who is looking for ways to improve his techniques. Because the material is composed of fragmented selections rather than comprehensive material, it would be of less value to the resident or surgeon who is not well acquainted with and experienced in this area. The text was prepared from material originally formulated for a symposium, which included audiovisual aids, and thus some of the material is presented with less than optimal clarity. However, there is no doubt about the value of this book, and I recommend it without reservation.

STANLEY GORDON, MD
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

ATLAS OF STAPLING TECHNIQUES

By Pedro A. Rubio, MD. 368 pp, illustrated. Rockville, Md, Aspen Publishers, Inc, 1986. $95.00 (hardcover)

The author has successfully written a uniquely comprehensive source book of operative procedures that involve stapling techniques. The instrumentation available from American manufacturers has been integrated into an atlas-type display of 113 operations. An introduction precedes the atlas, and each operative procedure is accompanied by a useful commentary. The abundant line drawings are outstanding for their clarity and utility.

Although there are differences of opinion about the merits of stapling, this technique is clearly here to stay. The proliferation and acceptance of stapling methods has undoubtedly contributed to the avoidance of permanent colostomy for many patients with mid-rectal cancers. However, this reviewer would like to see evidence that stapling techniques contribute to a quicker recovery and shortened hospital stay, as is indicated in the foreword of the book. It is unclear at present whether stapling methods shorten operative time when compared to the skillful use of suture techniques.

Nonetheless, this text will serve as an exceptionally valuable, even unique, resource for surgeons who use stapling techniques. The price may seem high, but considering the scope of the book I believe the cost is reasonable. This atlas is a valuable contribution to the field, and I recommend it to all surgeons who use these techniques in the operating room.

JEROME DECOSSE, MD
The New York Hospital-
Cornell Medical Center
New York, NY 10021

PRACTICAL MANAGEMENT OF EYE, EAR, NOSE, MOUTH AND THROAT EMERGENCIES

Edited by Thomas O. Stair, MD. 183 pp, illustrated. Rockville, Md, Aspen Systems Corporation, 1986. $27.50 (hardcover)

The stated purpose of this book is to fill a gap in the fields of ophthalmology, otolaryngology, and oral and maxillofacial surgery from an emergency room perspective. The editor states that the book was primarily written by emergency room physicians and then edited and rewritten by specialists in the various fields. I thus expected that I would be able to use it as a reference for particular types of emergency problems. However, there is a plethora of information in the book which included topics from the mundane to the esoteric. I do not believe that this is just an emergency textbook, but one that covers a large range of subjects, and usually not in enough detail to permit the practical treatment of patients.

The book is divided into five chapters covering emergencies involving the eye, ear, nose and paranasal sinuses, mouth, and throat. There is also a chapter on facial trauma. At the end of each chapter is a bibliography which can be useful for in-depth follow-up of any of the subjects covered. The chapters themselves are divided into topics. When these topics are of an emergent nature I find that the book is helpful in elucidating proper treatment. However, when the problem is one of a diagnostic dilemma or one that requires specialized treatment, the book gets bogged down in trying to make the reader an instant expert in areas where specialists would find the same problems controversial and perplexing. For example, in discussing sinusitus, the author also discusses chronic granulomatous disease, common variable hypogammaglobulinemia, chronic mucocutaneous candidiasis, etc. This is not appropriate for an emergency room textbook.

One is left with the impression that some of the information in the text is taken from other authoritative texts as opposed to representing the authors' first-hand information. More would have been achieved, perhaps, if specialists had written the chapters from an emergency perspective, as opposed to emergency physicians' writing from the perspective of specialists. Most of the information seems quite correct. However, if a patient came into the emergency room with a problem discussed in the text I would not find this book helpful in a practical sense unless I were already well trained in handling that particular problem.

In conclusion, I think that this text is a good attempt to provide information in an area that has probably been neglected. However, a more successful and practical book would be one that limits itself to the ophthalmological and otolaryngological problems encountered by emergency room physicians.

IRA H. BUCHALTER, MD
Maimonides Medical Center
Brooklyn, NY 11219

Autograft procedure seen as new mode of therapy for Parkinson disease

A surgical procedure in which fragments of the adrenal medulla are autotransplanted to the right caudate nucleus of the brain may offer hope to victims of Parkinson disease (PD). The procedure yielded marked clinical improvement in the signs of the disease in two patients in Mexico. Investigators Madrazo et al (*N Engl J Med* 1987; 316:831–834) reported reduced rigidity, akinesia, and tremor—classic signs of the impaired motor function associated with PD—in two young patients with intractable and incapacitating Parkinson disease.

If further studies corroborate these findings, and if long-term follow-up shows continued improvement, neural transplantation could represent a significant advance in the treatment of PD. Parkinson disease is characterized by deficient levels of the neurotransmitter dopamine in the basal ganglia and cell loss in the substantia nigra. Currently, therapeutic options for PD consist of dopamine replacement therapy through the administration of the dopamine precursor levodopa and the recent introduction of dopamine-receptor agonist therapy. Although both these methods of treatment have proven effective, they merely control the symptoms of the disease—and can produce a variety of adverse effects—while the disabling disease continues to progress.

In the 1970s, researchers developed an animal model of PD by chemically destroying regions of the substantia nigra. They then demonstrated that transplants with tissue from fetal substantia nigra or adrenal medulla restored normal motor function in the animals. This procedure has now apparently been replicated in human patients.

In an editorial (*N Engl J Med* 1987; 316:872–873) accompanying the Madrazo study, Robert Y. Moore, MD, PhD, from the State University of New York at Stony Brook, analyzes the significance of this report. He describes the findings as "dramatic" and "remarkable." Moore questions the authors' proposed mechanism by which the transplants restored motor function. The theory of Madrazo et al holds that "adrenal medullary cells differentiate as dopaminergic neurons and innervate the adjacent neostriatum." Although such a mechanism is plausible, Moore finds it unlikely that "small grafts placed at the surface of the neostriatum would provide a widespread innervation of such a large structure." In addition, this mechanism would not account for the bilateral improvement in the signs of Parkinson disease in these patients.

Moore cites an alternative theory: The transplanted adrenal medullary tissue produces a growth promoting substance that diffuses via the cerebrospinal fluid and promotes growth and regeneration of intrinsic dopaminergic axons. Examining the implications of such a mechanism, Moore projects that autotransplantation of adrenal medullary tissue would probably not be effective in patients with advanced PD, in whom there is widespread destruction of nigrostriatal axons, and that in the long term the transplants could lose their effectiveness with progression of the disease.

Based on the successful results reported by Madrazo et al and the potential implications of such a new therapeutic method, additional controlled studies are needed to assess the effectiveness of neural transplantation as a treatment for patients with Parkinson disease.

Physician payment reform

There is rising societal and governmental pressure for physician payment reform. Health care costs are skyrocketing, and the payment system currently in operation rewards physicians for overutilizing valuable resources. In a recent editorial in *Annals of Internal Medicine* (1987; 106:468–469), William B. Stason, MD, MS, from the Harvard School of Public Health, lays the groundwork for physician payment reform.

"The goal should be to create a physician payment system that adequately recognizes the physician's worth to society, fairly reflects the value of individual services and specialties, and is relatively easy to administer." Stason supports the approach of a relative value scale for medical services. The scale should be based primarily on cost as well as on the benefits of a service to the patient, rather than on the existing charge structure. Many diagnostic and surgical procedures are currently overpriced, Stason notes, and there are significant discrepancies among fees for certain medical services. These factors weigh against a charge-based relative value scale.

A cost-based relative value scale would factor in the time expended by a physician—taking into account the "skill, physical and mental effort, and risk involved in providing a given service"—overhead costs, and training expenses. All of these variables could then be updated based on the changing health care marketplace and advances in medical technology. Other factors to be considered would include patient preferences and financial incentives for quality of care and outcome records.

Whatever the method used to achieve physician payment reform, gradual change is essential, Stason emphasizes. "Dramatic changes will be disruptive to physicians and may compromise patients' access to care."

Have prospective reimbursement systems affected physicians' awareness of health care costs?

One of the theories behind prospective reimbursement systems is that if physicians are more aware of the costs of hospital services, they will be more likely to reduce ordering of tests and, in general, to offer more cost-effective care. Retrospective reimbursement encourages consumption of services. However, Thomas and Davis report that prospective reimbursement has not led to increased physician awareness of costs (*Med Care* 1987; 25:181–184).

According to the authors, "physicians must bear the major responsibility for setting levels of expenditures through their control over admitting patients to hospitals and nursing homes, ordering tests, prescribing drugs, and assigning return visits." Hospital charges account for 42%

NEWS BRIEFS are compiled and written by Vicki Glaser.

of overall health care costs, with about a quarter of those charges stemming from ordering of tests—specifically, laboratory and radiologic services. Therefore, physicians can play a major role in reducing health care expenditures.

Thomas and Davis distributed a questionnaire—aimed at assessing physician awareness of hospital costs—to faculty members and residents at a medical center, following the institution of a prospective reimbursement system. The authors sought cost estimates of both common and less common tests, of facility charges—including room fees, outpatient services, and emergency care—and of common prescription drug regimens. A correct estimate could fall within 20% of the actual cost. The results were as follows: 34% correct responses for common tests; 24% for less common tests; 35% for hospital facility charges; and 17% for drug expenditures. "Lack of effective economic education, lack of feedback, administrative policies, and lack of financial incentives" are the most common reasons given to explain poor physician cost awareness.

AIDS risk following therapy with blood-based factor concentrates

Persons with hemophilia, whose blood lacks sufficient amounts of clotting factors VIII and/or IX, must obtain these components from factor concentrates that have been isolated from donor blood. With the advent of the AIDS epidemic, these individuals are at increased risk of exposure to human immunodeficiency virus (HIV)—the virus that causes AIDS. In 1984, increasing awareness of this risk led to the introduction of heat-treated factor concentrates. Now the Centers for Disease Control (CDC) has reported HIV seroconversion following the use of heat-treated factor concentrates (*MMWR* 1987; 36:121–124). The CDC surveyed several hemophilia treatment centers outside the United States, because of the earlier use of heat-treated factor concentrates in some countries.

Twenty-three patients were identified as having had their first positive HIV antibody test after receiving heat-treated factor concentrate. Of these 23, three pediatric patients were shown with certainty to be seronegative from eight to 16 months after first receiving heat-treated factor VIII concentrate and, subsequently, to become seropositive during a period from ten to 22 months after this treatment. The three patients have fared as follows: one has AIDS; one has lymphadenopathy and encephalopathy; and one is asymptomatic with normal T-cell counts. It could not be determined with certainty whether infection occurred prior to or following the introduction of heat-treated concentrates, because the seroconversion latency period among persons with hemophilia is unknown.

Since the development of a test for HIV antibodies in blood samples, there have been no reported cases of seroconversion following treatment with donor-screened, heat-treated factor concentrates.

Potential AIDS drug derived from egg yolk

An experimental drug, called AL-721, has been effective in treating seven patients with advanced acquired immunodeficiency syndrome (AIDS). The lipid-based drug, which is derived from egg yolks, was developed by Meir Shinitsky, a professor at the Weizmann Institute of Science in Rehovot, Israel. Shinitsky hypothesizes that AL-721 eliminates the cholesterol from the membrane of the AIDS virus, preventing the virus from invading T-cells.

Following improvement in the conditions of the initial seven patients treated with AL-721, the Israeli Health Ministry approved the treatment of an additional group of 14 patients with AIDS. A ministry spokesman, however, cautioned against drawing premature conclusions as to the efficacy of this drug. Researchers in the United States are awaiting presentation of Shinitsky's data in a scientific format, including control patients and appropriate statistical analysis, before expressing any optimism.

Praxis Pharmaceutical Co. (Beverly Hills, Calif) is awaiting approval from the Food and Drug Administration to institute a large-scale test of AL-721 in the US. As yet, the drug has produced no adverse side effects.

FDA approves drug against lower respiratory infections caused by *B catarrhalis*

Augmentin®, a broad spectrum oral antibiotic, has received FDA approval for the treatment of lower respiratory infections, such as bronchitis and pneumonia, that are caused by *Branhamella catarrhalis.* Once considered a nonpathogenic bacterium commonly found in the nasopharyngeal region, *B catarrhalis* is now recognized as a common cause of otitis media, sinusitis, and lower respiratory infections.

B catarrhalis is resistant to antibiotic therapy: 75% of *B catarrhalis* isolates produce beta lactamase, an enzyme that inactivates antibiotics such as cephalosporins and penicillins. Augmentin® was first approved by the FDA in 1984 for treatment of a variety of infections due to resistant bacterial strains, including otitis media and sinusitis caused by beta lactamase producing *B catarrhalis.* Augmentin® (Beecham Laboratories, Bristol, Tenn) is a combination drug composed of clavulanate potassium and amoxicillin. The clavulanate potassium component binds to the troublesome beta lactamase, rendering the bacteria vulnerable to the antibiotic amoxicillin.

In this issue of the *Journal*, Coli et al (1987; 87:359–361) report on a fatal case of *B catarrhalis* infection.

"Vocal" antibodies used to track tumor tissue

Using a technique that incorporates radioactively labeled antibodies and a pen-shaped probe that makes noise when held near a source of radioactivity, physicians can detect even very small tumors—some less than one millimeter in diameter (*Science News* 1987; 131:232). Researchers at Ohio State University in Columbus developed the technique, in which patients with colorectal cancer who are about to undergo surgery receive injections of radioactive antibodies that will bind to the tumor tissue. Then, during surgery, the probe is used to locate antibody-bound tumor cells.

The probe has been able to accurately delineate the boundaries of tumors in 76% of an initial group of 54 patients. Further study is needed to expand the use of the probe to include other types of cancer and to assess the impact of this technique on cancer survival rates. In the future, the antibody/probe system might be able to reduce the need for exploratory surgery.

MSSNY House of Delegates convenes for annual meeting

The 181st session of the House of Delegates annual meeting was held March 12–15, 1987. The proceedings included the introduction of 94 resolutions and two commendation resolutions. Several key resolutions are reported below.

- That the MSSNY oppose legislation requiring that physicians accept assignment and reimbursement from Medicare or any other third-party payor as a condition for medical licensure or relicensure, as well as opposing legislation that would establish penalties for not accepting such assignments.
- That the MSSNY support the Board of Regents as having the ultimate authority to revoke, suspend, and reinstate physicians' licenses, as is the current system.
- That the MSSNY continue to actively support appropriate tort law reforms to ease the professional liability insurance crisis in New York State and, specifically, that the MSSNY introduce a resolution at the AMA House of Delegates meeting in June 1987 in support of any such congressional legislation, including the Mrazek bill (HR 2659).
- That the MSSNY encourage the New York Medical Political Action committee to provide financial assistance to viable physician candidates whose concepts and views are acceptable to organized medicine, so that such candidates can pursue elective office.
- That the MSSNY introduce a resolution at the AMA House of Delegates meeting requesting the AMA to institute an AIDS Public Awareness and Information Program, and that the MSSNY establish such a program on a local level in cooperation with the New York State Department of Health.
- That the MSSNY encourage the New York State legislature and the governor to institute strict anti-smoking measures in public places and in the workplace and to change the minimum allowable age for the purchase of tobacco products in New York State from 18 to 20 years; encourage the Hospital Association of New York State to urge its member hospitals to ban all smoking within all member hospitals; go on record as being opposed to smoking in airplanes; and support current legislation that would prohibit advertising and promotion of all tobacco products.

Council approves report on ethical considerations of reproductive technologies

On April 23, the Council of the Medical Society of the State of New York approved an educational and informational report on ethical considerations of reproductive technologies. The report was prepared by the society's Committee on Bioethical Issues which is chaired by Fred Rosner, MD, Director, Department of Medicine, Queens Hospital Center Affiliation of the Long Island Jewish Medical Center. The report provides an overview of the many legal, moral, ethical, and social questions involving reproductive biological procedures. It examines the findings of a number of groups that have studied these issues including the Warnock Committee of Inquiry into Human Fertilization and Embryology, the American Medical Association's Judicial Council, the Ethics Committee of the American College of Obstetricians and Gynecologists, and the Ethics Committee of the American Fertility Society. Although the report does not make specific recommendations, it carefully presents the recommendations of other groups that have studied these issues. The report will be published in its entirety in a future issue of the *Journal*.

MEDICAL SOCIETY NEWS is compiled and written by Vicki Glaser.

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Emanuel Joseph Amoury, MD, Staten Island. Died March 15, 1987; age 91. Dr Amoury was a 1921 graduate of Fordham University School of Medicine. He was a Qualified Fellow of the International College of Surgeons and a member of the Richmond County Medical Society and the Medical Society of the State of New York.

Samuel Atkin, MD, New York. Died March 5, 1987; age 89. He was a 1924 graduate of State University of New York Health Science Center at Buffalo. Dr Atkin was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the Academy of Medicine, the American Psychoanalytic Association, the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Jacob Anthony Begner, MD, New York. Died March 24, 1987; age 78. Dr Begner was a 1935 graduate of Temple University School of Medicine, Philadelphia. He was a Fellow of the American College of Surgeons, a Diplomate of the American Board of Urology, and a member of the Pan American Medical Association, the American Urological Association, the New York County Medical Society, and the Medical Society of the State of New York.

Winifred Bronson, MD, Bronx. Died December 6, 1986; age 81. Dr Bronson was a 1933 graduate of Columbia University College of Physicians and Surgeons, New York. She was a member of the New York County Medical Society and the Medical Society of the State of New York.

Emanuel Dickler, MD, Southbury, Connecticut. Died February 3, 1987; age 75. Dr Dickler was a 1940 graduate of the Medical School, American University of Beirut. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Anczel Dresner, MD, Ozone Park. Died November 15, 1986; age 79. He was a 1936 graduate of Facolta di Medicina e Chirurgia dell'Universita di Bologna. Dr Dresner was a member of the American Academy of Family Practice, the American Geriatrics Society, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Dominic R. Elia, MD, Staten Island. Died February 23, 1987; age 67. Dr Elia was a 1949 graduate of University of Alabama School of Medicine, Birmingham. He was a member of the Richmond County Medical Society and the Medical Society of the State of New York.

Wilbur Jerome Fisher, MD, Buffalo. Died February 24, 1987; age 82. He was a 1930 graduate of Johns Hopkins University School of Medicine, Baltimore. Dr Fisher was a Diplomate of the American Board of Pediatrics and a member of the American Academy of Pediatrics, the American Pediatric Society, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Margaret Evelyn Fries, MD, New York. Died March 2, 1987; age 91. She was a 1920 graduate of Cornell University Medical College, New York. Dr Fries was a Diplomate of the American Board of Psychiatry and Neurology. She was a member of the Academy of Medicine, the American Academy of Pediatrics, the American Psychoanalytic Association, the American Psychiatric Association, the American Academy of Child Psychiatry, the American Medical Women's Association, the New York County Medical Society, and the Medical Society of the State of New York.

Ferdinand Geiger, MD, Syracuse. Died March 25, 1987; age 79. Dr Geiger was a 1936 graduate of the University of Pennsylvania School of Medicine, Philadelphia. He was a Diplomate of the American Board of Nuclear Medicine and a member of the Academy of Medicine, the Society of Nuclear Medicine, the Onondaga County Medical Society, and the Medical Society of the State of New York.

M. J. Gerbasi, MD, Rochester. Died March 6, 1987; age 77. Dr Gerbasi was a 1935 graduate of the University of Rochester School of Medicine-Dentistry, Rochester. Dr Gerbasi was a member of the Academy of Medicine, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Harry Gershman, MD, New York. Died March 6, 1987; age 74. He was a 1938 graduate of New York University School of Medicine, New York. Dr Gershman was a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the Academy of Medicine, the American Psychiatric Association, the Academy of Psychoanalysis, the New York County Medical Society, and the Medical Society of the State of New York.

Leon Goldberg, MD, Miramar, Florida. Died March 10, 1987; age 82. Dr Goldberg was a 1930 graduate of State University of New York Health Science Center at Brooklyn. He was a Fellow of the American College of Chest Physicians and a member of the New York Society for Thoracic Surgery, the American Thoracic Society, the New York County Medical Society, and the Medical Society of the State of New York.

June Helene Hagedorn, MD, Levittown. Died March 28, 1987; age 69. She was a 1943 graduate of State University of New York Health Science Center at Brooklyn. Dr Hagedorn was a Diplomate of the American Board of Obstetrics and Gynecology and a member of the New York State Surgical Society, the Nassau County Medical Society, and the Medical Society of the State of New York.

Frank Coleman Hamm, MD, Shelter Island. Died March 4, 1987; age 86. Dr Hamm was a 1925 graduate of the University of Michigan Medical School, Ann Arbor. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Urology. His memberships included the Academy of Medicine, the American Association of Genito-Urinary Surgeons, the American Urological Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Abraham Karver, MD, Brooklyn. Died March 8, 1987; age 80. Dr Karver was a 1932 graduate of New York Medical College, Flower and Fifth Avenue Hospitals, New York. He was a Fellow of the American Academy of Family Practice and a member of the New York Cardiological Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Thurston L. Keese, MD, Fayetteville. Died October 3, 1986; age 79. He was a 1935 graduate of State University of New York Health Science Center at Syracuse. Dr Keese was a member of the American Academy of Family Practice, the Onondaga County Medical Society, and the Medical Society of the State of New York.

James H. Kidder, MD, Riverdale. Died March 27, 1987; age 85. Dr Kidder was a 1928 graduate of Cornell University Medical College, New York. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Aaron E. Koblentz, MD, New York. Died February 9, 1987; age 78. He was a 1943 graduate of Chicago Medical School, Chicago. Dr Koblentz was a Diplomate of the American Board of Psychiatry and Neurology and a member of the American Psychiatric Association, the Academy of Psychoanalysis, the New York County Medical Society, and the Medical Society of the State of New York.

Charles Joseph Leslie, MD, Southhold. Died March 10, 1987; age 81. Dr Leslie was a 1932 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Pediatrics and a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Herbert Lourie, MD, Syracuse. Died March 5, 1987; age 58. He was a 1952 graduate of Duke University School of Medicine, Durham. Dr Lourie was a Diplomate of the American Board of Neurological Surgery. His memberships included the American Neurological Association, the American Association of Neurological Surgeons, the American Academy of Neurological Surgery, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Robert N. Monaco, MD, Jamaica. Died March 1, 1987; age 61. Dr Monaco was a 1950 graduate of State University of New York Health Science Center at Brooklyn. He was a Fellow of the American College of Physicians, a Diplomate of the American Board of Internal Medicine, and a member of the New York State Society of Internal Medicine, the American Society of Internal Medicine, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Saul Charles Newman, MD, Massapequa Park. Died February 4, 1987, age 86. He was a 1929 graduate of the University of Maryland School of Medicine, Baltimore. Dr Newman was a Fellow of the American College of Surgeons, a Diplomate of the American Board of Otolaryngology, and a member of the American Academy of Otolaryngology, the Suffolk County Medical Society, and the Medical Society of the State of New York.

John Joseph O'Brien, MD, Syracuse. Died March 1, 1987; age 57. Dr O'-Brien was a 1956 graduate of Albany Medical College of Union University, Albany. He was a Diplomate of the American Board of Internal Medicine and a member of the Onondaga County Medical Society and the Medical Society of the State of New York.

Rudolph Henry Pelzer, MD, Yonkers. Died February 9, 1987; age 74. He was a 1943 graduate of New York University School of Medicine, New York. Dr Pelzer was a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Plastic Surgery. His memberships included the Academy of Medicine, the American Society of Maxillofacial Surgeons, the American Society of Plastic and Reconstructive Surgery, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Jay Robert Rabinowitz, MD, Miami, Florida. Died March 1987; age 87. Dr Rabinowitz was a 1924 graduate of New York Medical College, Flower and Fifth Avenue Hospitals, New York. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Joseph Rescigno, MD, Flushing. Died March 8, 1987; age 70. He was a 1946 graduate of Georgetown University School of Medicine, Washington. Dr Rescigno was a Diplomate of the American Board of Internal Medicine and a member of the New York State Society of Internal Medicine, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Clarence E. Sanford, MD, Buffalo. Died February 13, 1987; age 69. He was a 1950 graduate of State University of New York Health Science Center at Buffalo. Dr Sanford was a Fellow of the American College of Angiology and the American College of Surgeons, a Qualified Fellow of the International College of Surgeons, and a Diplomate of the American Board of Surgery. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Roswell L. Schmitt, MD, Old Greenwich, Connecticut. Died February 19, 1987; age 93. Dr Schmitt was a 1919 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American College of Surgeons and a member of the Medical Society of the County of Orange and the Medical Society of the State of New York.

Wilbur Kenneth Smith, MD, Rochester. Died July 20, 1986; age 84. Dr Smith was a 1927 graduate of Johns Hopkins University School of Medicine, Baltimore. He was a member of the American Neurological Association, the American Academy of Neurology, the American Physiological Society, the Association for Research in Nervous and Mental Disease, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Michael Smitonick, MD, Bayside. Died February 27, 1987; age 75. He was a 1939 graduate of St Louis University School of Medicine, St Louis. Dr Smitonick was a Fellow of the American Academy of Family Practice and a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Robert Stock, MD, Long Island City. Died January 21, 1987; age 68. Dr Stock was a 1943 graduate of New York University School of Medicine, New York. He was a Diplomate of the American Board of Internal Medicine and a member of the Association of Life Insurance Medical Directors of America, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Robert Julius Strobos, MD, Valhalla. Died March 15, 1987; age 66. He was a 1947 graduate of Faculteit der Geneeskunde Universiteit van Amsterdam, Amsterdam. Dr Strobos was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Academy of Neurology, the Association for Research in Nervous and Mental Disease, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Mark Teichmann, MD, Long Island City. Died February 7, 1987; age 81. Dr Teichmann was a 1934 graduate of the Universite de Paris VI, Paris. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Francis P. Twinem, MD, Sarasota, Florida. Died January 29, 1987; age 91. Dr Twinem was a 1925 graduate of Harvard Medical School, Boston. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Charles H. Voorhees, MD, Elmira. Died March 22, 1987; age 70. He was a 1943 graduate of State University of New York Health Science Center at Buffalo. Dr Voorhees was a Diplomate of the American Board of Ophthalmology, the American Academy of Ophthalmology and Otolaryngology, the Medical Society of the County of Chemung, and the Medical Society of the State of New York.

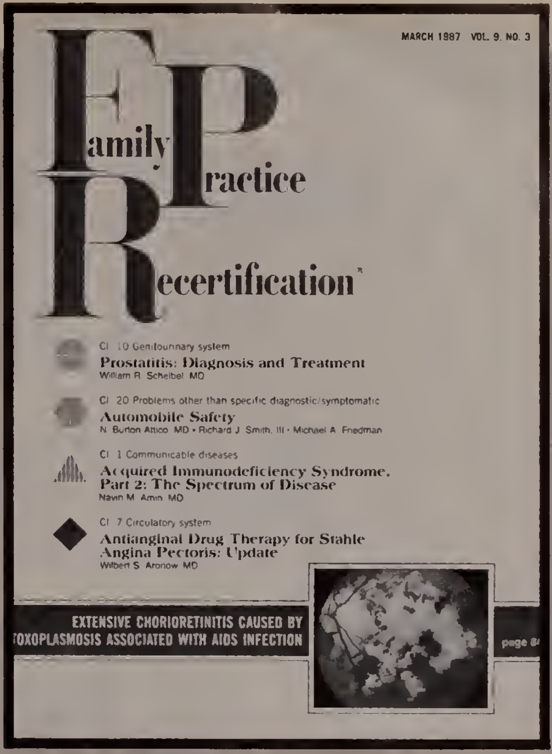
Herbert Lourie Memorial Fund in Neurosurgery

The family, friends, patients, and colleagues of Dr Herbert Lourie, with the cooperation and support of Crouse Irving Memorial Hospital, wish to create a fund to establish a significant and permanent memorial in memory of Dr Lourie.

Contributions to create the fund may be sent to:

Herbert Lourie Memorial Fund in Neurosurgery
Community Foundation of Central New York Inc
500 South Salina Street
Syracuse, NY 13202

Donations are tax deductible.



MARCH 1987 VOL. 9, NO. 3

Family Practice Recertification

CI 10 Genitourinary system
Prostatitis: Diagnosis and Treatment
William R. Scheibel MD

CI 20 Problems other than specific diagnostic/symptomatic
Automobile Safety
N. Burton Attico MD • Richard J. Smith, III • Michael A. Friedman

CI 1 Communicable diseases
Acquired Immunodeficiency Syndrome, Part 2: The Spectrum of Disease
Navin M. Amin MD

CI 7 Circulatory system
Antianginal Drug Therapy for Stable Angina Pectoris: Update
Wilbert S. Aronow MD

EXTENSIVE CHORIORETINITIS CAUSED BY TOXOPLASMOSIS ASSOCIATED WITH AIDS INFECTION

page 84

The complete journal for family practice physicians

- Reaches 79,000 family physicians monthly
- Presents the most commonly seen patient problems in family practice
- Written by physicians for physicians
- The most current clinical updates in:

Cardiology	Ob/Gyn
Diabetes	Psychiatry
Pediatrics	Gastroenterology

- Provides 20 hours of CME Category 1 Credit

PRACTICAL ▪ CLINICAL ▪ EDUCATIONAL ▪ CURRENT

---

guna Niguel, CA 92677. Tel: (714) 495-4499.

MONTANA

July 23–25. **Infertility, Laparoscopy, and Hysteroscopy.** 16 Cat 1 Credits. Huntley Lodge, Big Sky. Contact: The American College of Obstetricians and Gynecologists, 600 Maryland Ave, SW, Suite 300 East, Washington, DC 20024-2588.

VIRGINIA

July 12–15. **Reconstructive and Aesthetic Oculoplastic Surgery.** 12 Cat 1 Credits. Williamsburg. Contact: Plastic Surgery Educational Foundation, 233 North Michigan Ave, Suite 1900, Chicago, IL 60601.

July 17–19. **Practical Internal Medicine: Selected Topics for the Internist.** *July 31–August 2.* **The 9th Annual Pediatric Primary Care Conference: Pediatrics at the Beach.** The Cavalier Resort, Virginia Beach. Contact: Kathy

(*continued on p 24A*)

DOCTOR
DRUG
DISPENSING

"Catching On"

- Greater convenience & savings for patients
- More effective patient treatment
- A new profit center. . . no additional overhead
- Computerized labeling, inventory and ordering

For more information please call or write:

DOCTORS'

DOCTORS' Rx
145 Algonquin Parkway
Whippany, NJ 07981
(201) 428-1117
1 (800) 752-5588

ADVERTISEMENT

Reprinted from *NEW JERSEY MEDICINE*
the Journal of the Medical Society of New Jersey
February 1987, Vol. 84, No. 2, Pp. 101-103

Reprint permission does not imply endorsement of this advertisement by the authors or the Medical Society of New Jersey.

COMPUTERIZATION OF THE MEDICAL OFFICE

PAUL J. HIRSCH, M.D., STUART A. HIRSCH, M.D., LYNN REEDMAN, C.M.A., ANDREW WEISS, M.D., BRIDGEWATER[*]

Increasing numbers of medical offices use computers for many reasons: to be up to date; to manage increasingly complex information; to follow changing regulations; and to take advantage of the decreasing cost of computers. Most computerized medical offices do billing only or tasks directly related to billing. This ignores the potential and promise of the computer. Advancing technology and lower costs have made fully integrated multi-tasking computer systems, which are capable of comprehensive information management, economically feasible. With the current state of the art, simple billing systems are virtually obsolete the moment they are purchased.

INTEGRATED SYSTEMS

An integrated comprehensive computer program is likely to provide the physician with many competitive advantages in today's difficult medical environment. These advantages include the ability to understand more fully and to obtain information about the practice; to provide coding and other information for insurance forms and for the government; and to retrieve and manipulate the information already available in the practice, to direct the growth of that practice, and to market the practice, if necessary.

THE SYSTEM

The system should be capable of supporting multiple terminals, multiple printers, and other devices. More than one terminal is desirable, so several people can enter or retrieve information simultaneously and it is probable that a busy practice will want more than one printer. A letter-quality printer is desirable for reports that will be leaving the office; a high-speed dot matrix printer is useful for billing. A very busy office may want to use a third printer, solely to produce insurance forms, while the patient still is in the office.

The system should support a modem, a device that allows the computer system to communicate with other computer systems. This will permit the office to access public data bases. The modem also will allow the vendor to access the program for modification, or to handle problems. Indeed, most difficulties that are encountered with a program can be corrected by the vendor's programmer over this telephone line.

STARTING UP

The first step in computerization of the medical office is for the physician to begin investigation of various programs. It is the program—the software—that is key to the use of the computer. The hardware—the computer—is of secondary importance. Most often, after identifying the most suitable program, the office will purchase that computer to run the program. It is important to obtain information about computer pro-

---

[*] Dr. Paul Hirsch is Chief of Orthopaedic Surgery, Somerset Medical Center and Clinical Associate Professor, Orthopaedic Surgery, UMDNJ-New Jersey Medical School. Dr. Stuart Hirsch is Attending, Orthopaedic Surgery, Somerset Medical Center. Ms. Reedman is Office Administrator and Computer Manager for Hirsch and Hirsch Orthopaedic Practice. Dr. Weiss is Professor and Chairman, Department of Orthopaedic Surgery, UMDNJ-New Jersey Medical School. Correspondence may be addressed to Dr. Hirsch, Green Knoll Professional Park, 720 U.S. Highway 202-206, Bridgewater, NJ 08807.

ADVERTISEMENT

FOR FURTHER INFORMATION ON THE COMPUTER SYSTEM CHOSEN BY THE AUTHORS OF THIS ARTICLE,
PLEASE CALL OR WRITE: MD SYSTEMS, 214 LINCOLN STREET, ALLSTON, MA 02134, (617) 254-6611.

grams from as many sources as possible; most important is to speak with colleagues who are utilizing programs, as well as members of their staff. Observe the program in action; it will do only what you see it do, unless the program is modified.

The owner of a computer system is dependent upon the computer vendor for support, service, modifications, and enhancements. The company's interest in providing these, and ability to provide these, can best be determined by questioning their customers; this is a crucial step in determining the proper program. A vendor should be willing to make modifications in the program, so that the program can adapt to the office, rather than making the office adapt fully to the program. Enhancements, based upon advances in computer technology and improvements in programming, should be available at least annually; two or three times a year would be better. The vendor also must be willing to alter the programs, in accordance with changes in state, federal, and insurance regulations affecting medical practice.

Once the decision to purchase a computer has been made, and the vendor selected, careful attention must be paid to the contract. This contract always should be reviewed by an attorney.

Cost, of course, is a consideration. Nevertheless, the program that sounds quite expensive to the physician, may not be. The total cost should be considered a five-year investment. For most medical offices, it makes business sense to borrow money for the system, with a five-year repayment; or to arrange a five-year lease-purchase. On this basis, the monthly cost for the system and the service contract, for even "expensive" medical office systems, should be well within the budget of most medical practices. Indeed, it is probable that enhanced billing techniques and improved cash flow will pay for the system quite quickly.

When starting up, a computer manager should be designated. This should be a person within the office who cares about the process of computerization and is committed to it, understands the information generated by the computer and the retrieval of this information, and is responsible for presenting that information to the physician. In most instances, a physician will not have the time to spend on this, nor would it be economically appropriate for a physician to spend the required amount of time on this process.

Support from the vendor also is essential in starting up. The vendor must provide education and training for key employees. The vendor should provide one or more people on-site, immediately following the installation, for a minimum of one week; two weeks or more would be preferable. In part, these representatives will complete the education of the physician's staff. Equally important, their presence will give the staff the confidence that they need to use the system and to make it work. It is important to initiate the process slowly and gradually. There will be many phases to the integrated medical office computer program. It is not necessary to begin all aspects at once. It is best to begin with some segments and gradually introduce the remaining segments of the program, into the office procedures, over a period of months.

We have divided the many functions of an integrated system into three basic applications: business applications, medical applications, and appointment scheduling. It is important to note that these interrelate closely. Indeed, the fact that they do interrelate in actual practice makes it essential that they interrelate within the computer program also.

BUSINESS APPLICATIONS

For most offices, the business applications, particularly patient billing, must and will pay for the system. The first business application, however, and the first step when a patient enters the office, is registration. This is the process by which the patient's name and other important information is entered into the computer. With some systems, all of this data is entered as the patient is registered at the desk. In other systems, the data are entered at the end of the day. Information must include such basic data as birthdate, address, telephone number, and insurance information. The computer should assign an account number. In many practices, it may be important that the system have the capability to assign more than one account number for the patient, for different problems. This may become necessary when different events or illnesses are covered by different insurance companies. A good system will have no difficulty in tracking and maintaining separate accounts for the same patient.

This basic information should allow the computer to produce a "super bill" for the patient. Most offices now are using this super bill format to communicate services performed to the receptionist, and then for recording in billing. The patient should leave the office with a completed super-bill insurance form. The computer system also should have the ability to complete other insurance forms, such as those required under New Jersey no-fault laws.

The complete computer system also will include the CPT-4 and ICD-9 coding systems. All diagnoses and procedures should be coded automatically. At the present time, providing such code numbers on insurance forms, provides a more rapid response time from the insurance companies; and, it seems likely that full diagnostic and procedural coding will be required by all third-party carriers in the near future. Furthermore, coding makes it simple to track diagnoses and procedures for business or clinical purposes.

Even the most basic medical office computer systems have a good billing system. For some of these programs, billing indeed will be the primary or only function performed. An integrated system may or may not perform this in a better fashion for a given practice; but, it should integrate the information and allow ease of retrieval of practice information. Certainly, the billing process should be more efficient with a computer, save time, and produce better cash flow. A good system will initiate the billing process when "told" to do so by the computer manager. Many programs now will produce the bills, already within prestamped and sealed envelopes, and ready for mailing. In association with this, management reports on the accounts receivable, are essential. This should provide breakdown into accounts of 30, 60, 90, and 120 days; and a careful analysis of the monies owed by insurance companies.

Management reports provide an analysis of referral sources and a breakdown of patients by zip code, age, or diagnosis. The reports similarly should provide average charges for each coded item, and the total monthly and year-to-date charges for these items.

The billing system should allow for tracking of in-hospital patients. The name of every patient seen in the hospital should be entered into the system each day. Charges should be entered automatically, but not billed until the patient leaves the hospital. This same process should produce a daily list of all hospital inpatients, whether on service or consultations, so that the physician has a current list of these each day.

All basic office systems should allow the production of mailing lists and labels. This usually will be a product of the registration system. Although many offices may not use a mailing list at the current time, increased competition and the need for marketing of medical practices may require this in the future. It would be useful for such functions as the mailing of a patient newsletter.

Other business applications which might be helpful to some offices include a system for management of accounts payable, inventory control, and a spread sheet program for further practice evaluation and projection analyses. Although the practice may not require all of these items at once, the capability must be there, if obsolescence is to be avoided.

MEDICAL APPLICATIONS

Word processing is essential for any office that produces long notes or reports. Integration of word processing into the computer system will have many advantages. A good word processing system should be easy to use, and should include a spelling checker, with the ability to add an unlimited number of words to the spelling checker. Some programs today will automatically enter any word that is not already included after first "asking" the transcriber to check the spelling. If this is part of the program, then the medical terminology peculiar to any specific practice is likely to be fully entered within a few weeks, without any effort on the part of the transcriber. A high-quality word processing system will reduce the time required to produce medical records, will improve the appearance of the records and of the reports, and will improve the morale of the transcriber.

In addition to the word processing capability, an integrated medical record program will store records and allow retrieval by diagnosis, procedure, or medication. It will assist with quality assurance and with patient management. The integrated medical records program should allow for a data base search, and for providing computerized summaries of the clinical practice.

A medical application provided by some of the better programs will allow direct access to drug information from the *United States Pharmacopoeia*. This information is quite readable; and it may be appropriate to provide printouts of this information for patients.

In addition, a drug interactions index is extremely helpful. When faced with prescribing a new medication for a patient already taking other medications, the physician can enter each of these into the computer and receive immediate information regarding any potential known interactions.

The system should allow the physician access to the public data bases that provide easy retrieval of extraordinary amounts of information.

A comprehensive program should allow the tracking of laboratory data, so that this information is not ignored inadvertently. This is particularly important in our litigious society. When laboratory tests are ordered and recorded as part of the medical record, or independently, they should be placed on a list which is produced regularly, until the information is received.

APPOINTMENT SCHEDULING

Appointment schedulers have been most difficult for computer programs to provide. Many offices have found these to be difficult and tedious, requiring more time for entry than their previous appointment system, and therefore, have preferred to avoid computerized appointment systems. Nevertheless, good appointment systems are available and provide important advantages for the practice.

The appointment system must be adaptable to an individual practice. The program should allow each practice to select the appropriate time intervals for each appointment and the numbers of patients to be seen within each time interval. The system should be capable of searching for an opening, in any one of several possible fashions: first available appointment; first available appointment for a specific physician within the practice; first available appointment on a Monday; first available appointment after 3:00 P.M.; and so on. It should provide this information with a minimum of effort on the part of the receptionist and should do so rapidly. If not, it simply will not work within a busy practice.

The appointment scheduling system should produce at least three daily patient lists for each office. The first, an alphabetized list, will make the pulling of records for the following day much easier. The second, the daily schedule, should be reproduced and kept at several locations in the office, to monitor patient flow. The third, a listing of the subsequent day's patients with telephone numbers, will help with reminder phone calls.

The appointment system also can be utilized to automatically produce reminder postcards for mailing several days prior to a scheduled appointment. The system should produce recall notices for patients who have missed appointments; it should provide a list of those patients for the physician and office manager.

The appointment system also can be utilized to produce patient recalls for routine re-evaluations. Finally, some offices have found it useful to monitor the daily appointment list, as an additional accounting check on the day's billing.

FOR FURTHER INFORMATION ON THE COMPUTER SYSTEM CHOSEN BY THE AUTHORS OF THIS ARTICLE, The System of Choice

MD SYSTEMS

Chosen by the Authors of this Article for the Total Computerization of their Practice.

For Further Information, Please Contact

MediData Systems, Inc.
214 Lincoln Street
Allston, Massachusetts 02134
1-800-451-4522

I am interested in learning how MD Systems can help automate my office.

☐ Please call me to arrange for a **free** office consultation and needs analysis
☐ Please call me to arrange for a demonstration
☐ Please send literature
Number of physicians in practice ☐ 1-3 ☐ 3-6 ☐ 6-10 ☐ 10 or more

Name ______

Name of Group ______

Street Address ______

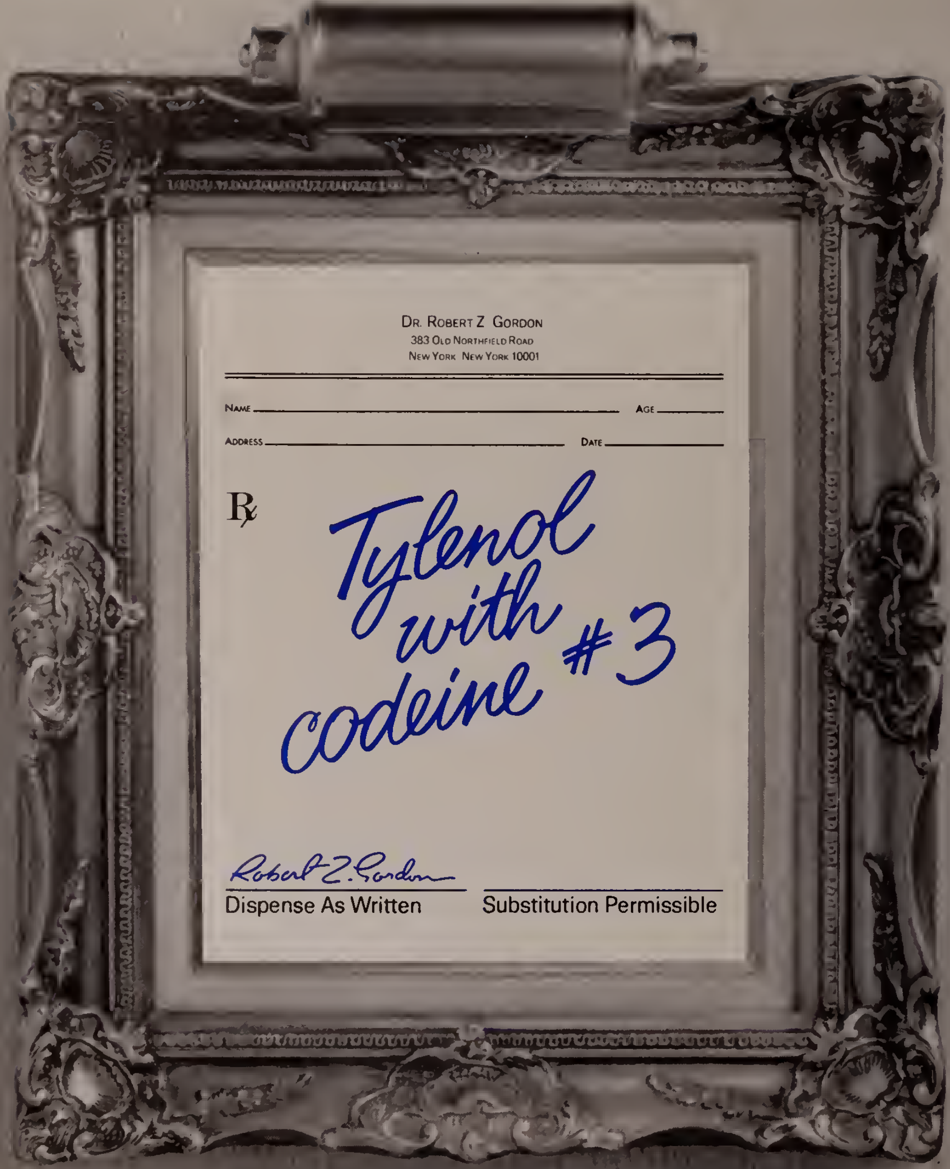
City, State, Zip ______

Telephone ______

I plan to purchase a system.

☐ Within 30 days ☐ 30-60 days ☐ 60-90 days ☐ 90-120 days ☐ gathering information

MediData Systems, Inc.
214 Lincoln Street
Allston, Massachusetts 02134
1-800-451-4522



Dr. Robert Z Gordon
383 Old Northfield Road
New York New York 10001

Name ________ Age ________

Address ________ Date ________

℞

Tylenol
with
codeine #3

Robert Z. Gordon
Dispense As Written

Substitution Permissible

CREATE A SIGNED ORIGINAL

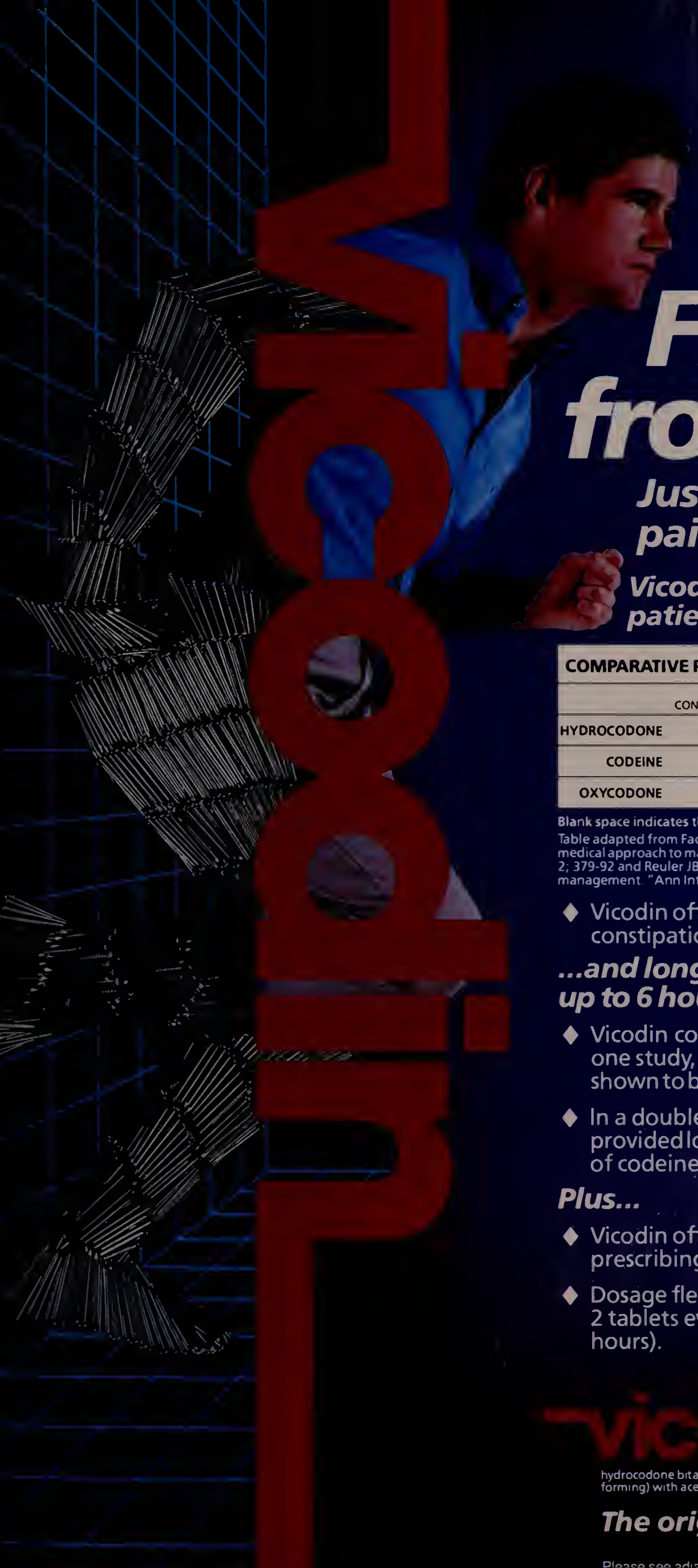
to be sure your patients get...

Tylenol®
ACETAMINOPHEN
with codeine
phosphate
TABLETS Ⓒ III ELIXIR Ⓒ V

Tablets: Contain Codeine Phosphate*: No. 3—30 mg;
No. 4—60 mg—plus Acetaminophen 300 mg.
Elixir: Each 5 mL contains 12 mg Codeine Phosphate* plus
120 mg Acetaminophen (Alcohol 7%).
*Warning: May be habit forming.

© McNEILAB, INC., 1987

TYCT 6445-A



Freedom from pain

Just one part of pain relief therapy.

Vicodin® provides greater patient acceptance

COMPARATIVE PHARMACOLOGY OF THREE ANALGESICS

	CONSTIPATION	RESPIRATORY DEPRESSION	SEDATION	EMESIS	PHYSICAL DEPENDENCE
HYDROCODONE		X			X
CODEINE	X	X	X	X	X
OXYCODONE	XX	XX	XX	XX	XX

Blank space indicates that no such activity has been reported.

Table adapted from Facts and Comparisons (Nov.) 1984 and Catalano RB. The medical approach to management of pain caused by cancer. "Semin Oncol" 1975; 2; 379-92 and Reuler JB, et. al. The chronic pain syndrome: misconceptions and management. "Ann Intern Med" 1980; 93; 588-96.

- Vicodin offers: less nausea, less sedation, less constipation.

...and longer lasting pain relief—up to 6 hours.

- Vicodin contains hydrocodone not codeine. In one study, 10 mg. of hydrocodone alone was shown to be as effective as 60 mg. of codeine.[1]
- In a double-blind study, Vicodin (2 tablets), provided longer lasting pain relief than 60 mg. of codeine.[2]

Plus...

- Vicodin offers the convenience of CIII prescribing.
- Dosage flexibility—1 tablet every 6 hours or 2 tablets every 6 hours (up to 8 tablets in 24 hours).

1. Hopkinson JH III: *Curr Ther Res* 24: 503-516, 1978
2. Beaver, WT *Arch Intern Med*, 141.293-300, 1981

vicodin

hydrocodone bitartrate 5 mg. (Warning: May be habit forming) with acetaminophen 500 mg

The original hydrocodone analgesic.

Please see adjacent page for brief summary of prescribing information.

vicodin®

hydrocodone bitartrate 5mg (Warning May be habit forming)
and acetaminophen 500mg

INDICATIONS AND USAGE: For the relief of moderate to moderately severe pain.
CONTRAINDICATIONS: Hypersensitivity to acetaminophen or hydrocodone.
WARNINGS
Drug Abuse and Dependence: VICODIN is subject to the Federal Controlled Substances Act (Schedule III). Psychic dependence, physical dependence and tolerance may develop upon repeated administration of narcotics; therefore, VICODIN should be prescribed and administered with the same caution appropriate to the use of other oral-narcotic-containing medications.
Respiratory Depression: At high doses or in sensitive patients, hydrocodone may produce dose-related respiratory depression by acting directly on brain stem respiratory centers. Hydrocodone also affects centers that control respiratory rhythm, and may produce irregular and periodic breathing.
Head Injury and Increased Intracranial Pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a preexisting increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.
Acute Abdominal Conditions: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions.
PRECAUTIONS
Special Risk Patients: VICODIN should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture.
Information for Patients: VICODIN, like all narcotics, may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery; patients should be cautioned accordingly.
Cough Reflex: Hydrocodone suppresses the cough reflex, caution should be exercised when VICODIN is used postoperatively and in patients with pulmonary disease.
Drug Interactions: The CNS-depressant effects of VICODIN may be additive with that of other CNS depressants. When combined therapy is contemplated, the dose of one or both agents should be reduced. The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone. The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus
Usage in Pregnancy: Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose. There are no adequate and well-controlled studies in pregnant women. VICODIN should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus
Nonteratogenic Effects: Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent. The intensity of the syndrome does not always correlate with the duration of maternal opioid use or dose.
Labor and Delivery: Administration of VICODIN to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used
Nursing Mothers: It is not known whether this drug is excreted in human milk; therefore, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.
Pediatric Use: Safety and effectiveness in children have not been established
ADVERSE REACTIONS
Central Nervous System: Sedation, drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, dizziness, psychic dependence, mood changes
Gastrointestinal System: Nausea and vomiting may occur; they are more frequent in ambulatory than in recumbent patients Prolonged administration of VICODIN may produce constipation.
Genitourinary System: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported
Respiratory Depression: (See WARNINGS)
DOSAGE AND ADMINISTRATION: Dosage should be adjusted according to the severity of the pain and the response of the patient However, tolerance to hydrocodone can develop with continued use, and the incidence of untoward effects is dose related The usual dose is one tablet every six hours as needed for pain. (If necessary, this dose may be repeated at four-hour intervals) In cases of more severe pain, two tablets every six hours (up to eight tablets in 24 hours) may be required

Revised January, 1987 MR 1987 5786

Knoll Pharmaceuticals
A Unit of BASF K&F Corporation
Whippany, New Jersey 07981

BASF Group

knoll®

"SELLING PRACTICES IS OUR BUSINESS"

Want to maximize the return on your investment? Before you buy or sell, call for an appraisal to assure the best financial and transfer terms. We are a full service practice broker, not simply a listing service. Since 1981, Countrywide has been instrumental in helping hundreds of doctors like yourself buy and sell their practices. We guide you through the entire sales process from initial meeting to closing. Our offices serve New York, New Jersey and New England.

Countrywide Business Brokerage, Inc.
319 East 24th Street, Suite 23-G, New York, NY 10010
(212) 686-7902 (203) 868-3666 (617) 232-1075

REAL ESTATE FOR SALE OR RENT—CONT'D

OFFICE SPACE AVAILABLE: Throgs Neck section of Bronx. Modern medical complex, 1500 square feet, part time/full time. Call (212) 409-9494.

WILL RENT BY THE HOUR, fully equipped and active general ENT office, located in the southwest corner of Nassau County, NY. Hire your help or mine. Address: Mineola P.O. Box #26, Mineola, L.I., NY 11501-0026.

PROFESSIONAL CO-OP APARTMENT FOR SALE. Prime location in the heart of Forest Hills, suitable for any medical specialty. No doctor in 562 apartment building, ground floor, central air conditioning, garage available. One waiting, three examination and one reception rooms. Inquire 70-25 Yellowstone Blvd., Apt. 1-Z, Forest Hills, NY, 1-6 P.M. weekdays.

PRIME CENTRAL GREENWICH VILLAGE, Fifth Ave/10th Street. New street entrance office with waiting room to share. Perfect for all medical, surgical specialties. Call days (212) 473-3049, evenings and weekends (914) 638-2087.

REGO PARK medical suite for rent in new high-rise condo building, near Alexander's Department Store on Queens Blvd., by owner. Future purchase possible. Call (718) 275-2907.

MEDICAL OFFICE RENTAL OPPORTUNITY. Ideal location for patients and close to North Shore University and St. Francis Hospitals. No stairs. Parking. Three examining rooms, private and business offices, reception room, dark room. Port Washington, Nassau County, New York. For more information call (516) 883-8810.

ARE YOU INTERESTED IN SHARING bright sunny newly decorated medical office space in White Plains on a part time basis. Call: (914) 686-9300.

OFFICE SPACE: Our four Medical Centers offer opportunity to open practice without investing in long term lease or capital equipment. EKG, x-ray, cardiac stress, ultrasound, pulmonary function, audio, vision, proctology, colonoscopy. Locations: Manhattan, Brooklyn, Plainview, Hauppauge. Send resume, requirements to: Tara Magee-Prager, NYDC, 146 Manetto Hill Road, Plainview, New York 11803.

MISCELLANEOUS

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

MISCELLANEOUS—CONT'D

HAVE YOU RECEIVED ANY COMMUNICATION FROM THE OFFICE OF PROFESSIONAL MEDICAL CONDUCT? If affirmative, contact Susan Kaplan, Attorney-at-Law, (212) 877-5998. Practice limited to the crucial legal problems directly affecting your license to practice medicine—with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professions), and as an Assistant District Attorney in Nassau County. Susan Kaplan, Esq., 165 West End Avenue, Suite 27P, New York, N.Y. 10023.

TUNA THERAPY SEMINARS: Does your brain need a rest, your body need exercise, are your lungs in need of fresh air? Giant tuna fishing from Montauk Point, New York, aboard Montauk's finest charter boat "Dawn". Charters available for 1 to 4 serious medical anglers. Capt. Greg Beacher, RD-2, Box 16, Benson Drive, Montauk, NY 11954, (516) 668-9056.

COMPUTERIZE YOUR BILLING—Remarkably easy-to-use software automatically prints bills, statements, insurance forms. Menu-driven. Reports ageing balances, referral sources, income by time period, procedure & more. Installs automatically. IBM and compatibles. Solo practice, only $399. Group practice, only $499. Free information. Demonstration disk with complete manual, $19 plus $3 shipping, credited to purchase. Call or write REM Systems, Inc., Dept. NY6, 281 Dug Hill Road, Brownsboro, AL 35741 (205) 534-3457. VISA, MC Accepted.

PORTABLE RADIOLOGY SERVICE: abdominal + pelvic ultrasound, cardiac echos including 2D + m-mode. portable x-rays. NYC and suburbs. Schedule weekly/bi-weekly in your office or visits to your patient's home. New portable hi-tech equipment. Hi-quality like standard office images, far superior to hospital portable images. No lead shielding needed for x-rays. University Radiology Service, (212) 928-5321 or (718) 339-5363.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta, GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuarial certification and employee statements of participation—partnership agreements and professionals incorporation—No insurance required—references upon request—Wachstock and Dienstag Attorneys at Law, 122 Cutter Mill Road, Great Neck, NY 11021 (516) 773-3322.

TELEBILL

A FEE COLLECTION BREAKTHROUGH

- ALL CLAIMS PAPERLESS FROM YOUR OFFICE.
- PATIENT STATEMENTS . . . ATTRACTIVE, EFFECTIVE, PROMPT AND PAPERLESS FROM YOUR OFFICE WITHOUT USING A BILLING SERVICE.
- NO PAPER, NO COMPUTERS, NO LOSS OF CONTROL.
- $3495.00 OR LEASE

A revolutionary new approach to medical billing is now available. With Telebill you can send paperless insurance claims over a standard office phone without a computer. Until now electronic claim submission was expensive, complex, and limited to a small number of insurance carriers. NTC has developed a medical billing data network which makes it possible to send Blue Shield, Medicare, Medicaid, most private, and HMO claims through Telebill, a small, typewriter-like unit that connects to your office telephone. Telebill is easy to install and operate. For a small setup charge, it can be used to transmit patient statements and to generate a full accounting ledger that will replace the conventional card system found in most medical offices.

NTC has been operating its data network for over two years and is the largest processor of private practice insurance claims in New England. Telebill is the first product designed specifically for the health care provider's unique billing needs. At $3495.00, it is priced for the small solo practice, but it will handle the volume of a busy medical group.

To receive a Telebill brochure or to schedule a short Telebill demonstration in your office write National Teledata Corporation, 7 Great Valley Parkway East, Malvern, PA 19355 or Phone 1-800-642-5029.

MISCELLANEOUS—CONT'D

PROFESSIONAL MISCONDUCT ATTORNEYS. William L. Wood Jr., formerly Executive Director of the New York State Office of Professional Discipline and Anthony Z. Scher, formerly Director of Prosecutions, announce the opening of their law office, Wood & Scher. Our recent tenure as chief enforcement officers has given us experience which will allow us to represent physicians in professional misconduct proceedings, malpractice licensure, license restoration, controlled drug proceedings, reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practitioners. Wood & Scher, Attorneys at Law, One Chase Road, Scarsdale, New York 10583. Telephone 914-723-3500.

SIXTH ANNUAL ELLA T. GRASSO MEMORIAL CONFERENCE, November 18, 1987, The Park Plaza Hotel, New Haven, Conn. For registration and further information (after August 1, 1987): Yale University School of Medicine, Office of Graduate and Continuing Education, 333 Cedar Street, P.O. Box 3333, New Haven, CT 06510, (203) 785-4578.

ADVICE, COUNSEL & REPRESENTATION of physicians and other health care professionals in licensure and professional conduct matters, reimbursement issues and practice-related litigation. David E. Ruck, Esq., former Chief, Criminal Division, Office of the New York State Special Prosecutor for Medicaid Fraud Control and Alain M. Bourgeois, Esq., former Acting Justice, Supreme Court of the State of New York, announce the opening of their offices for the practice of law under the name of Bourgeois & Ruck, 101 Park Avenue, New York, New York 10178. Telephone 212-661-8070.

It's 8:00 A.M. Do You Know Where Your Securities Are?

Isn't the safekeeping of your securities as important to you as their performance? You can depend on the safety and service provided by a Bankers Trust Custody Account. We manage the complex administrative details of your securities investments with personal and highly responsive service. Detailed monthly statements and cash management in a totally secured environment are more of the services you'll benefit from.

For more information and our "Guide to Services," write or call: Daisey M. Holmes, Assistant Vice President, Private Clients Group, 529 Fifth Avenue, New York, N.Y. 10017. Tel. 212/696-7837.

© 1987 Bankers Trust Company
Member FDIC

Bankers Trust Company
Private Clients Group
We Make Money For People Who Make Money

There's never been a better time for her...

and PREMARIN®

Proven benefits beyond relief of vasomotor symptoms

No other estrogen proven effective for osteoporosis

Only conjugated estrogens tablets have established efficacy in both osteoporosis[1] and vasomotor symptoms* at 0.625 mg/day. No other estrogen, oral or transdermal, has established clinical evidence or minimum effective dose in both indications.

No estrogen proven safer

PREMARIN is the most extensively tested estrogen, with an unsurpassed record of long-term safety.
And clinical evidence shows a significantly reduced risk of endometrial hyperplasia when cycled with a progestin.[2]

PREMARIN®
(conjugated estrogens tablets)

Most trusted for more reasons

*PREMARIN is indicated for moderate-to-severe vasomotor symptoms.

Please see following page for brief summary
of prescribing information.

For moderate-to-severe vasomotor symptoms and for osteoporosis

PREMARIN®
(conjugated estrogens tablets)

0.3 mg 0.625 mg 0.9 mg 1.25 mg 2.5 mg

The appearance of these tablets is a trademark of Ayerst Laboratories.

For atrophic vaginitis

PREMARIN®
(conjugated estrogens)

Vaginal Cream
0.625 mg/g

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION AND PATIENT INFORMATION, SEE PACKAGE CIRCULARS.)*

PREMARIN® Brand of conjugated estrogens tablets, USP
PREMARIN® Brand of conjugated estrogens Vaginal Cream, in a nonliquefying base

> 1 ESTROGENS HAVE BEEN REPORTED TO INCREASE THE RISK OF ENDOMETRIAL CARCINOMA.
> Three independent, case-controlled studies have reported an increased risk of endometrial cancer in postmenopausal women exposed to exogenous estrogens for more than one year This risk was independent of the other known risk factors for endometrial cancer. These studies are further supported by the finding that incidence rates of endometrial cancer have increased sharply since 1969 in eight different areas of the United States with population-based cancer reporting systems, an increase which may be related to the rapidly expanding use of estrogens during the last decade. The three case-controlled studies reported that the risk of endometrial cancer in estrogen users was about 4.5 to 13.9 times greater than in nonusers The risk appears to depend on both duration of treatment and on estrogen dose In view of these findings, when estrogens are used for the treatment of menopausal symptoms, the lowest dose that will control symptoms should be utilized and medication should be discontinued as soon as possible When prolonged treatment is medically indicated, the patient should be reassessed on at least a semi-annual basis to determine the need for continued therapy Although the evidence must be considered preliminary, one study suggests that cyclic administration of low doses of estrogen may carry less risk than continuous administration; it therefore appears prudent to utilize such a regimen. Close clinical surveillance of all women taking estrogens is important. In all cases of undiagnosed persistent or recurring abnormal vaginal bleeding, adequate diagnostic measures should be undertaken to rule out malignancy There is no evidence at present that "natural" estrogens are more or less hazardous than "synthetic" estrogens at equi-estrogenic doses
> 2. ESTROGENS SHOULD NOT BE USED DURING PREGNANCY.
> The use of female sex hormones, both estrogens and progestogens, during early pregnancy may seriously damage the offspring It has been shown that females exposed in utero to diethylstilbestrol, a nonsteroidal estrogen, have an increased risk of developing, in later life, a form of vaginal or cervical cancer that is ordinarily extremely rare This risk has been estimated as not greater than 4 per 1,000 exposures Furthermore, a high percentage of such exposed women (from 30% to 90%) have been found to have vaginal adenosis, epithelial changes of the vagina and cervix Although these changes are histologically benign, it is not known whether they are precursors of malignancy Although similar data are not available with the use of other estrogens, it cannot be presumed they would not induce similar changes Several reports suggest an association between intrauterine exposure to female sex hormones and congenital anomalies, including congenital heart defects and limb-reduction defects. One case-controlled study estimated a 4.7-fold increased risk of limb-reduction defects in infants exposed in utero to sex hormones (oral contraceptives, hormone withdrawal tests for pregnancy, or attempted treatment for threatened abortion). Some of these exposures were very short and involved only a few days of treatment. The data suggest that the risk of limb-reduction defects in exposed fetuses is somewhat less than 1 per 1,000 In the past, female sex hormones have been used during pregnancy in an attempt to treat threatened or habitual abortion. There is considerable evidence that estrogens are ineffective for these indications, and there is no evidence from well-controlled studies that progestogens are effective for these uses If PREMARIN is used during pregnancy, or if the patient becomes pregnant while taking this drug, she should be apprised of the potential risks to the fetus, and the advisability of pregnancy continuation

DESCRIPTION: PREMARIN (conjugated estrogens, USP) contains a mixture of estrogens, obtained exclusively from natural sources, blended to represent the average composition of material derived from pregnant mares' urine It contains estrone, equilin, and 17α-dihydroequilin, together with smaller amounts of 17α-estradiol, equilenin, and 17α-dihydroequilenin as salts of their sulfate esters. Tablets are available in 0.3 mg, 0.625 mg, 0.9 mg, 1.25 mg, and 2.5 mg strengths of conjugated estrogens Cream is available as 0.625 mg conjugated estrogens per gram.

INDICATIONS AND USAGE: PREMARIN (conjugated estrogens tablets, USP): Moderate-to-severe *vasomotor* symptoms associated with the menopause (There is no evidence that estrogens are effective for nervous symptoms or depression without associated vasomotor symptoms and they should not be used to treat such conditions.) Osteoporosis (abnormally low bone mass). Atrophic vaginitis. Kraurosis vulvae Female castration

PREMARIN (conjugated estrogens) Vaginal Cream is indicated in the treatment of atrophic vaginitis and kraurosis vulvae

PREMARIN HAS NOT BEEN SHOWN TO BE EFFECTIVE FOR ANY PURPOSE DURING PREGNANCY AND ITS USE MAY CAUSE SEVERE HARM TO THE FETUS (SEE BOXED WARNING)

Concomitant Progestin Use: The lowest effective dose appropriate for the specific indication should be utilized Studies of the addition of a progestin for 7 or more days of a cycle of estrogen administration have reported a lowered incidence of endometrial hyperplasia Morphological and biochemical studies of the endometrium suggest that 10 to 13 days of progestin are needed to provide maximal maturation of the endometrium and to eliminate any hyperplastic changes Whether this will provide protection from endometrial carcinoma has not been clearly established There are possible additional risks which may be associated with the inclusion of progestin in estrogen replacement regimens (See PRECAUTIONS.) The choice of progestin and dosage may be important, product labeling should be reviewed to minimize possible adverse effects.

CONTRAINDICATIONS: Estrogens should not be used in women (or men) with any of the following conditions: 1 Known or suspected cancer of the breast except in appropriately selected patients being treated for metastatic disease 2 Known or suspected estrogen-dependent neoplasia 3. Known or suspected pregnancy (see Boxed Warning) 4 Undiagnosed abnormal genital bleeding 5 Active thrombophlebitis or thromboembolic disorders 6 A past history of thrombophlebitis, thrombosis, or thromboembolic disorders associated with previous estrogen use (except when used in treatment of breast or prostatic malignancy)

WARNINGS: Estrogens have been reported to increase the risk of endometrial carcinoma (see Boxed Warning). However, a recent large, case-controlled study indicated no increase in risk of breast cancer in postmenopausal women. A recent study has reported a 2- to 3-fold increase in the risk of surgically confirmed gallbladder disease in women receiving postmenopausal estrogens

Adverse effects of oral contraceptives may be expected at the larger doses of estrogen used to treat prostatic or breast cancer or postpartum breast engorgement, it has been shown that there is an increased risk of thrombosis in men receiving estrogens for prostatic cancer and women for postpartum breast engorgement Users of oral contraceptives have an increased risk of diseases, such as thrombophlebitis, pulmonary embolism, stroke, and myocardial infarction. Cases of retinal thrombosis, mesenteric thrombosis, and optic neuritis have been reported in oral contraceptive users An increased risk of postsurgery thromboembolic complications has also been reported in users of oral contraceptives If feasible, estrogen should be discontinued at least 4 weeks before surgery of the type associated with an increased risk of thromboembolism, or during periods of prolonged immobilization Estrogens should not be used in persons with active thrombophlebitis, thromboembolic disorders, or in persons with a history of such disorders in association with estrogen use They should be used with caution in patients with cerebral vascular or coronary artery disease Large doses (5 mg conjugated estrogens per day), comparable to those used to treat cancer of the prostate and breast, have been shown to increase the risk of nonfatal myocardial infarction, pulmonary embolism, and thrombophlebitis When doses of this size are used, any of the thromboembolic and thrombotic adverse effects should be considered a clear risk

Benign hepatic adenomas should be considered in estrogen users having abdominal pain and tenderness, abdominal mass, or hypovolemic shock Hepatocellular carcinoma has been reported in women taking estrogen-containing oral contraceptives. Increased blood pressure may occur with use of estrogens in the menopause and blood pressure should be monitored with estrogen use A worsening of glucose tolerance has been observed in patients on estrogen-containing oral contraceptives For this reason, diabetic patients should be carefully observed Estrogens may lead to severe hypercalcemia in patients with breast cancer and bone metastases

PRECAUTIONS: Physical examination and a complete medical and family history should be taken prior to the initiation of any estrogen therapy with special reference to blood pressure, breasts, abdomen, and pelvic organs, and should include a Papanicolaou smear. As a general rule, estrogen should not be prescribed for longer than one year without another physical examination being performed Conditions influenced by fluid retention, such as asthma, epilepsy, migraine, and cardiac or renal dysfunction, require careful observation. Certain patients may develop manifestations of excessive estrogenic stimulation, such as abnormal or excessive uterine bleeding, mastodynia, etc. Prolonged administration of unopposed estrogen therapy has been reported to increase the risk of endometrial hyperplasia in some patients Oral contraceptives appear to be associated with an increased incidence of mental depression Patients with a history of depression should be carefully observed. Pre-existing uterine leiomyomata may increase in size during estrogen use. The pathologist should be advised of estrogen therapy when relevant specimens are submitted If jaundice develops in any patient receiving estrogen, the medication should be discontinued while the cause is investigated Estrogens should be used with care in patients with impaired liver function, renal insufficiency, metabolic bone diseases associated with hypercalcemia, or in young patients in whom bone growth is not yet complete If concomitant progestin therapy is used, potential risks may include adverse effects on carbohydrate and lipid metabolism

The following changes may be expected with larger doses of estrogen:

a Increased sulfobromophthalein retention.

b. Increased prothrombin and factors VII, VIII, IX, and X, decreased antithrombin 3, increased norepinephrine-induced platelet aggregability

c Increased thyroid binding globulin (TBG) leading to increased circulating total thyroid hormone, as measured by PBI, T_4 by column, or T_4 by radioimmunoassay Free T_3 resin uptake is decreased, reflecting the elevated TBG; free T_4 concentration is unaltered

d Impaired glucose tolerance.

e. Decreased pregnanediol excretion

f Reduced response to metyrapone test

g. Reduced serum folate concentration.

h. Increased serum triglyceride and phospholipid concentration

As a general principle, the administration of any drug to nursing mothers should be done only when clearly necessary since many drugs are excreted in human milk

Long-term, continuous administration of natural and synthetic estrogens in certain animal species increases the frequency of carcinomas of the breast, cervix, vagina, and liver However, in a recent, large case-controlled study of postmenopausal women there was no increase in risk of breast cancer with use of conjugated estrogens.

ADVERSE REACTIONS: The following have been reported with estrogenic therapy, including oral contraceptives: breakthrough bleeding, spotting, change in menstrual flow, dysmenorrhea, premenstrual-like syndrome, amenorrhea during and after treatment, increase in size of uterine fibromyomata, vaginal candidiasis, change in cervical erosion and in degree of cervical secretion, cystitis-like syndrome, tenderness, enlargement, secretion (of breasts); nausea, vomiting, abdominal cramps, bloating; cholestatic jaundice; chloasma or melasma which may persist when drug is discontinued, erythema multiforme, erythema nodosum; hemorrhagic eruption, loss of scalp hair; hirsutism; steepening of corneal curvature; intolerance to contact lenses; headache, migraine, dizziness, mental depression, chorea, increase or decrease in weight, reduced carbohydrate tolerance, aggravation of porphyria, edema, changes in libido.

ACUTE OVERDOSAGE: May cause nausea, and withdrawal bleeding may occur in females.

DOSAGE AND ADMINISTRATION:
PREMARIN® Brand of conjugated estrogens tablets, USP

1. *Given cyclically for short-term use only* For treatment of moderate-to-severe *vasomotor* symptoms, atrophic vaginitis, or kraurosis vulvae associated with the menopause (0.3 mg to 1.25 mg or more daily). The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible Administration should be cyclic (eg, three weeks on and one week off). Attempts to discontinue or taper medication should be made at three- to six-month intervals

2. *Given cyclically*: Osteoporosis. Female castration. Osteoporosis —0.625 mg daily Administration should be cyclic (eg, three weeks on and one week off). Female castration—1.25 mg daily, cyclically Adjust upward or downward according to response of the patient. For maintenance, adjust dosage to lowest level that will provide effective control

Patients with an intact uterus should be monitored for signs of endometrial cancer and appropriate measures taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding

PREMARIN® Brand of conjugated estrogens Vaginal Cream

Given cyclically for short-term use only For treatment of atrophic vaginitis or kraurosis vulvae.

The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible.

Administration should be cyclic (eg, three weeks on and one week off).

Attempts to discontinue or taper medication should be made at three- to six-month intervals

Usual dosage range: 2 g to 4 g daily, intravaginally, depending on the severity of the condition

Treated patients with an intact uterus should be monitored closely for signs of endometrial cancer and appropriate diagnostic measures should be taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding

References:

1. Lindsay R, Hart DM, Clark OM: The minimum effective dose of estrogen for prevention of postmenopausal bone loss *Obstet Gynecol* 1984,63:759-763. **2.** Studd JWW, Thom MH, Paterson MEL, et al: The prevention and treatment of endometrial pathology in postmenopausal women receiving exogenous estrogens, in Pasetto N, Paoletti R, Ambrus JL (eds): *The Menopause and Postmenopause*. Lancaster, England, MTP Press Ltd, 1980, chap 13

© 1987 Ayerst Laboratories
All rights reserved.

6437/587

Ayerst®
AYERST LABORATORIES
New York, NY 10017

AMASOURCE

Current information on AMA publications for physicians and patients

Health Policy Agenda for the American People Over 425 representatives from 172 different health, health-related, business, government, and consumer groups met over the past five years to develop the *Health Policy Agenda for the American People*. Their mission: to examine immediate contemporary concerns, as well as issues expected to confront the health sector into the next decade and the next century. The *Health Policy Agenda* appears in three publications: the introductory *Summary Report;* the *Final Report,* in which health care issues, recommendations, and implementation plans are discussed in detail; and the *Reference Report,* containing background material developed during the project.

(OP-024) Summary Report ... 12.00
(OP-025) Final Report ... 30.00
(OP-026) Reference Report ... 50.00
(OP-027) Combination of OP-024 and OP-025 ... 35.00
(OP-023) 3-volume set .. 80.00

Drug Evaluations Sixth Edition (DE6) Contains comprehensive information about drugs, arranged by therapeutic use and class of drug. It compares and evaluates drugs as well as recommending drugs and regimens of choice. This new edition features more than 70 new drugs and 100 new investigational agents, a new chapter on drug therapy and expanded coverage on the immune system.
1800 pages (OP-255/6) . 75.00

Guides to the Evaluation of Permanent Impairment The world's best reference for evaluating the impaired. Each chapter of "Guides" explores a particular body system, and gives examples to assist you in your determination. Plus–this updated 2nd edition includes new tests, more clinical examples, the latest measurement techniques, more tables, greater depth, and a new chapter on sleep and arousal disorders.
454 pages (OP-254) . 27.50

AMA Health Insurance Claim Form This is the standardized form required by the federal government and accepted by most insurers. Each order includes a free, detailed 12-page instruction booklet for proper completion of forms.

(OP-501) Single form, 1 page, 100 per pad, has Kodak bar code, ten pads/carton 34.10
(OP-502) Snap-out form, 2-part NCR, has Kodak bar code, 1000/carton ... 49.50
(OP-503) Continuous form, 2-part NCR with pinfeeds for computer printers, has Kodak bar code, 1000/carton .. . 51.75
(OP-505) Continuous form, 2-part NCR with pinfeeds for computer printers, has no Kodak bar code, 1000/carton 51.75

Physicians' Resource Guide to Health Delivery Systems There's nothing else like it. Contents of this unique and comprehensive publication include: history and overview of current systems; national resource listing of organizations and agencies involved with HMOs, PPOs, IPAs; glossary of terms; over 140 abstracts on 18 key topics.
122 pages (OP-157) ... 15.00

Current Procedural Terminology 1987 Simplify claims processing with the five-digit code system provided in *Current Procedural Terminology (CPT).* Identifying more than 7,000 procedures and services, *CPT* is the most widely used procedure coding system in the U.S. *CPT* is employed by the Health Care Financing Administration as the basis for the HCPCS coding system, and is required for Medicare claims.
500 pages (OP-341/7) 30.00

CPT 1987 floppy disk is the medical procedures coding system of choice for physicians, third parties, and the federal government. CPT-1987 on floppy disk eliminates the time-consuming chore of entering all the procedures you perform into your data base or software. All procedure descriptions are presented in layman's terms, ideal for use with practice management and patient billing software.

Includes one copy of the CPT 1987 book and one copy of the data on the disk.
(OP-224) 175.00

For Credit Card Orders Call: 1-800/621-8335 Illinois Residents Call Collect: 312/645-4987

OP Number	Quantity	Title/Description	Price Per Unit	Line Total
			$	$
			$	$
			$	$
			$	$

Please send your order and full payment to:
American Medical Association
P.O. Box 10946
Chicago, IL 60610-0946

$10 minimum order. Costs include shipping and handling. Price is for 1-4 copies. For information and additional quantity costs, contact: Customer Service Department, American Medical Association, 535 N. Dearborn, Chicago, Illinois 60610 (312) 280-7168.

Subtotal $________
AMA members (subtract 20%) $________
ADD Sales Tax– Illinois & New York State Purchasers $________
Foreign Orders Add $10 Per Unit $________
Total Amount Due $________

Coupon expires December 31, 1987
Prices subject to change.
Please allow 4-6 weeks for delivery.
Orders are filled on the basis of product availability and date of receipt.

Customer Name ________
(print clearly or type)
Company Name ________
Office/Department ________
Street Address ________
City/State/Zip (U.S.) ________
Foreign Country Name ________

☐ AMA Member ☐ Non-member
☐ Enclosed is my check made payable to the American Medical Association
☐ VISA ☐ MasterCard

GIVE ADDED CHANCE TO YOUR
RESPIRATOR DEPENDENT PATIEN
TO IMPROVE DIFFUSION AND
GET OFF THE RESPIRATOR

with

**Effective Lobe to Lobe
Aspirations with the
Rockey-Thompson Bronch
Suction Catheters will hel**

AVAILABLE IN SETS OF 4:
F14, F16, F18, F20

For more information call or write:

from:
R.E.P. DIVISION PECK PACKAGING C
528 Carter Street, New Canaan,
CONN. 06840 Tel. (203) 972-0119

(*continued from p 11A*)

Martin, Office of Continuing Medical Education, Medical College of Virginia, Box 48, MCV Station, Richmond VA 23298-0001. Tel: (804) 786-0494.

AROUND THE WORLD

CANADA

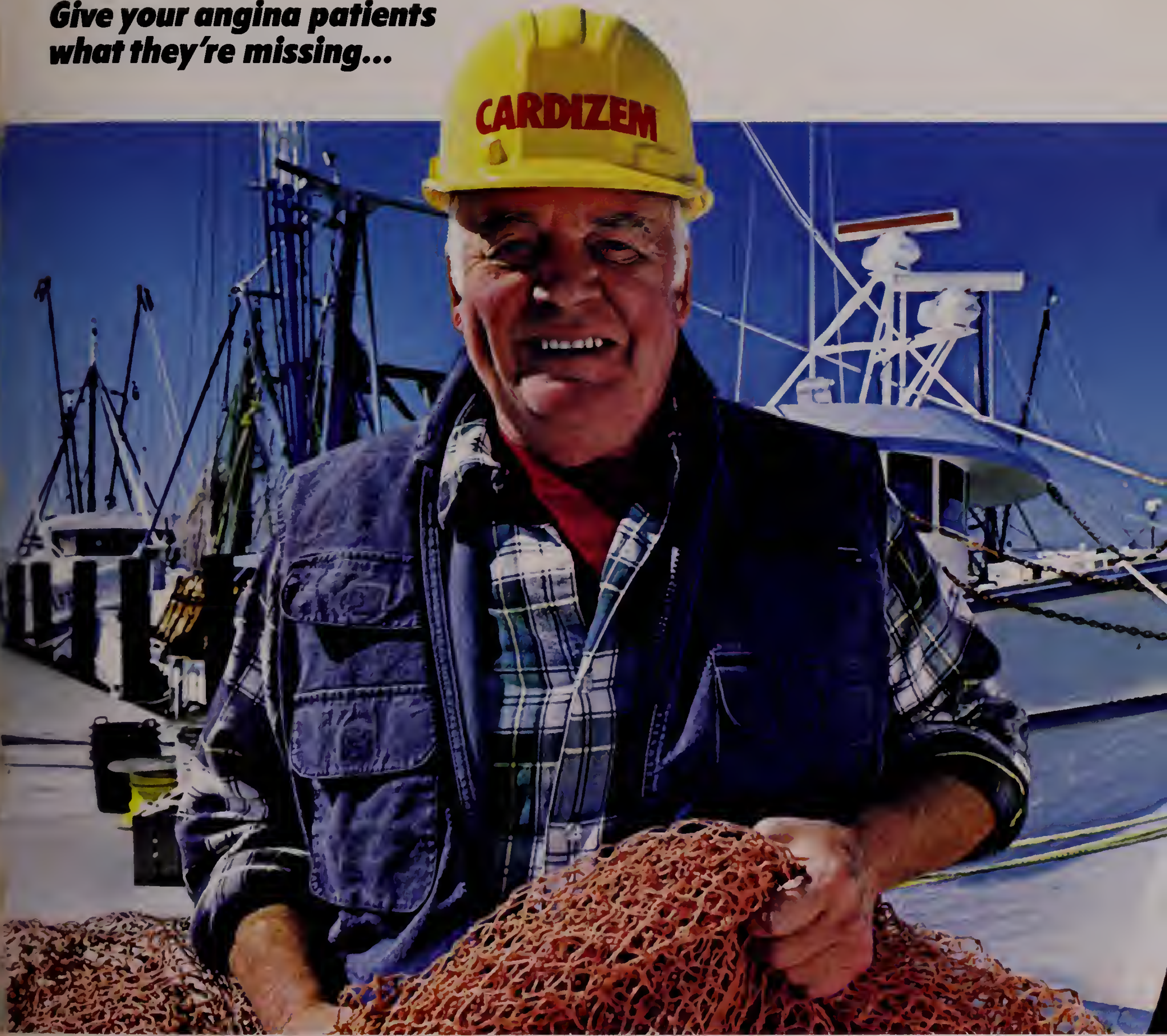
July 16–18. **Gynecologic Surgery and Urology.** 16 Cat 1 Credits. Westin Hotel, Toronto, Ontario. Contact: The American College of Obstetricians and Gynecologists, 600 Maryland Ave, SW, Suite 300 East, Washington, DC 20024-2588.

July 24–26. **The 10th National Conference on Pediatric/Adult Allergy & Clinical Immunology.** The Sheraton Centre, Toronto, Ontario. Contact: Rayna Saville, Coordinator, Continuing Medical Education, Children's Hospital, 219 Bryant St, Buffalo, NY 14222-2099. Tel: (716) 878-7630. ☐

Index to Advertisers

Ayerst Laboratories 20A, 21A, 22
Ciba Pharmaceuticals 5A, 6
St. Clare's Hospital 7
Classified Advertising 6A, 9A, 11A, 18A, 19
Family Practice Recertification Magazine 11
Knoll Pharmaceuticals 1A, 2A, 3A, 17A, 18
Eli Lilly and Company 10
Marion Laboratories 25A, 26
McNeil Laboratories 16
MediData Systems, Inc. 12A, 13A, 14A, 15
Medical Liability Mutual Insurance Company 8
Mt. Sinai Center 9
National Teledata Corporation 19
Peck Packaging Corp. 24
Roche Laboratories .... 3rd & 4th Cov
Upjohn Company .... 2nd Cov

Give your angina patients what they're missing...



CARDIZEM®: FEW SIDE EFFECTS

diltiazem HCl/Marion

90 mg GREATER DOSAGE FLEXIBILITY NEW 120 mg

- ***Antianginal action includes dilatation of coronary arteries, a decrease in vascular resistance/afterload, and a reduction in heart rate***
- ***Proven efficacy when used alone in angina[1]***
- ***Compatible with other antianginals[2,3]****
- ***A safe choice for angina patients with coexisting hypertension, asthma, COPD, or PVD[4,5]***

****See Warnings and Precautions.***

Please see brief summary of prescribing information on the next page.

CARDIZEM® diltiazem HCl/Marion FEW SIDE EFFECTS IN ANTIANGINAL THERAPY

60 mg tid or qid

Brief Summary
Professional Use Information

CARDIZEM®
(diltiazem HCl) 30 mg and 60 mg Tablets

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1,243 patients for 0 48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt).
   Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, significant elevations in enzymes such as alkaline phosphatase, CPK, LDH, SGOT, SGPT, and other symptoms consistent with acute hepatic injury have been noted These reactions have been reversible upon discontinuation of drug therapy. The relationship to CARDIZEM is uncertain in most cases, but probable in some (See PRECAUTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women, therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. Diltiazem is excreted in human milk. One report suggests that concentrations in breast milk may approximate serum levels. If use of CARDIZEM is deemed essential, an alternative method of infant feeding should be instituted

Pediatric Use. Safety and effectiveness in children have not been established

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established The most common occurrences as well as their frequency of presentation are: edema (2.4%), headache (2 1%), nausea (1.9%), dizziness (1.5%), rash (1 3%), asthenia (1.2%). In addition, the following events were reported infrequently (less than 1%):

Cardiovascular:	Angina, arrhythmia, AV block (first degree), AV block (second or third degree — see conduction warning), bradycardia, congestive heart failure, flushing, hypotension, palpitations, syncope.
Nervous System:	Amnesia, gait abnormality, hallucinations, insomnia, nervousness, paresthesia, personality change, somnolence, tinnitus, tremor
Gastrointestinal.	Anorexia, constipation, diarrhea, dysgeusia, dyspepsia, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH (see hepatic warnings), vomiting, weight increase.
Dermatologic:	Petechiae, pruritus, photosensitivity, urticaria.
Other.	Amblyopia, dyspnea, epistaxis, eye irritation, hyperglycemia, nasal congestion, nocturia, osteoarticular pain, polyuria, sexual difficulties

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM. alopecia, gingival hyperplasia, erythema multiforme, and leukopenia. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established. Issued 7/86

See complete Professional Use Information before prescribing

References: 1. Pepine CJ, Feldman RL, Hill JA, et al: Clinical outcome after treatment of rest angina with calcium blockers: Comparative experience during the initial year of therapy with diltiazem, nifedipine, and verapamil Am Heart J 1983, 106(6):1341-1347 **2.** Shapiro W: Calcium channel blockers. Actions on the heart and uses in ischemic heart disease. Consultant 1984,24(Dec):150-159 **3.** Johnston DL, Lesoway R, Humen DP, et al: Clinical and hemodynamic evaluation of propranolol in combination with verapamil, nifedipine and diltiazem in exertional angina pectoris: A placebo-controlled, double-blind, randomized, crossover study Am J Cardiol 1985,55 680-687. **4.** Cohn PF, Braunwald E: Chronic ischemic heart disease, in Braunwald E (ed): Heart Disease: A Textbook of Cardiovascular Medicine, ed 2. Philadelphia, WB Saunders Co, 1984, chap 39 **5.** Schroeder JS: Calcium and beta blockers in ischemic heart disease: When to use which. Mod Med 1982;50(Sept):94-116

Another patient benefit product from

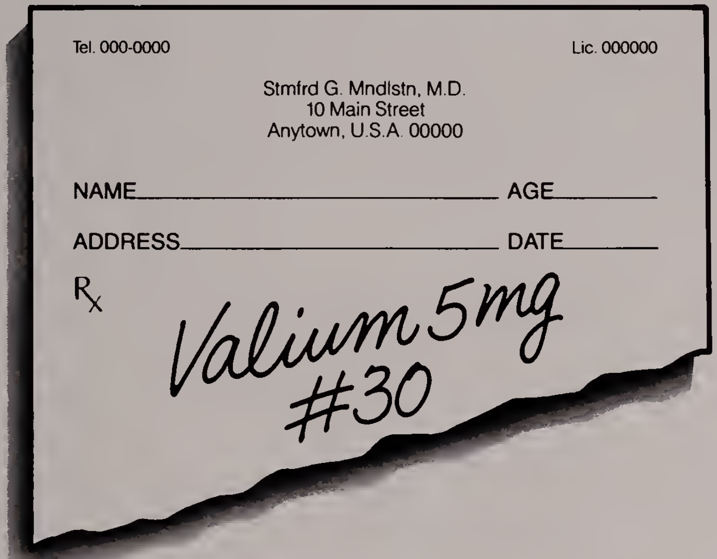
PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC.
KANSAS CITY, MO 64137

0246M6

Do Not Substitute

VALIUM® brand of diazepam/Roche Ⓘⓥ

The One You Know Best



Tel. 000-0000 Lic. 000000

Stmfrd G. Mndlstn, M.D.
10 Main Street
Anytown, U.S.A. 00000

NAME ____________ AGE ______

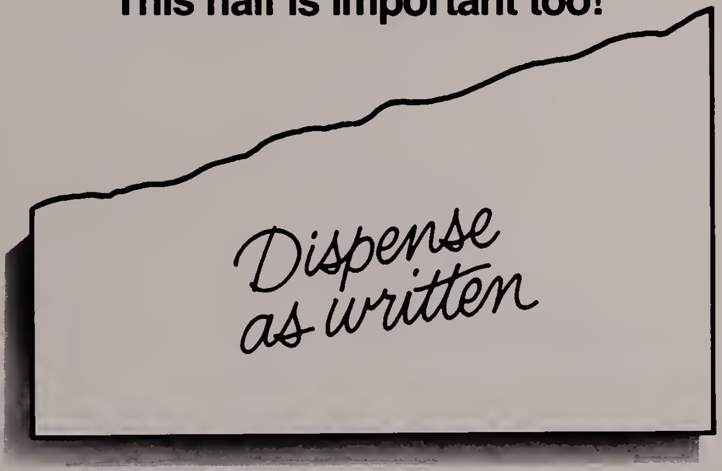
ADDRESS ____________ DATE ______

℞

Valium 5mg
#30

Are you writing only half a prescription for it?

This half is important too!



Dispense
as written

Be sure to write a complete prescription.

Specify "Dispense as written."

ROCHE®

Roche Products Inc.
Manati, Puerto Rico 00701

Copyright © 1987 by Roche Products Inc.
All rights reserved.

State flag of New York

Flag It.

To complete your prescription,
be sure to specify
"Dispense As Written."
This "flags" both pharmacist and patient
that you want the *brand* to be dispensed.
And it protects your decision.

VALIUM®
brand of
diazepam/Roche Ⓘⓥ

scored tablets

2 mg 5 mg 10 mg

The one you know best.

The cut out "V" design is a registered trademark
of Roche Products Inc.

ROCHE ®

NEW YORK STATE JOURNAL OF MEDICINE

JULY 1987 • Volume 87, Number 7

STACKS

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

Contents

COMMENTARIES

Adrenal medullary grafts transplanted into the brain as a treatment for Parkinson disease 377
ABRAHAM LIEBERMAN, MD; MAX KOSLOW, MD; PAUL MAESTRONE, DVM

Ethical dilemmas imposed by the Medicare hospice regulations 379
LAWRENCE M. SHERMAN, MD, WILLIAM E. FINN, BS

Cutaneous polyarteritis nodosa 381
GENE V. BALL, MD

RESEARCH PAPERS

The effectiveness of film and videotape in teaching cardiopulmonary resuscitation to the lay public 382
JOSEPH SCHLUGER, MD; JOSEPH G. HAYES, MD; GERARD M. TURINO, MD; SYLVIA FISHMAN, EdD; ARTHUR C. FOX, MD

Medical students' exposure to formaldehyde in gross anatomy laboratories 385
JAY M. FLEISHER

REVIEW ARTICLE

Current management of tuberculosis 389
ALJE VENNEMA, MD

CLINICAL CONFERENCE

Adult measles 391

COMMITTEE REPORT

Ethical considerations of reproductive technologies 398
FRED ROSNER, MD; ERIC J. CASSELL, MD; MICHAEL L. FRIEDLAND, MD; ALLISON B. LANDOLT, MD; LAURENCE LOEB, MD; PATRICIA J. NUMANN, MD; FERNANDO V. ORA, MD; HERMAN M. RISEMBERG, MD; PETER P. SORDILLO, MD

CASE REPORTS

Polyarteritis nodosa presenting with migratory soft tissue swelling 402
GARY S. MEREDITH, MD; HAL J. MITNICK, MD; HARRIS E. BURSTIN, MD; SOL S. ZIMMERMAN, MD

Tuberculosis mimicking acute pyogenic bacterial pneumonia 404
KRISHNAN PADMANABHAN, MD; SANTI R. DHAR, MD; B. BHATT, MD

Volvulus of the descending colon 406
JOHN S. O'SHEA, MD; ROBERT ZUBOWSKI, MD; JAMES C. DiLORENZO, MD

Endotracheal hamartoma 408
JENNIFER E. ALEXANDER, MD, RICHARD BRODMAN, MD

LETTERS TO THE EDITOR

Medicaid recordkeeping requirements and professional misconduct 410
DONALD R. MOY, JD

Homelessness in Paris 411
JEAN-MARC NORES, MD, JEAN DALAYEUN, MD

Continuing use of x-ray pelvimetry for pregnant women 411
ALFRED GELLHORN, MD; ALAN LEVENSOHN, PhD; GERALD KAUFMAN, PhD; LAURA IWAN, MS

Massive pulmonary embolism with right atrial thrombus 413
NASIRUDDIN M. JAMAL, MD, GEORGE M. GHATTAS, MD

Obturator hernias 414
DENNIS WADLER, MD, RICHARD H. ROSE, MD

BOOK REVIEWS 415
NEWS BRIEFS 419
OBITUARIES 421
GUIDELINES FOR AUTHORS 424
MEDICAL MEETINGS AND LECTURES 8A

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA
JUL 14 1987 M. D. S.

Motrin® 800 TABLETS mg

ibuprofen

Extra strength
Convenience
Economy

Upjohn
100
A Century
of Caring
1886·1986

©1986 The Upjohn Company

J-6138 January 1986

Give your angina patients what they're missing...

CARDIZEM®: FEW SIDE EFFECTS

diltiazem HCl/Marion

- ***Antianginal action includes dilatation of coronary arteries, a decrease in vascular resistance/afterload, and a reduction in heart rate***
- ***Proven efficacy when used alone in angina[1]***
- ***Compatible with other antianginals[2,3]****
- ***A safe choice for angina patients with coexisting hypertension, asthma, COPD, or PVD[4,5]***

90 mg GREATER DOSAGE FLEXIBILITY mg **NEW 120**

****See Warnings and Precautions.***

Please see brief summary of prescribing information on the next page.

CARDIZEM® diltiazem HCl/Marion FEW SIDE EFFECTS IN ANTIANGINAL THERAPY

60 mg tid or qid

Brief Summary
Professional Use Information

CARDIZEM®
(diltiazem HCl) 30 mg and 60 mg Tablets

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1,243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt).
   Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, significant elevations in enzymes such as alkaline phosphatase, CPK, LDH, SGOT, SGPT, and other symptoms consistent with acute hepatic injury have been noted. These reactions have been reversible upon discontinuation of drug therapy. The relationship to CARDIZEM is uncertain in most cases, but probable in some. (See PRECAUTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. Diltiazem is excreted in human milk. One report suggests that concentrations in breast milk may approximate serum levels. If use of CARDIZEM is deemed essential, an alternative method of infant feeding should be instituted.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences as well as their frequency of presentation are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%). In addition, the following events were reported infrequently (less than 1%):

Cardiovascular:	Angina, arrhythmia, AV block (first degree), AV block (second or third degree — see conduction warning), bradycardia, congestive heart failure, flushing, hypotension, palpitations, syncope.
Nervous System:	Amnesia, gait abnormality, hallucinations, insomnia, nervousness, paresthesia, personality change, somnolence, tinnitus, tremor.
Gastrointestinal:	Anorexia, constipation, diarrhea, dysgeusia, dyspepsia, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH (see hepatic warnings), vomiting, weight increase.
Dermatologic:	Petechiae, pruritus, photosensitivity, urticaria.
Other:	Amblyopia, dyspnea, epistaxis, eye irritation, hyperglycemia, nasal congestion, nocturia, osteoarticular pain, polyuria, sexual difficulties.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: alopecia, gingival hyperplasia, erythema multiforme, and leukopenia. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

Issued 7/86

See complete Professional Use Information before prescribing

References: 1. Pepine CJ, Feldman RL, Hill JA, et al. Clinical outcome after treatment of rest angina with calcium blockers. Comparative experience during the initial year of therapy with diltiazem, nifedipine, and verapamil. Am Heart J 1983; 106(6): 1341-1347. **2.** Shapiro W: Calcium channel blockers: Actions on the heart and uses in ischemic heart disease. Consultant 1984;24(Dec): 150-159. **3.** Johnston DL, Lesoway R, Humen DP, et al: Clinical and hemodynamic evaluation of propranolol in combination with verapamil, nifedipine and diltiazem in exertional angina pectoris: A placebo-controlled, double-blind, randomized, crossover study. Am J Cardiol 1985;55:680-687. **4.** Cohn PF, Braunwald E: Chronic ischemic heart disease, in Braunwald E (ed): Heart Disease: A Textbook of Cardiovascular Medicine, ed 2. Philadelphia, WB Saunders Co, 1984, chap 39. **5.** Schroeder JS: Calcium and beta blockers in ischemic heart disease: When to use which. Mod Med 1982;50(Sept):94-116

Another patient benefit product from

M PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC.
KANSAS CITY, MO 64137

0246M6

NEW YORK STATE JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
JOHN T. PRIOR, MD
ELIZA H. CALDWELL, MD
GITA S. SINGH*
JOSEPH F. MURATORE, MD
STANFORD WESSLER, MD

**Medical student*

Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Consulting Editor and Book Review Editor	RICHARD B. BIRRER, MD
Consulting Editor	NAOMI R. BLUESTONE, MD, MPH
Consulting Editor	CARL POCHEDLY, MD
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Managing Editor	CAROL L. MOORE
Advertising Director	ROBERT MILLER
Consulting Medical Writer	VICKI GLASER
Editorial Assistant	MILDRED J. ARFMANN
Secretary	ELIZABETH J. SOMERS
Librarian	ELLA ABNEY
Assistant Librarian	ELEANOR BURNS

ASSOCIATE EDITORIAL BOARD 1987

MICHAEL E. BERLOW, MD
RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MYLES S. GOMBERT, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
JAMES M. MORRISSEY, ESQ
STEPHEN NORDLICHT, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. United Media Associates, Inc, 16 Bruce Park Ave, Greenwich, CT 06830. Telephone: (203) 661-9702.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

SAMUEL M. GELFAND, MD,	*President*
JOHN A. FINKBEINER, MD,	*Past-President*
CHARLES D. SHERMAN, JR, MD,	*President-Elect*
JOSEPH F. SHANAPHY, MD,	*Vice-President*
JOHN H. CARTER, MD,	*Secretary*
GEORGE LIM, MD,	*Assistant Secretary*
MORTON KURTZ, MD,	*Treasurer*
ROBERT A. MAYERS, MD,	*Assistant Treasurer*
CHARLES N. ASWAD, MD,	*Speaker*
SEYMOUR R. STALL, MD,	*Vice-Speaker*

Councilors

Term Expires 1988

JAMES H. COSGRIFF, JR, MD,	Erie
RICHARD A. HUGHES, MD,	Warren
ANTONIO F. LASORTE, MD,	Broome
SIDNEY MISHKIN, MD,	Nassau

Term Expires 1989

ROBERT E. FEAR, MD,	Suffolk
STANLEY L. GROSSMAN, MD,	Orange
THOMAS D. PEMRICK, MD,	Rensselaer
RALPH E. SCHLOSSMAN, MD,	Queens

Term Expires 1990

DAVID M. BENFORD, MD,	New York
DUANE M. CADY, MD,	Onondaga
WILLIAM A. DOLAN, MD,	Monroe
ROBERT E. GORDON, MD,	Kings

Resident Councilor (*representing the resident physician membership*)

KATHLEEN E. SQUIRES, MD,	New York

Student Councilor (*representing the medical student membership*)

MICHAEL PACIOREK,	Onondaga

Trustees

RICHARD D. EBERLE, MD, (*Chairman*)	Onondaga
EDGAR P. BERRY, MD,	New York
JAMES M. FLANAGAN, MD,	Wayne
ALLISON B. LANDOLT, MD,	Westchester
DANIEL F. O'KEEFFE, MD,	Warren
BERNARD J. PISANI, MD,	New York
VICTOR J. TOFANY, MD,	Monroe

Executive Vice-President	DONALD F. FOY
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Vice-President Emeritus	GEORGE J. LAWRENCE, JR, MD
Director, Division of Policy Coordination	IRMA A. ERICKSON
Director, Division of Scientific Publications	PASCAL JAMES IMPERATO, MD
Director, Division of Communications	EDWARD A. HYNES
Director, Division of Computer Services	IVAN H. NAJMAN
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Governmental Affairs	GERARD L. CONWAY, ESQ
General Counsel	DONALD R. MOY, JD
Director, Division of Management and Meeting Services	ROBERT MILLER
Director, Division of Membership Support Services	EUNICE M SKELLY
Director, Division of Scientific and Educational Activities	PETER B. FARNSWORTH, MD
Director, Division of Socio-Medical Economics	MARIO V. MENGHINI
Medical Director, Committee for Physicians' Health	BARTON A. HARRIS, MD
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York.

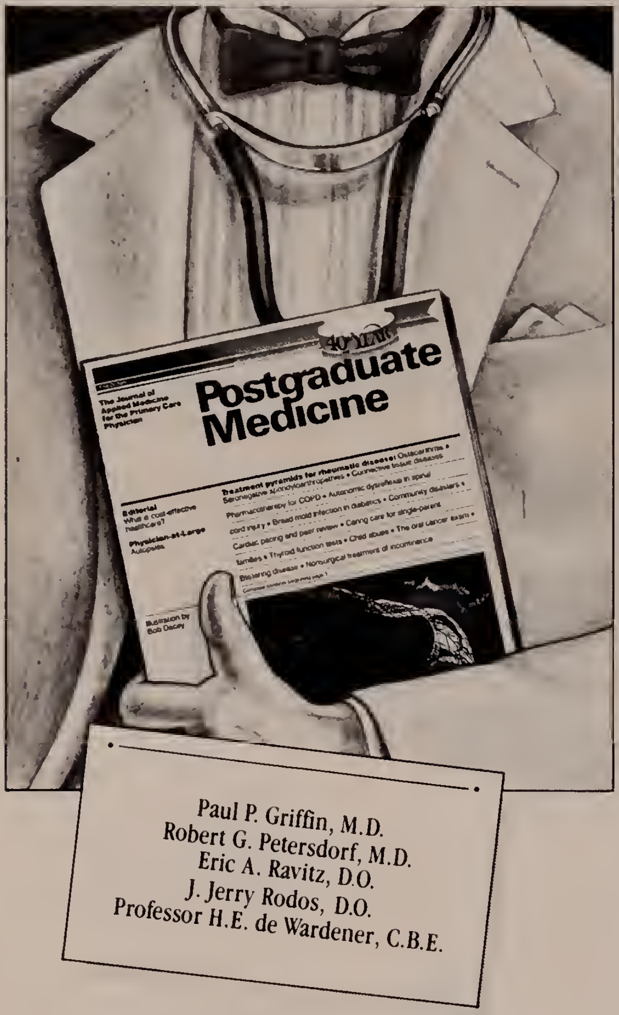
Copyright © 1987, Medical Society of the State of New York. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.

FAMOUS PHYSICIANS ARE SHOWING UP FOR OUR 40th YEAR CELEBRATION.


40 YEAR
Postgraduate Medicine
The Journal of Applied Medicine for the Primary Care Physician
Paul P. Griffin, M.D.
Robert G. Petersdorf, M.D.
Eric A. Ravitz, D.O.
J. Jerry Rodos, D.O.
Professor H.E. de Wardener, C.B.E.

Read Every Issue From Cover to Cover:

July
Symposium: Skeletal Problems
Special Article: Orthopedics 1947-1987 . . . Paul P. Griffin, M.D.

August
Symposium: Infectious Diseases
Special Article: Infectious Diseases 1947-1987 . . . Robert G. Petersdorf, M.D.

September 1
Symposium: Focus on Osteopathic Medicine . . . Eric A. Ravitz, D.O. Coordinator
Special Article: Osteopathic Medicine 1947-1987 . . . J. Jerry Rodos, D.O.

September 15
Symposium: Renal Disease
Special Article: Renal Disease 1947-1987 . . . Professor H.E. de Wardener, C.B.E.

40 YEAR
Postgraduate Medicine

Every Issue is a Celebration of Clinical Information

LIBRIUM®
chlordiazepoxide HCl/Roche ⒸIV
5-mg, 10-mg, 25-mg capsules

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Management of anxiety disorders; short-term relief of anxiety symptoms, acute alcohol withdrawal symptoms, preoperative apprehension and anxiety. Usually not required for anxiety or tension associated with stress of everyday life. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended.
Contraindications: Known hypersensitivity to the drug.
Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage. Withdrawal symptoms (including convulsions) reported after abrupt cessation of extended use of excessive doses are similar to those seen with barbiturates. Milder symptoms reported infrequently when continuous therapy is abruptly ended. Avoid abrupt discontinuation; gradually taper dosage.

Usage in Pregnancy: **Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically. Due to isolated reports of exacerbation, use with caution in patients with porphyria.
Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.
Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral Adults* Mild and moderate anxiety disorders and symptoms, 5 or 10 mg *t.i.d.* or *q.i.d.*; severe states, 20 or 25 mg *t.i.d.* or *q.i.d.* *Geriatric patients:* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)
Supplied: Librium® (chlordiazepoxide HCl/Roche) *Capsules*, 5 mg, 10 mg and 25 mg bottles of 100 and 500; Tel-E-Dose® packages of 100, available in boxes of 4 reverse-numbered cards of 25, and in boxes containing 10 strips of 10. Libritabs® (chlordiazepoxide/Roche) *Tablets*, 5 mg and 10 mg bottles of 100 and 500; 25 mg bottles of 100. With respect to clinical activity, capsules and tablets are indistinguishable.

ROCHE
Roche Products Inc.
Manati, Puerto Rico 00701

Do not substitute

Librium®

brand of

chlordiazepoxide HCl/Roche Ⓘⓥ

Nobody does it better.

ROCHE®

Copyright © 1986 by Roche Products Inc. All rights reserved.

Please see adjacent page for summary of product information.

Classified Advertising

PHYSICIANS WANTED

EMERGENCY MEDICINE POSITIONS—Full/part time emergency medicine physicians sought by multi state professional association for openings in metropolitan NY, PA, MD, DC, FL, New England and throughout U.S. Contact or send CV to Liberty Healthcare Corporation, 399 Market Street, Suite 400, Philadelphia, PA 19106, (215) 592-7400 or outside Pa (800) 331-7122.

ARIZONA-BASED PHYSICIAN RECRUITMENT firm has quality opportunities coast to coast. Available positions in most primary care and surgical specialties to include OB/GYN, Orthopedics, ER, and ENT. "Quality Physicians for Quality Clients since 1972." Call 602-990-8080; or send CV to: Mitchell & Associates, Inc., PO Box 1804, Scottsdale, AZ 85252.

PRIMARY CARE PRACTITIONERS (BC/BE) needed for unique practice situation, with ownership potential, in Long Island. Please reply with full particulars including telephone number to Dept. 443 c/o NYSJM.

BOARD CERTIFIED INTERNIST wanted for community of 10,000 (service area 45,000) in the lush Yakima Valley of central Washington. Financial assistance. Excellent income potential. Share coverage with other internists. First rate school system. Outdoor recreation second to none. Contact Prosearch, 305 NE 102nd Avenue, Portland, Oregon 97220. (503) 256-2070.

NEW YORK, BUFFALO: Seeking residency trained physicians for full time emergency department position. Moderate volume. Hourly compensation plus malpractice insurance provided. Directorship position available. Would include attractive monthly stipend. Contact: Emergency Consultants, Inc., 2240 S. Airport Road, Room 42, Traverse City, MI 49684; 1-800-253-1795 or in Michigan 1-800-632-3496.

NEW YORK, WESTERN: Seeking primary care trained physicians for full time Emergency Department positions. Moderate volume. Attractive hourly compensation, plus malpractice insurance, Director position available. Contact: Emergency Consultants, Inc., 2240 S. Airport Road, Room 42, Traverse City, MI 49684: 1-800-253-1795 or in Michigan 1-800-632-3496.

FAMILY PRACTICE—Urban/rural community in south central New York seeks qualified physicians for hospital based, structured hours position or private practice association. 300+ bed progressive acute care facility with serving area of 150,000. Close proximity to Finger Lakes provides summer/winter recreation. Excellent family environment. Guarantee plus benefits. Reply Dept. #445 c/o NYSJM.

PHYSICIANS WANTED—CONT'D

EMERGENCY MEDICINE OPPORTUNITIES in the Millard Filmore Hospital System in Buffalo, New York. This well-respected hospital has urban and suburban emergency departments that see 27,000 and 18,000 patients in the respective ERs. Associate Director and staff physicians needed. Excellent compensation plus professional liability insurance procured on your behalf. For additional information, call Mr. Dorian West at (800) 367-8952, Coastal Emergency Services, Inc., 1730 N. Lynn Street, Suite 401, Arlington, VA 22209.

POSITIONS WANTED

ROUTINE X-RAY DIAGNOSIS: Mature, U.S. trained, Board Certified Radiologist will have time available commencing summer of 1987, to participate in radiographic installation in New York City Metropolitan area. Not interested in large volume, salary commensurate. Private practice coverage would also be suitable. Reply Dept. 441 c/o NYSJM.

PRACTICES AVAILABLE

FLORIDA FAMILY/MEDICAL PRACTICE—N.E. Fort Lauderdale. Physician retiring, established 20 years. High gross, desirable demographic patient profile. Fully equipped including x-ray. Top location, terms (305) 946-2472 evenings. Excellent opportunity to get started, by-pass long years of building.

6½ YEAR INTERNAL MEDICINE PRACTICE FOR SALE in Dutchess County. Rapidly growing area, near two hospitals. Reasonable sale price. Reply Dept. 444 c/o NYSJM.

EQUIPMENT

50% OFF PREVIOUSLY OWNED MEDICAL—Laboratory, x-ray, ultrasound equipment. Buy, Sell, Repair. Appraisals by certified surgical consultants. Medical Equipment Resale & Repair, Inc., 24026 Haggerty Road, Farmington Hills, MI 48018. 1-800-247-5826. (313) 477-6880.

DISCOUNT HOLTZER SCANNING SERVICES starting at $35.00. Spacelabs holter recorders available from $1,275.00. Smallest and lightest holters update. Fast service (24–48 hours) turnover. Hook-up kits starting at $4.95. Special introductory offer of three free tests at the purchase or lease of the holter. Stress test electrodes available at 34 cents. Scanning paper available at $18.95. Cardiologist over-read available at $15.00. If interested call (301) 870-3626.

EQUIPMENT FOR SALE: Ritter hydraulic chair, Sorensen De-Luxe treatment unit and cabinet, Bausch & Lomb Ferree-Rand perimeter, Marita cuspidor, Bausch & Lomb examiners stool, Castle sterilizer, two Bausch & Lomb equipment stands—all in excellent condition. Contact Jerome Lehner, M.D., 141 Hooker Avenue, Poughkeepsie, New York 12601.

EQUIPMENT—CONT'D

ELECTROCARDIOGRAPH (EKG) Hewlett-Packard, 12 years old, perfect $395. Phone (516) 789-2891.

NEW BIOSOUND 2000 II SA & DUPLEX SCANNER less than one year old, will sell for half price. If interested call (315) 824-2414. Peter B. Del Santo, M.D., Box 304, Hamilton, NY 13346-0304.

FLUOROSCOPIC PICKER X-RAY MACHINE, 12 years old, with automatic colimation and phototiming and upright Bucky with mirror image intensifier but no television. In excellent working condition. Please call (313) 358-3410 9 A.M. to 5 P.M.

REAL ESTATE FOR SALE OR RENT

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

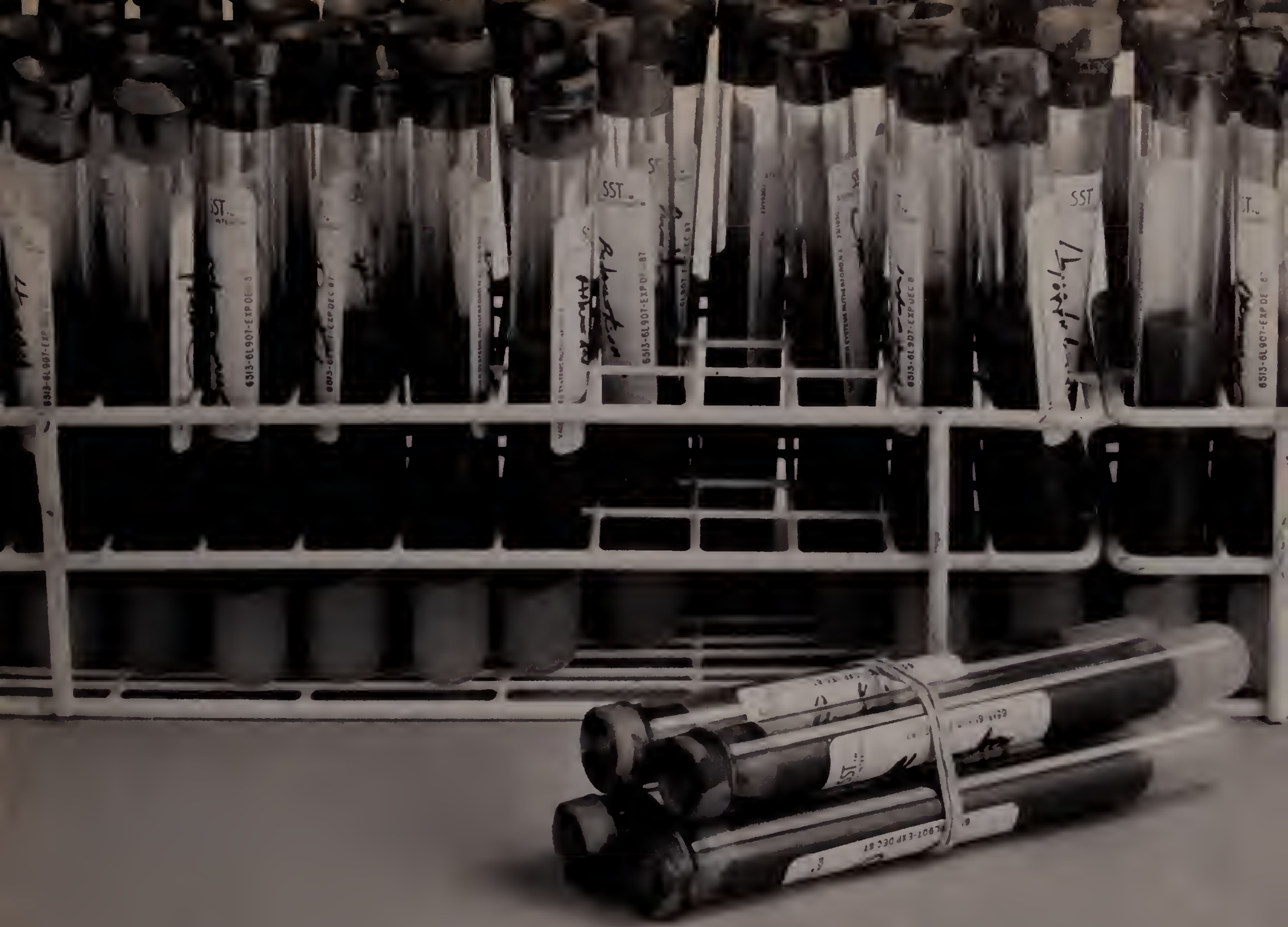
EAST SIXTIES, PROFESSIONAL BUILDING. The preferred location for internists, cardiologist, rheumatologist. 7 treatment rooms, 2 reception areas to share with three other internists. Share NYC licensed lab, radiology, thermography. Full or part time. (212) 838-2860.

PRACTICE LOCATION: Private Home with Duplex Apartment, spacious grounds, detached garage, and a fully-equipped centrally air-conditioned, 9 room Medical Suite; including large waiting room, business office, consultation room, nurses station, pharmacy, therapy room, 3 complete examining rooms, x-ray, clinical laboratory, 2 bathrooms. Apartment and office each suitable for sub-lease and added income. Turn-key operation ready for active solo or multi-specialty group. Large middle and upper class residential community, South Shore, Long Island. Write: Box 414, West Lawrence, NY 11690.

GREAT NECK COOP MEDICAL SUITE for immediate sale, 1,000 sq. ft., $180,000. Gross income $24,000 if not owner used. Can subdivide to 500 sq. ft. and reduce price proportionately. (516) 766-1860 Mr. Weinberg.

GREAT NECK MEDICAL OFFICE SPACE available for rent in New Building; 935 Northern Boulevard, State of the Art building. Near North Shore Hospital, 750 sq. ft. (516) 482-6893.

ESTATE LIKE GARDENS. Corner, 1/2 acre plus custom designed artist home. 3 bedrooms, 3 baths, a 100 sq. ft. master suite, skylights, cathedral beamed ceiling plus 10 ft. hardwood floors. 30 ft. L.R., D.R., Fireplace, French doors onto deck and patio; ground floor suite suitable for Doctor's office. Excellent location, convenient to shopping and parkways, School District #13. $350,000 Principals Only. (516) 427-4170.



HIV Positive Confirmed. Then What?

AIDS is tough to treat. Ruthless to an increasing patient population, it challenges physicians and nurses as never before in their commitment to quality care.

The Spellman Center for HIV-Related Disease at St. Clare's Hospital and Health Center in New York City, accepts the challenge posed by AIDS and HIV infection. The first facility in the tri-state region to devote its resources solely to the care of AIDS patients, the Spellman Center designates its highest priority as compassionate patient care. A dedicated staff provides comprehensive medical, nursing, psychosocial, and dental treatment in an atmosphere that is warm and understanding.

The Spellman Center—a 60-bed Acute Care Unit, Emergency Service, Outpatient, Ambulatory Infusion and Dental Clinics—accepts referrals of patients with CDC-confirmed AIDS or ARC (AIDS-related complex), and those who are seropositive for HIV. Spellman also reaches out to offer testing, counseling and treatment to persons at risk for AIDS.

If you need consultation concerning your patients, or more information, please call Dr. Debra Spicehandler, the Medical Director, at (212) 307-1161.

The Spellman Center also maintains an information hotline for patients with questions about AIDS: 1-800-433-AIDS.

The Spellman Center
for HIV-Related Disease
A Designated AIDS Treatment Facility
St. Clare's Hospital and Health Center

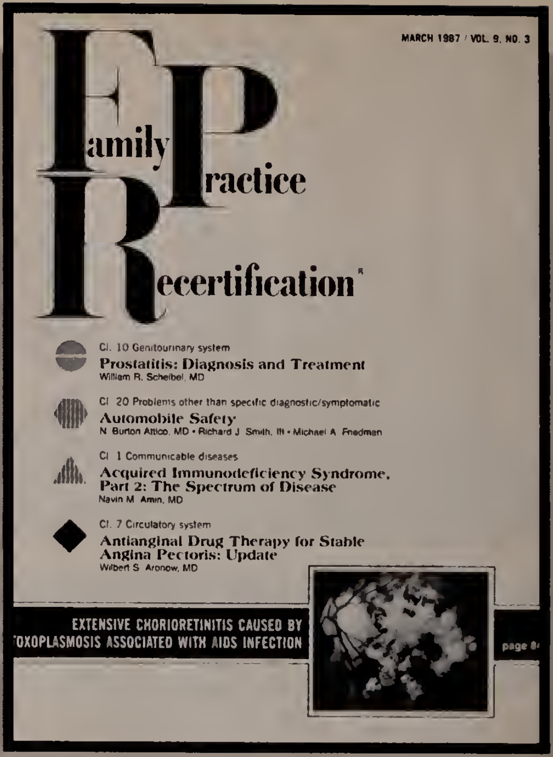
415 W 51st St
New York, NY 10019

An Affiliate of New York Medical College

The complete journal for family practice physicians

- Reaches 79,000 family physicians monthly
- Presents the most commonly seen patient problems in family practice
- Written by physicians for physicians
- The most current clinical updates in:
  - Cardiology
  - Diabetes
  - Pediatrics
  - Ob/Gyn
  - Psychiatry
  - Gastroenterology
- Provides 20 hours of CME Category 1 Credit

PRACTICAL ▪ CLINICAL ▪ EDUCATIONAL ▪ CURRENT



MARCH 1987 / VOL. 9, NO. 3

Family Practice Recertification®

Cl. 10 Genitourinary system
Prostatitis: Diagnosis and Treatment
William R. Scheibel, MD

Cl 20 Problems other than specific diagnostic/symptomatic
Automobile Safety
N Burton Attico, MD • Richard J Smith, III • Michael A Friedman

Cl 1 Communicable diseases
Acquired Immunodeficiency Syndrome, Part 2: The Spectrum of Disease
Navin M Amin, MD

Cl. 7 Circulatory system
Antianginal Drug Therapy for Stable Angina Pectoris: Update
Wilbert S Aronow, MD

EXTENSIVE CHORIORETINITIS CAUSED BY TOXOPLASMOSIS ASSOCIATED WITH AIDS INFECTION

page 8

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices unless the information is submitted at least three months prior to the event.*

AUGUST 1987

AROUND THE NATION

CALIFORNIA

August 3–7. **Modern Radiology Practice.** 30 Cat 1 Credits. Red Lion Ocean Resort, Santa Barbara. *August 8–9.* **Magnetic Resonance Imaging Update and New Techniques: A Mini-Symposium.** 12 Cat 1 Credits. Ritz-Carlton Hotel, Laguna Niguel. *August 27–30.* **Third Annual Physics and Biology of Radiology.** 26½ Cat 1 Credits. Town and Country Hotel, San Diego. *August 28–30.* **Third Annual Sonographers' Update and Review.** 16 Cat 1 Credits. Bahia Resort Hotel, San Diego. Contact: Dawne Ryals, Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

August 13–15. **Advanced Ultrasound.** 16 Cat 1 Credits. Silverado, Napa. Contact: Registrar, The American College of Obstetricians and Gynecologists, 600 Maryland Ave, SW, Suite 300 East, Washington, DC 20024-2588. Tel: 1-800-533-8811.

August 24–26. **Cardiology Update.** 15 Cat 1 Credits. San Diego. Contact: Lisa Krehbiel, Institute for Medical Studies, 30131 Town Center Dr, Suite 215, Laguna Niguel, CA 92677. Tel: (714) 495-4499.

COLORADO

August 3–7. **Differentiation of Normal and Neoplastic Cells.** University of Colorado at Boulder. Contact: Office of Conference Services, Department of Housing, University of Colorado, 500 30th St, Boulder, CO 80309-0454.

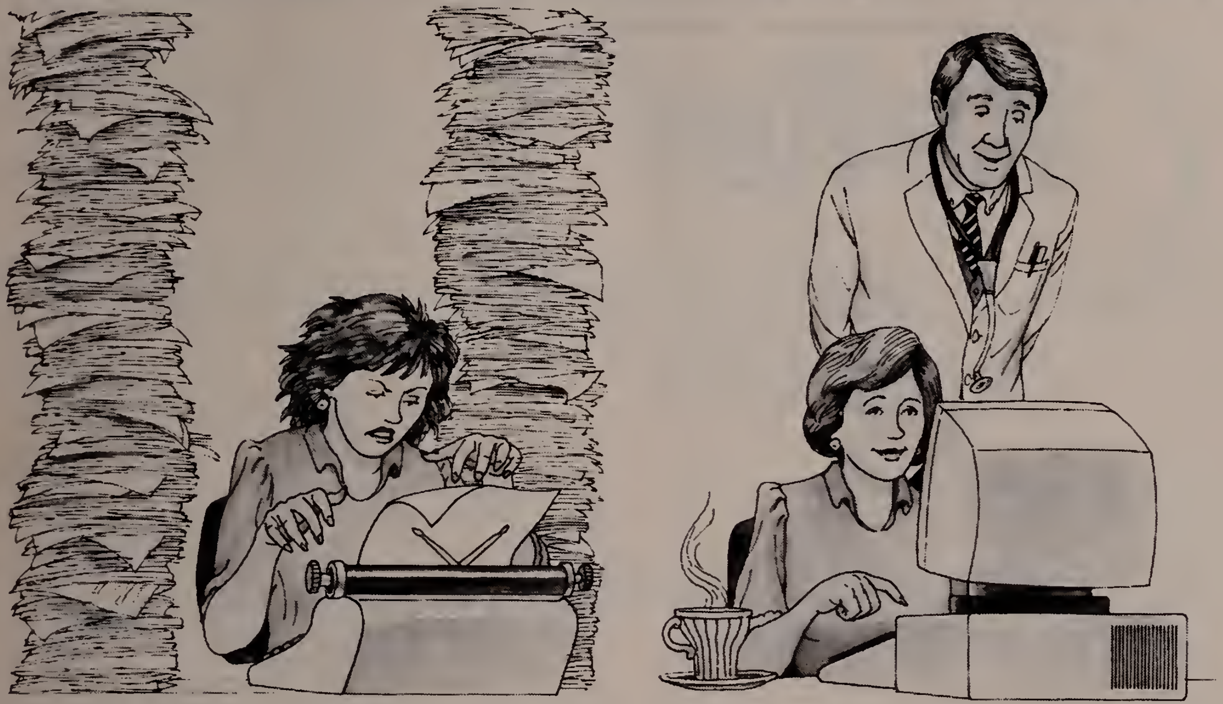
August 3–6. **30th Annual Pediatric Program.** The Given Institute of Pathobiology, Aspen. *August 7–10.* **Perinatal Medicine Conference.** Snowmass Conference Center, Snowmass. *August 6–11.* **13th Annual Primary Care Orthopedics.** The Given Institute of Pathobiology, Aspen. *August 12–15.* **Aspen Basic Science: Receptors and Disease.** The Given Institute of Pathobiology, Aspen. *August 17–21.* **Renal Disease for the Internist and Nephrologist.** The Given Institute of Pathobiology, Aspen. *August 24–28.* **Fifth Annual Internal Medicine Board Review.** USHSC, Denver. Contact: Office of Continuing Medical Education, 4200 East 9th Ave, Box C-295, Denver, CO 80262. Tel: (303) 394-5195.

HAWAII

August 22–29. **Seventh Annual Symposium on Fine Needle Aspiration.** 24½ Cat 1 Credits. Royal Lahaina Resort, Kaanapali Beach, Maui. Contact: Extended Programs in Medical Education, Registration Office, Room 575-U, University of California, San Francisco, CA 94143-0766. Tel: (415) 476-4251.

(*continued on p 16A*)

THERE ARE ONLY TWO WAYS TO SUBMIT INSURANCE CLAIMS...



HOURS vs. OURS

Now receive your insurance payments in about 10 days, while ridding your practice of cumbersome, time-consuming paperwork. You know what a burden it is to submit insurance claim forms—the paperwork, the filing, the mailing, the waiting, and *waiting*, and *waiting*.... But all that's changed. Now, with **Step-Saver** *Paperless Claims*, you can submit insurance claims and receive your money in days instead of months, with no per-claim charge.

The Step-Saver way involves 3 easy steps:
1. Log in the patient information.
2. Enter the procedure and diagnosis codes.
3. Transmit electronic information to insurer's computer, automatically by phone.

If you make an error, you can correct it immediately—no waiting 4 to 6 weeks to find out your claim was not processed. With *Paperless Claims* from **Step-Saver**, there's no paperwork and no inconvenience.

In minutes, you transmit insurance claims and get a print-out receipt of all submissions. You get your money *quicker, with less hassle!*

To prove our point, we'll be glad to come to your office and give you a free demonstration of our time-saving Medical Turnkey Computer System.

Call **Step-Saver** today and let us show you how to streamline your practice and receive your third party insurance payments faster.

Step-Saver®
Step-Saver Medical Systems

1-800-441-0216
In PA: (215) 668-1600

One Bala Plaza, Suite 417, Bala Cynwyd, PA 19004

IBM
Authorized Value Added Dealer
Personal Computers

©1987 Step-Saver Medical Systems, Inc.

News from DISTA about a new dosage form of cephalexin

ANNOUNCING NEW

Keflet™ TABLETS

cephalexin

All the advantages of cephalexin in a convenient tablet form

- Backed by over 15 years of clinical experience
- Smaller tablet is specially shaped and coated for easier swallowing
- May enhance patient compliance, particularly among the elderly
- Tablet dosage form may be appreciated by patients of all ages

NEW Keflet Tablets are available as:

250-mg
Tablets

500-mg
Tablets

Keflet is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-sensitive patients.

© 1987 DISTA PRODUCTS COMPANY

Brief Summary. Consult the package literature for prescribing information.
Indications and Usage: Keflet™ Tablets (cephalexin, Dista) are indicated for the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

Respiratory tract infections caused by *Streptococcus pneumoniae* and group A β-hemolytic streptococci (Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. Keflet is generally effective in the eradication of streptococci from the nasopharynx; however, substantial data establishing the efficacy of Keflet in the subsequent prevention of rheumatic fever are not available at present.)

Otitis media due to *S pneumoniae*, *Haemophilus influenzae*, staphylococci, streptococci, and *Neisseria catarrhalis*

Skin and skin-structure infections caused by staphylococci and/or streptococci

Bone infections caused by staphylococci and/or *Proteus mirabilis*

Genitourinary tract infections, including acute prostatitis, caused by *Escherichia coli*, *P mirabilis*, and *Klebsiella* sp.

Note—Culture and susceptibility tests should be initiated prior to and during therapy. Renal function studies should be performed when indicated

Contraindication: Keflet is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: BEFORE CEPHALEXIN THERAPY IS INSTITUTED, CAREFUL INQUIRY SHOULD BE MADE CONCERNING PREVIOUS HYPERSENSITIVITY REACTIONS TO CEPHALOSPORINS AND PENICILLIN CEPHALOSPORIN C DERIVATIVES SHOULD BE GIVEN CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS

SERIOUS ACUTE HYPERSENSITIVITY REACTIONS MAY REQUIRE EPINEPHRINE AND OTHER EMERGENCY MEASURES

There is some clinical and laboratory evidence of partial cross-allergenicity of the penicillins and the cephalosporins. Patients have been reported to have had severe reactions (including anaphylaxis) to both drugs.

Any patient who has demonstrated some form of allergy, particularly to drugs, should receive antibiotics cautiously. No exception should be made with regard to Keflet.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics Such colitis may range in severity from mild to life-threatening

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C difficile* Other causes of colitis should be ruled out.

Usage in Pregnancy—Safety of this product for use during pregnancy has not been established.

Precautions: *General*—Patients should be followed carefully so that any side effects or unusual manifestations of drug idiosyncrasy may be detected If an allergic reaction to Keflet occurs, the drug should be discontinued and the patient treated with the usual agents (eg, epinephrine or other pressor amines, antihistamines, or corticosteroids).

Prolonged use of Keflet may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug

Keflet should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

Indicated surgical procedures should be performed in conjunction with antibiotic therapy.

As a result of administration of Keflet, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy—Pregnancy Category B—The daily oral administration of cephalexin to rats in doses of 250 or 500 mg/kg prior to and during pregnancy, or to rats and mice during the period of organogenesis only, had no adverse effect on fertility, fetal viability, fetal weight, or litter size. Note that the safety of cephalexin during pregnancy in humans has not been established

Cephalexin showed no enhanced toxicity in weanling and newborn rats as compared with adult animals. Nevertheless, because the studies in humans cannot rule out the possibility of harm, Keflet should be used during pregnancy only if clearly needed

Nursing Mothers— The excretion of cephalexin in the milk increased up to 4 hours after a 500-mg dose; the drug reached a maximum level of 4μg/mL, then decreased gradually, and had disappeared 8 hours after administration Caution should be exercised when Keflet is administered to a nursing woman.

Adverse Reactions: *Gastrointestinal*—Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely. The most frequent side effect has been diarrhea. It was very rarely severe enough to warrant cessation of therapy Dyspepsia and abdominal pain have also occurred As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely

Hypersensitivity— Allergic reactions in the form of rash, urticaria, angioedema, and, rarely, erythema multiforme, Stevens Johnson Syndrome, or toxic epidermal necrolysis have been observed. These reactions usually subsided upon discontinuation of the drug Anaphylaxis has also been reported

Other reactions have included genital and anal pruritus, genital moniliasis, vaginitis and vaginal discharge, dizziness, fatigue, and headache Eosinophilia, neutropenia, thrombocytopenia, and slight elevations in SGOT and SGPT have been reported.

PV 2211 DPP

[111786]

Additional information available to the profession on request from

Dista Products Company
Division of Eli Lilly and Company
Indianapolis, Indiana 46285
Mfd by Eli Lilly Industries, Inc
Carolina, Puerto Rico 00630

720073

NEW YORK STATE
JOURNAL OF MEDICINE

July 1987 • Volume 87, Number 7

COMMENTARIES

Adrenal medullary grafts transplanted into the brain as a treatment for Parkinson disease

Brain tissue transplantation is now a major tool for treating Parkinson disease (PD), and conceivably other neurodegenerative diseases.[1] PD is a progressive disorder of the central nervous system that affects approximately one million people in the United States. Clinically it is characterized by bradykinesia, gait difficulty, postural instability, rigidity, and tremor.[2] Pathologically it is characterized by a degeneration of the pigmented neurons in the substantia nigra. Biochemically it is characterized by a decrease in dopamine and its major metabolite, homovanillic acid, in the striatum (caudate and putamen). The first clinical symptoms of PD do not usually appear until there has been at least an 80% loss of pigmented nigral neurons. This loss is usually associated with an 80% depletion of striatal dopamine. Repletion of the deficient dopamine through the administration of its immediate precursor, levodopa, combined with an extracerebral dopa decarboxylase inhibitor, has led to clinical improvement in most patients. Inevitably, however, despite treatment, patients develop more symptoms and their response to levodopa becomes erratic. While the response can be improved with the addition of dopamine agonists, medical therapy of patients with advanced Parkinson disease remains unsatisfactory.

Because the clinical symptoms of PD result from the loss of brain tissue (substantia nigra neurons), some investigators have turned to the idea of replacing the lost brain tissue with new tissue. Two sources of donor tissue have been investigated. One involves using nigral cells from embryos and transplanting them into adults.[3] The embryonal nigral tissue must be of an optimal age (which varies from species to species), and must be placed in an optimal site (the caudate versus the putamen versus the nigra, in continuity or not in continuity with the ventricles). Because of the complex moral and ethical issues involved in transplantation of human fetal tissue (the tissue is obtained from aborted fetuses), attempts have been made at cross-species transplantation. While the brain is an immunologically privileged site, these cross-species transplants have not been uniformly successful. Some degree of rejection of donor tissue takes place, requiring that the host be treated for a short time with cyclosporine. Researchers in Sweden have recently demonstrated that human fetal substantia nigra can be successfully grafted to the denervated striatum of immunosuppressed rats.[4]

A second approach to treating Parkinson disease with transplanted tissue involves obtaining the tissue from body organs other than the brain, such as the adrenal medulla.[5,6] Since the patients serve as their own donors, the moral and ethical issues involved in transplanting tissue from one individual to another are circumvented. Studies using different animal models of PD have shown that grafting tissue from the adrenal medulla into the animal's striatum or into the adjacent ventricles can temporarily ameliorate the symptoms of PD.

Investigators in Sweden applied these techniques to four patients with advanced Parkinson disease.[2] Fifteen to 20 pieces of adrenal medulla, each piece measuring 1–2 mm^3, were placed into a cell suspension. The suspension was then implanted into the right caudate of two patients, and into the right putamen of two other patients. Two of the patients showed temporary improvement. At the time it was thought that since adrenal grafts do not reinnervate the host brain, the temporary improvement was due to a release of dopamine by the adrenal tissue. As adrenal tissue normally makes norepinephrine, and not dopamine, it was postulated that under the influence of the brain, the adrenal medullary cells were "induced" into making dopamine.

Undeterred by these negative results, investigators in Mexico have reported marked improvement in 11 patients (two reports published and nine unpublished as of this writing) through autografts of adrenal medulla into the caudate nucleus.[7] The Mexican study differs from the Swedish study in that the Mexicans used more adrenal medullary tissue, did not put the tissue into a suspension (presumably preserving the continuity of the adrenal cells), implanted the cells directly into the caudate, and also anchored them in such a way that some of the cells remained in contact with the ventricular fluid (and thus could influence the striatum on the opposite side).[7]

On April 10, 1987, we visited the Hospital de Especialidades Centro Medico La Raza and spoke with Dr Ignacio Madrazo Navarro, the Chief of Neurosurgery, who performed the operation, and Dr Rene Drucker-Colin, of the Department of Neurosciences, Instituto de Fisiologia Universidad Nacional Autonoma de Mexico, who coordinated the scientific evaluation of the procedure.

At that time we examined four of the 11 patients who had had the operation, including two patients described in the *New England Journal of Medicine*.[7] We also examined one patient who was about to undergo the operation and who has since successfully undergone it. Although the patients were young (range, 34–52 years) compared to most patients with Parkinson disease, clinically they did have PD. They did not have progressive supranuclear palsy, Shy-Drager syndrome, essential tremor, or any other condition. There was no history of toxic exposure. Based on a comparison of the patients' preoperative appearance, as seen on videotape, with their conditions when we examined them (with all the limitations inherent in such a comparison), the patients appeared to be much improved. The most dramatic improvement occurred in the two patients described in the *New England Journal of Medicine*.[7] The first patient had been wheelchair-bound and almost totally dependent on others for performing all the activities of daily living. Although still clearly parkinsonian, he was now without rigidity or tremor. He was mildly to moderately bradykinetic and had moderate gait difficulty. The second patient had had severe parkinsonism preoperatively, with a marked tremor. He was almost totally dependent on others for performing the activities of daily living. At the time of our examination the patient was without rigidity or tremor. He was mildly bradykinetic and had virtually no gait difficulty. Although there was some variability in the patients' parkinsonism throughout the day, there were no dramatic fluctuations such as those seen in patients on levodopa who are experiencing "wearing-off" or "on/off" phenomena. Before surgery, two of the four patients we examined had been receiving levodopa, 1,500 mg, combined with carbidopa, 150 mg. The other two patients had been unable to tolerate levodopa. After surgery, one of the patients had been taken off levodopa/carbidopa completely, and the other was receiving one sixth the preoperative dose. All four patients were receiving anticholinergics. Although none of the patients was "cured," the improvement appeared to be comparable to that initially achieved with levodopa by Cotzias et al.[8] The sustained improvement, which began two weeks after the surgery (following an initial but evanescent improvement in the first few days), has been maintained in the first two patients for more than a year (as of the time of our examination).

Two of the 11 patients have died. Because they had undergone craniotomies, all 11 patients had been placed on anticonvulsants as a prophylactic measure after the surgery. Seizures developed in one patient despite the anticonvulsive therapy, and he died 45 days after surgery. Postmortem examination revealed only dilation of the cortical veins without hemorrhage or infarction at the side of the brain where the craniotomy was performed. The surgeons speculate that this death, although not an immediate consequence of the surgery, was somehow related to the larger than usual amount of adrenal medullary tissue used in this patient, who also had particularly severe PD. The analysis of this patient's death is still ongoing and full details will appear later. The examination also confirmed that the patient had Parkinson disease, in that there was a paucity of pigmented neurons in the substantia nigra and Lewy bodies were seen in some of the neurons. The grafted tissue appeared viable, and there was no evidence of rejection.

The second patient died during a trip to California, apparently of a nonneurological event. His death also occurred 45 days after surgery. A portmortem examination of this patient has been performed but the details of the examination are not available as of the time of this writing.

A surgical team at Vanderbilt University in Nashville, Tennessee, has performed a comparable operation in the United States. It is clear that the idea of reversing the symptoms of parkinsonism (and perhaps other neurologic disorders) by grafting tissue into the brain, initially done in Sweden, has caught the imagination of the world. Although the team from Mexico is impressive and their results are promising, the two deaths are disturbing and indicate that initially the operations should be performed by experienced surgical teams with sound scientific back-up at large medical centers.

It is not yet clear which of the two tissue transplant approaches to treating Parkinson disease will prove successful: autologous grafts of adrenal medulla (or sympathetic nerve ganglia), or fetal transplants (from human fetuses, from other species, or from cells grown in tissue culture[9]). At present, attention is being directed toward adrenal implants. However, understanding the mechanism through which the adrenal transplants survive in the brain and reverse the symptoms of Parkinson disease will require an ongoing basic research effort, including studies of embryonal tissue and cells grown in tissue culture.

ABRAHAM LIEBERMAN, MD
Chairman, Medical Advisory Board
American Parkinson Disease Association

Professor of Neurology
New York University Medical Center
New York, NY 10016

MAX KOSLOW, MD
Research Associate Professor of
Neurosurgery
New York University Medical Center
New York, NY 10016

PAUL MAESTRONE, DVM
Director, Scientific Advisory Board
American Parkinson Disease Association
New York, NY 10038

1. Lieberman A: Update on Parkinson disease. *NY State J Med* 1987; 87:147–153.
2. Lieberman A: Parkinson's disease: A clinical review. *Am J Med Sci* 1974; 267:66–80.
3. Freed WJ, Perlow MJ, Karoum F, et al: Restoration of dopaminergic function by grafting on fetal rat substantia nigra to the caudate nucleus: Long term behavioral, biochemical, and histochemical studies. *Ann Neurol* 1980; 8:510–519.
4. Stromberg I, Bygdeman M, Goldstein M, et al: Human fetal substantia

nigra grafted to the dopamine denervated striatum of immunosuppressed rats. Evidence for functional reinnervation. *Neurosci Lett* 1986; 71:271–276.

5. Freed WJ, Morihisa JM, Spoor E, et al: Transplanted adrenal chromaffin cells in rat brain reduce lesion induced rotational behaviour. *Nature* 1981; 292:351–352.

6. Olson L, Backlund EO, Gerhardt G: Nigral and adrenal grafts in parkinsonism. Recent basic and clinical studies. *Adv Neurol* 1986; 45:85–94.

7. Madrazo I, Drucker-Colin R, Diaz V, et al: Open microsurgical autograft of adrenal medulla to the right caudate nucleus in two patients with intractable Parkinson's disease. *N Engl J Med* 1987; 316:831–834.

8. Cotzias GC, Van Woert MH, Schiffer LM: Aromatic amino acids and modification of parkinsonism. *N Engl J Med* 1967; 276:374–379.

9. Jaeger CB: Immunocytochemical study of PC12 cells grafted to the brain of immature rats. *Exp Brain Res* 1985; 59:615–624.

Ethical dilemmas imposed by the Medicare hospice regulations

The development of the hospice movement in the United States more than a decade ago brought with it conflict about a number of ethical questions.[1,2] Debate over the quality of life, the right to refuse treatment, and the right to die with dignity has filled the pages of the medical and philosophical journals as well as those of the lay press. Certainly the hospice movement has sparked research into the badly neglected area of pain control. But few in the hospice movement would have been able to predict the moral issues that the hospice programs must now confront as a result of the Medicare hospice regulations. An examination of those regulations, their implications, and the dilemmas they pose is important, not only for that segment of the population requiring hospice care, but as a window through which to view the philosophic approach of the Reagan administration towards health care for the elderly.

Hospice programs must comply with a number of specific regulations in order to become certified. The Medicare hospice regulations define the eligible population as those patients receiving Medicare benefits who carry a diagnosis of a disease for which life expectancy is six months or less, and for whom a shift from curative care to palliative care is planned.[3] Patients who wish to elect the Medicare hospice benefit through a Medicare-certified hospice program sign a form stating their awareness of their terminal illness and waiving all further aggressive therapy.[3] The hospice program then becomes financially responsible for the patient's disease-related care, including medical equipment, therapy, supplies, ambulance transfer, and virtually the entire cost of medication.[3] The hospice program is reimbursed at a per diem rate,[3] which has been fixed at $63.17 for home care, $65.33 for inpatient respite care, and $281 for inpatient hospice care, irrespective of the actual costs incurred by the program. For the Medicare hospice program, 80% of the aggregate patient days must be spent at home, and an average of the total costs for hospice patients may not exceed $6,500.[3]

A patient may opt out of the program at any point before 90, 180, or 210 days. Medicare does not reimburse the hospice program for care provided beyond 210 days despite the fact that the patient may survive and require care indefinitely, nor is the hospice allowed to seek alternate sources of reimbursement for any of the cancer-related care of the Medicare patient.[3] It is the expectation of the Medicare program that a patient following an undramatic course for more than 90 days but less than 210 days allows the hospice to realize a profit from such care.

Currently only 10% of the hospices registered with the National Hospice Organization have applied for Medicare certification.[4] While a sizable percentage of the other nonparticipating programs have yet to take on any patient care responsibility, many larger, active programs have deliberately chosen not to accept the yoke of the regulations. For those programs that have become Medicare certified, some very critical issues have arisen solely because of the regulations themselves.[5-7]

Two dilemmas become immediately obvious: What happens to the patient whose disease and therapy can be anticipated to be expensive, either at the time of admission or once in the Medicare hospice program, and what of the newly referred patient not anticipated to survive 90 days? Certainly, a reasonable percentage of patients whose life expectancy is less than six months could benefit from some form of palliative treatment including radiation, chemotherapy, and even surgery. Other patients require expensive delivery systems for medication, such as continuous intravenous, subcutaneous, or epidural narcotics. Other patients require expensive equipment or services at home to provide them some degree of quality of life. Is a hospice program to deny these patients admission because of the financial burden they impose? Should a hospice risk its future by assuming the weight of that expense?[7,8]

Other patients later develop disease complications which require expensive hospitalization and treatment. Pathologic long-bone fractures are best handled by internal fixation and radiation; bowel obstruction requires surgical decompression or bypass; spinal cord compression is best handled by radiation and laminectomy, or perhaps with an intensive course of chemotherapy which the hospice must subsidize.[9] Must a hospice risk bankruptcy to do so? May a hospice deny its Medicare patients these treatments?

Quality of life, though hard to measure, intuitively seems enhanced by allowing patients to take full advantage of all their physical capabilities.[10] Beyond the physician and nurse in a program, such allied health professionals as physical, occupational, and speech therapists; social workers; and dentists may immeasurably contribute to a patient's regaining or maintaining skills and redeveloping self-esteem. Bereavement follow-up for the surviving family members is required by regulation, but is not reimbursed. Can a hospice restrict access to these resources? Must the hospice outstrip its per diem reimbursement to provide sufficient manpower for all of its patients?

By far the greatest financial expense in caring for the terminally ill occurs in the last two weeks of life.[11] As a patient becomes progressively debilitated, more expensive equipment is required, such as hospital beds, oxygen therapy, urinary catheters, and new types of medication. Family stress sometimes requires the availability of more manpower, both for hands-on delivery of care and for psychosocial backup. A program providing care for such a person for extended periods of time may have built up sufficient financial reserves to cover these new costs, or it may have been able to foresee and prevent some problems, but newly referred patients at this late point in their course may be suffering from inexorable pain and other severe, acute symptoms. Their families may be extremely stressed by the situation.[12] Can a hospice turn its back on these people? Who will reimburse the financial losses for the hospice's act of compassion?

The hospice movement has always advocated the provision of appropriate levels of care. Because acute care facilities are usually inappropriate for the hospice population, hospice care may be much less costly to deliver than its standard counterpart.[12,13] The cost effectiveness of hospice care occurs because inappropriate therapies are avoided, and is not a specific goal in itself.[5]

In order to provide all appropriate care and eliminate inappropriate treatments, expertise in all aspects of care must be available. However, the structure of the Medicare hospice regulations rewards cost-cutting without enforcing the provision of quality care. Recent moves in Congress which would allow for brokering of services by nonhospice proprietary agencies would dilute the quality of care for hospice patients by bringing in professional caregivers who are not oriented to the hospice philosophy.[3] Even now the hospice medical director is not required to provide hands-on service.[3] It is particularly disturbing that the structure of Medicare hospice reimbursement varies so markedly from that of other federal programs, even the system of diagnosis-related groups.[5,6] At least in the latter program, compensation is provided for appropriate and effective therapy. Under the hospice regulations, fixed per diem reimbursement without regard to cost or to patient longevity may penalize programs that provide appropriate care.[14,15] It may also lead to the development of a two-tiered system in which Medicare patients must meet unduly harsh standards for program admission, and in which a risk is created that appropriate services will be withheld from Medicare patients.[4]

The National Hospice Study demonstrated that although hospice patients were much less likely to receive costly interventions such as surgery, chemotherapy, transfusions, and intravenous therapy than patients in a conventional oncology setting, they were provided equal access to other expensive treatments such as respiratory support and palliative radiation therapy.[16] That study ended in March 1983, prior to the implementation of the Medicare hospice regulations, so it fails to address the concerns expressed here, that financial considerations may lead to the withholding of appropriate care.[17]

Equally appalling are the demographic effects these regulations promulgate. Because of the fixed-rate reimbursement schedule, the hospice program will naturally tend to select patients dying of cancer (since this is one of the few areas in which physicians think that they can reliably prognosticate) whose care needs are modest and whose families are both healthy and supportive, with adequate resources to sacrifice the time needed to provide care.[18,19] As a result, the typical hospice patient will continue to be white and middle class. The Medicare patient with poor family supports or with major needs is at risk for exclusion from hospice care, widening a social gap between those who are served and those who are left out.[18]

The hospice movement was founded on the premise that patients with end-stage disease for whom cure or prolonged preservation of life was impossible were being inappropriately treated. The fact that care directed to the patients' physical symptoms and emotional needs might also be more cost effective was an inconsequential byproduct. The formulators of the Medicare hospice legislation have seized the means of such cost-cutting, the avoidance of inappropriate, highly technical care, and made that an end in itself. However, the use of advanced and expensive techniques is sometimes necessary. Acknowledgment of this fact by adjusting the financial risks is now imperative. Denial of appropriate care for the terminally ill to ensure fiscal survival makes a mockery of the hospice philosophy.

LAWRENCE M. SHERMAN, MD
Clinical Instructor
Department of Surgery
SUNY at Buffalo
School of Medicine

Medical Director
Hospice Buffalo, Inc

WILLIAM E. FINN, BS
Administrator
Hospice Buffalo, Inc
Snyder, NY 14226

1. Bayer R, Callahan D, Fletcher J, et al: The care of the terminally ill. Morality and economics. *N Engl J Med* 1983; 302:1490–1494.
2. Gibson DE: Hospice: Mortality and economics. *Gerontologist* 1984; 24: 4–8.
3. US Dept of Health and Human Services, HCFA: Medicare program, hospice care final rule. *Federal Register* 1983; 48:56008–56036.
4. Jehan CB: Is hospice Medicare certification worth the risk? *Caring* 1985; 2:41–44.
5. Proffitt L: *Hospice Handbook*. Rockville, Md, Aspen Publications, 1986, pp 303–322.
6. Pryga E, Bachofer H: *Hospice Care Under Medicare*. Chicago, Ill, Office of Public Policy Analysis, American Hospital Association, 1984.
7. McDonnell A: *Quality Hospice Care*. Owings Mills, Md, National Health Publishing, 1986, pp 81–83.
8. US Dept of Health and Human Services, Inspector General: *A Program Inspection of Hospice Care*. Washington, DC, June 1984.
9. National Association for Home Care: *NAHC Report*. Washington, DC, 1983, p 212.
10. Tigges KN, Sherman LM: The treatment of the hospice patient. From occupational history to occupational role. *Am J Occup Ther* 1983; 37:235–238.
11. Brooks CH, Smyth-Staruch K: Hospice home care cost savings to third-party insurers. *Med Care* 1984; 22:691–703.
12. Amenta M: Accreditation and the movement's future. *Am J Hospice Care* 1984; 1:10–14.
13. Congressional Budget Office: Cost estimate of HR 5180. Washington, DC, June 25, 1982.
14. Sherman L: Implications of the Medicare regulations on hospice care. Presented at the First Annual Symposium on Hospice Care, Boston, June 1985.
15. Knowles C: Reimbursement and the Medicare-certified hospice. *Am J Hospice Care* 1985; 5:15–21.

16. Greer D, Mor V: An overview of national hospice study findings. *J Chron Dis* 1986; 39:5-7.
17. Taylor L: Appropriate patient characteristics for hospice care. *Am J Hospice Care* 1985; 1:11-18.
18. Lynn J: Ethics in hospice care, in *Hospice Handbook—A Guide for Managers and Planners*. Rockville, Md, Aspen Publications, 1985, pp 303-324.
19. Finn Paradis L, Schultz J: Hospice and home health together in harmony or discord? *Caring* 1985; 2:9-16.

Cutaneous polyarteritis nodosa

In this issue of the *Journal*, Meredith et al[1] describe a patient who may have a form of polyarteritis differing from most in its protracted clinical course and in its predilection for skin and, to a lesser extent, muscle and nerves. Lesions of cutaneous polyarteritis nodosa (CPN) have been observed to persist in some patients for as long as 25 years.[2] CPN has been discounted as a cause of death in the handful of patients whose postmortem examinations failed to detect evidence of polyarteritis in locations other than the skin.[2,3]

I have had the opportunity of observing the erratic and protracted course of this disease in a male patient over a period of several years.[4] His illness, which began 16 years ago, exemplifies the salient features of CPN. In this patient the disease has been characterized by painful red streaks and nodules (and at one time redness of the entire right foot) that undergo spontaneous or drug-induced remissions lasting three to six months. The microscopic appearance of two biopsied nodules from this patient was classical for polyarteritis. Early in the course of his illness, when cutaneous arteritis was florid, the aorta and the iliac and renal arteries were normal on arteriography. In 1984, two nodules persisted for two weeks on the right hand; all other nodules have been on the right leg or foot. Since 1981, he has had pain in the right metatarsal-phalangeal joints and now has cock-up deformities of the toes, but with no abnormalities noted on radiographs of the foot. Nonsteroidal anti-inflammatory drugs appear to suppress both the arthralgia and the arterial inflammation.

CPN has been differentiated from the systemic form of the disease, and its protracted course, regardless of treatment, has been largely emphasized by dermatologists. It is seldom covered in textbooks of general medicine or even of rheumatology, where, with an occasional exception, it is not mentioned at all or is subsumed under the topic of children's diseases (many of the patients have been in their early teens). In their text *The Vasculitides*, Cupps and Fauci[5] discuss CPN as a distinct entity and note that its chronic course is associated with a good long-term prognosis, as opposed to that of systemic polyarteritis. They cite a five-year survival rate of 13% for untreated polyarteritis patients. Perhaps the emphasis on visceral lesions, with the threat of catastrophe, and the urgency of aggressive treatment have so colored the picture of polyarteritis that confinement of arteritis to the skin or muscles is not often considered. CPN may not be as rare as the scarcity of its description in general medicine publications would imply: 38 patients were described in two papers,[2,3] and Borrie[3] found that 10% of all polyarteritis patients in one institution had the chronic cutaneous form.

Biopsy provides the only sure means for diagnosis of CPN. The lesions may be ulcerated or gangrenous, or they may manifest as urticaria, livedo reticularis, or bullae. Nodules were found in 34 of 38 patients, and livedo in 28 of these 38.[2,3] The nodules are usually tender and painful; they vary from 0.5 to 2-3 cm in size and range up to 100 in number. They resemble the skin lesions of systemic polyarteritis in that they predominate on the lower extremities; however, the skin lesions of systemic disease may more often appear as nondiagnostic maculopapular, purpuric, or urticarial rash. Nodules have been described in about 15% of patients said to have polyarteritis.[3,5] Myositis, causing aching and stiffness, has often been observed. Arthralgias were noted in almost half of one group of 23 patients who were followed for a mean of nine years. Sensory disturbances, pain, absent reflexes, and weakness due to peripheral neuropathy were observed in six of the 23 patients.[2] Nondestructive arthritis has been reported in a few patients. The pathogenesis of the arthritis is not clear, inasmuch as biopsies have failed to show synovial arteritis.[6,7] The results of laboratory tests have been generally unremarkable except for elevated erythrocyte sedimentation rates.

The absence of visceral lesions and the long duration of active disease shape the conclusion that cutaneous polyarteritis nodosa differs sufficiently from the lethal systemic form of the disease to justify special consideration. It has been suggested that if the clinical and laboratory evaluations show no visceral disease at the initial examination of patients with nodular cutaneous polyarteritis nodosa, the patients will most often remain free of visceral disease.[2,3]

GENE V. BALL, MD
Jane Knight Lowe Professor of Medicine
in Rheumatology
Department of Medicine
Division of Clinical Immunology and
Rheumatology
The University of Alabama at Birmingham
Birmingham, AL 35294

1. Meredith GS, Mitnick HJ, Burstin HE, et al: Polyarteritis nodosa presenting with migratory soft tissue swelling. *NY State J Med* 1987; 87:402-403.
2. Diaz-Perez JL, Winkelmann RK: Cutaneous periarteritis nodosa. *Arch Dermatol* 1974; 110:407-414.
3. Borrie P: Cutaneous polyarteritis nodosa. *Br J Dermatol* 1972; 87:87-95.
4. Rawlinson T, Ball GV: Chronic cutaneous polyarteritis nodosa: A distinct subset of polyarteritis nodosa? [letter]. *Arch Intern Med* 1981; 141:961.
5. Cupps TR, Fauci AS: *The Vasculitides*. Philadelphia, WB Saunders, 1981.
6. Mekori YA, Awai LE, Wiedel JD, et al: Cutaneous polyarteritis nodosa associated with rapidly progressive arthritis. *Arth Rheum* 1984; 27:574-578.
7. Smukler NM, Schumacher HR Jr: Chronic nondestructive arthritis associated with cutaneous polyarteritis. *Arth Rheum* 1977; 20:1114-1120.

The effectiveness of film and videotape in teaching cardiopulmonary resuscitation to the lay public

JOSEPH SCHLUGER, MD; JOSEPH G. HAYES, MD; GERARD M. TURINO, MD; SYLVIA FISHMAN, EDD;
ARTHUR C. FOX, MD

ABSTRACT. "CPR: It's About Time" is a film/videotape program designed to teach the essential skills of cardiopulmonary resuscitation (CPR) to large numbers of the lay public, with or without hands-on manikin practice. This study evaluated the ability of 257 previously untrained volunteer viewers to perform the essential skills of single-rescuer cardiopulmonary resuscitation by testing them on recording manikins immediately following film viewing. Trained evaluators scored the volunteers' performance, and judged that 43% performed key skills adequately enough to "save a life." Preliminary results suggest that the film/videotape may be a valuable, inexpensive tool for teaching emergency care to large numbers of the lay public, who were previously otherwise unreachable through traditional courses and traditional teaching methods.

(NY State J Med 1987; 87:382-385)

Improved survival for victims of out-of-hospital cardiac arrest has been repeatedly observed and confirmed.[1-7] Two essential components for successful resuscitation are bystander-initiated cardiopulmonary resuscitation (CPR) of witnessed cardiac arrest[6,8] and the rapid response (eight minutes or less) of paramedic teams.[4] In the absence of either, there is little increase in survival.

The American Heart Association (AHA) has suggested that 20% of the adult population in any given community must be trained in cardiopulmonary resuscitation in order to substantially reduce out-of-hospital mortality from cardiac arrest.[9] Others have suggested that a higher percentage of trained lay persons and novel approaches to training[6] may be needed to effect a significant reduction in morbidity and mortality from unexpected cardiac arrest, particularly in large cities. Eisenberg et al[4] have pointed out that extending CPR training to the lay public is more cost-effective than other options designed to promote rapid intervention in life-threatening emergencies. The implementation of widespread training in large urban areas such as New York City, however, is thwarted by logistical and economic deterrents imposed by the time-consuming and instructor-intensive nature of traditional "hands-on" training methods using manikins.

The effectiveness of courses that incorporate manikin practice in the curriculum has been repeatedly confirmed.[10-14] In order to accommodate the large number of citizens who needed to be trained in New York City, however, the AHA found it necessary to consider other instructional methods that did not rely on small group instruction and did not use manikins. We sought to develop an audiovisual product that could teach large numbers of people, be available in different formats, and be useful for broadcast television, home, and institutional viewing. These considerations eventually led us to produce and evaluate a film/videotape.*

The film/videotape was professionally produced by a staff that had received traditional CPR training by the AHA. A scenario was developed using four survivors of cardiopulmonary arrest and their rescuers. Dramatic vignettes in which these individuals recounted their real-life experiences were reinforced with formal, simulated, classroom instruction by a professional instructor, who demonstrated single-rescuer techniques on a manikin. The essential components of basic CPR were introduced in a stepwise, modular format. Frequent repetition was used as the key teaching technique for reinforcing essential concepts.

The objective of this study was to record, measure, and evaluate the CPR performance skills of viewers who had no previous formal CPR training, after a single viewing of the film, "CPR: It's About Time."

SUBJECTS AND METHODS

The opportunity of participating in the research study and learning single-rescuer skills was advertised to the constituents of a variety of organizations in New York City: members of a large, inner city church; staff, faculty, and students at two city colleges; employees of both a trade organization and the corporate head-

---

From the Departments of Medicine, Long Island College Hospital (Dr Schluger); The New York Hospital-Cornell Medical Center (Dr Hayes); St Luke's Roosevelt Hospital Center (Dr Turino); and New York University School of Medicine (Dr Fox); and from Hunter College (Dr Fishman).

Address correspondence to Dr Schluger, Director, Department of Internal Medicine, The Long Island College Hospital, 340 Henry St, Brooklyn, NY 11201.

Abstract presented at the American Heart Association, Scientific Sessions, November 1985.

* The film/videotape used in this study was based on CPR standards then in effect. The film has recently been modified to conform with the new AHA standards published in June 1986 (*JAMA* 1986; 255:2905).

TABLE I. Study Participants by Age and Sex

Age	
18–55 years	234 (90%)
Over 55 years	21 (8%)
Other	2 (2%)
Total	257
Sex	
Women	164 (63%)
Men	93 (36%)
Total	257

quarters of a large bank. The study was carried out in the summer and fall of 1984. Individuals who volunteered to take the course filled out a simple questionnaire. Those who had prior instruction in CPR or were physically unable to perform the skills were excluded from the study. A total of 262 individuals were selected; 63% were women and 36% men. Their ages ranged from 18 to over 70 years; 90% were in the 18–55-year age group (Table I). All participants had at least a high school diploma; 52% had achieved a bachelor's or higher postgraduate degree, and 35% had some college education (Table II). Eighteen percent were students, 28% were office personnel, 37% were at the professional or managerial level, 2% were homemakers, and the rest were engaged in a variety of occupations (Table III).

None of the participants had had any formal training in cardiopulmonary resuscitation. Forty-four percent had close friends and relatives with a history of heart attack, and there was a history of death from heart attack among close friends and relatives of 38% of the participants.

Evaluation Methods. The control group for this study was originally intended to be an untrained group of participants who did not see the film. Accordingly, a preliminary trial with a small group of untrained individuals was conducted. Those persons who were tested on the manikin without seeing the film were perplexed and could not properly position themselves, or check for breathing or pulse; not one of the six was able to perform any aspect of cardiopulmonary resuscitation. Further testing of untrained individuals was considered to be of no value to the study, unnecessary, and wasteful of subjects, and therefore was abandoned.

Viewing stations which could accommodate six people, and six individual testing stations, were set up at each location selected for the study. The participants were scheduled for viewing and testing in groups of three to six persons. After a short introductory statement prior to the viewing, the film/videotape was shown to the group. Each participant was then immediately sent for individual testing on a recording Resusci Anne manikin, out of sight of the other subjects. The performance was observed by a trained evaluator and was recorded on a Skills Assessment Sheet. In addition, recorded manikin strips were collected during each performance for later analysis. The test was terminated after four cycles of 15 compressions and two ventilations had been attempted. On completion of the first manikin test, students were immediately retested in the same manner by a different evaluator. No suggestions or comments concerning the students' performance were made by the evaluators between tests, and there was no opportunity or provision for practice or remediation. Assignments of participants to evaluators was randomized.

A total of 14 highly experienced instructors affiliated with seven different New York City medical institutions served as evaluators. They were trained in the use of the Skills Assessment Sheet prior to the start of the study. On completion of the entire study, the coded recorded strips were measured and scored in a blinded manner by six evaluators working independently.

TABLE II. Educational Status of Participants

Some high school	1 (2%)
High school diploma	29 (11%)
Some college education	90 (35%)
Bachelor's degree	70 (27%)
Master's degree or higher	67 (25%)
Total	257

TABLE III. Occupations of Participants

Professional/managerial	97 (37%)
Clerical workers	72 (28%)
Students	47 (18%)
Homemakers	6 (2%)
Miscellaneous	35 (15%)
Total	257

RESULTS

In order to allow for a brief period of accommodation, the second cycle of 15 compressions and two ventilations during the second test was selected for analysis. The data reported below are based on the 257 individuals who completed both tests. Five other individuals (a total of 262 participants) volunteered for the study but did not complete both tests. They have not been included in the results.

As observed by the evaluators, unresponsiveness of the victim was properly determined by 76% of participants, 48% correctly established the absence of breathing, and 62% opened the airway. The absence of a pulse was determined by 52%, and the proper hand position for chest compression was located by 38%. Eighty-seven percent of the participants attempted 15 compressions; 51% of the compressions were of adequate depth, and 50% of the series of 15 compressions were followed by two ventilations (Table IV). It was the assessment of the evaluators that 53% of the participants properly determined the need for cardiopulmonary resuscitation, and 63% performed the essential skills in sequence (Table V). It was the judgment of the evaluators that 43% of participants demonstrated performance skills "adequate to save a life."

Based on chi-square analysis, there were significant differences in performance based on gender ($p < 0.004$), age ($p < 0.023$), and educational level ($p < 0.0005$).

The subsets of participants that achieved the highest percentages of overall performance judged "adequate to save a life" were men (56%), those in the 18–25-year (44%) or 26–40-year (51%) age groups, and those who had bachelor's (54%) or master's (62%) degrees.

The level of agreement between evaluator and tape was measured by the Kappa statistic.[20] Good agreement (Kappa values between 0.40 and 0.75)[21] was found in documenting four quick breaths, giving 15 compressions, and ventilating twice after compression. Poor agreement (Kappa value 0.36) was demonstrated in estimating the depth of compressions.

The lower percentage (37%) of women judged to have adequate overall skills compared to men (56%) may have been influenced by factors affected by physical strength. As an example, data on 117 of 164 women participants reveal that while 85% positioned themselves properly and 86% attempted compressions, only 30% compressed the thorax to an adequate depth. On the other hand, overall performance by women may have been adversely affected by factors other than those influenced by physical strength, such as failure to make initial assessment of the need for cardiopulmonary resuscitation

TABLE IV. Observations of Participants by Evaluators

Airway	
Established unresponsiveness	196 (76%)
Opened airway	161 (62%)
Breathing	
Gave four quick breaths	103 (40%)
Compressions	
Established lack of pulse	134 (52%)
Gave 15 compressions	226 (87%)
Adequate depth of compressions	133 (51%)
Total participants	257

TABLE V. Assessment of Participants by Evaluators

Overall assessment	
Properly assessed the need for CPR	137 (53%)
Performed essential skills in sequence	164 (63%)
Performed skills "adequate to save a life"	112 (43%)
Total participants	257

(35%) and failure to perform essential skills in the correct sequence (45%).

There were 253 technically satisfactory manikin strips recorded during the second test which were suitable for analysis and measurement. A comparison of some of the data available from these tapes with the corresponding subjective evaluations of the instructors is shown in Table VI.

DISCUSSION

A recent review by Cummins and Eisenberg[19] summarizes the published data confirming that early, bystander-initiated cardiopulmonary resuscitation saves lives and decreases subsequent neurologic dysfunction. The quality or adequacy of bystander-delivered cardiopulmonary resuscitation necessary to positively influence the eventual outcome in cases of cardiac arrest, however, has not been measured or defined.[10,15] The acquisition of CPR performance skills has been shown to be influenced by the duration of the course, the use of manikins, and the participation of instructors. Although skills retention is related to the duration of the course and the time spent on manikin practice, deterioration of skills after one year to a level below American Heart Association performance standards appears unrelated to the aforementioned factors.[12] When one considers the rapid deterioration of psychomotor skills following initial CPR instruction and the failure of most trainees to take refresher courses, the quality of CPR performed by bystanders must be questioned.[6] Cobb et al,[8] in Seattle, whose pioneering mass training initiatives have spurred widespread lay training nationally, have been unable to relate patient outcome to the quality of cardiopulmonary resuscitation delivered. They and others[6,17] point out that it is impractical to expect lay citizens to be as expert as medical professionals and indicate that the quality of bystander cardiopulmonary resuscitation is less important than the time of its initiation.

The use of audiovisual techniques for CPR training without instructors or manikins has been previously evaluated as a possible mass training vehicle. Such application would have obvious advantages provided the quality of performance skills achieved was adequate. Mandel et al[18] evaluated the effectiveness of videotaped cardiopulmonary resuscitation for initial training and concluded that students performed poorly but were not dangerous to a victim when tested after viewing a test tape. Performance varied depending on which of two videotapes was used. These authors found that performance improved significantly when a videotape was used in conjunction with instructor-led classes. Cardiopulmonary resuscitation skills following a single videotape viewing were dependent on many variables, including student age, motivation, educational level, background, and the quality and format of the videotape.

TABLE VI. Evaluators versus Manikin Tapes

	Evaluator	Tape
Gave four quick breaths	103 (40%)	60 (23%)
Gave 15 compressions	226 (87%)	216 (85%)
Adequate depth of compressions	133 (51%)	94 (37%)
Ventilated twice after compressions	128 (49%)	97 (38%)
Total participants	257	

The results obtained with "CPR: It's About Time" indicate its usefulness for training lay persons in essential single-rescuer skills quickly, inexpensively, and without the use of manikins or CPR instructors. One can only speculate about the reason for the effectiveness of this film compared with previous unsuccessful attempts at cardiopulmonary resuscitation instruction employing audiovisual techniques. We believe that this professionally produced film has a high dramatic intensity, created by actual survivors of cardiopulmonary arrest and their rescuers recounting their experiences, which serves to attract and keep the attention of the viewer. Once the attention of the student has been captured, the frequent reinforcement and repetition by the on-screen instructor appears to be effective in achieving the favorable results reported herein.

The preliminary results obtained with small groups of volunteers suggest that "CPR: It's About Time" may be a valuable, inexpensive vehicle for training the lay public in the key skills of cardiopulmonary resuscitation using television broadcast or other mass viewing situations. It can be shown alone or augmented by instructional aids such as a study guide (we have subsequently prepared one to accompany the film/videotape in future use). The adjunctive use of this film/videotape in instructor-led classes and its effectiveness in retraining health professionals and laymen are areas for future investigation and possible application.

Acknowledgments. The authors are grateful to Alice E. Austin for her assistance in conducting this study, and to the many individuals who took part in developing and implementing this film/videotape and study, including the members of the Task Force on Citizen CPR, the staff of the New York Heart Association, the study participants, and the evaluators.

REFERENCES

1. Baum RS, Alvarez H 3d, Cobb LA: Survival after resuscitation from out-of-hospital ventricular fibrillation. *Circulation* 1974; 50:1231-1235.
2. Lund I, Skulberg A: Cardiopulmonary resuscitation by lay people. *Lancet* 1976; 2:702-704.
3. Copley DP, Mantle JA, Rogers WJ, et al: Improved outcome for prehospital cardiopulmonary collapse with resuscitation by bystanders. *Circulation* 1977; 56:901-905.
4. Eisenberg MS, Berger L, Hallstrom A: Cardiac resuscitation in the community. Importance of rapid provision and implications for program planning. *JAMA* 1979; 241:1905-1907.
5. Tweed WA, Bristow G, Donen N: Resuscitation from cardiac arrest. Assessment of a system providing only basic life support outside of hospital. *Can Med Assoc J* 1980; 122:297-300.
6. Guzy PM, Pearce ML, Greenfield S: The survival benefit of bystander cardiopulmonary resuscitation in a paramedic served metropolitan area. *Am J Public Health* 1983; 73:766-769.
7. Hofkin GA: Survival after cardiopulmonary resuscitation. *JAMA* 1967; 202:652-654.
8. Cobb LA, Hallstrom AP, Thompson RG, et al: Community cardiopulmonary resuscitation. *Annu Rev Med* 1980; 31:453-462.
9. Murphy RJ, Luepker RV, Jacobs DR, et al: Citizen cardiopulmonary resuscitation training and use in a metropolitan area: The Minnesota Heart Survey. *Am J Public Health* 1984; 74:513-515.

10. Wilson E, Brooks B, Tweed WA: CPR skills retention of lay basic rescuers. *Ann Emerg Med* 1983; 12:482-484.
11. Winchell SW, Safar P: Teaching and testing lay and paramedical personnel in cardiopulmonary resuscitation. *Anesth Analg* 1966; 45:441-449.
12. Vanderschmidt H, Burnap TK, Thwaites JK: Evaluation of a cardiopulmonary resuscitation course for secondary schools. *Med Care* 1975; 13:763-764.
13. Ramirez AG, Weaver FJ, Raizner AE, et al: The efficacy of lay CPR instruction: An evaluation. *Am J Public Health* 1977; 67:1093-1095.
14. Flax P, Larke T, Walser G, et al: The mechanics of widespread training of cardiopulmonary resuscitation. A community project implemented by volunteers. *Am Heart J* 1976; 91:123-125.
15. Mandel LP, Cobb LA: Initial and long-term competency of citizens trained in CPR. *Emerg Health Sys Quart* 1982; 1:49.
16. Gombeski WR, Effron DM, Ramirez AG, et al: Impact on retention: Comparison of two CPR training programs. *Am J Public Health* 1982; 72:849-852.
17. Martin WJ, Lomis JH, Lloyd CW: Cardiopulmonary resuscitation skills: Do we expect too much? [editorial]. *Arch Intern Med* 1984; 144:699-701.
18. Mandel L, Bert J, Lavert H: *Citizen training: Video CPR. Effectiveness of Videotape CPR Instruction for the Initial Training of Lay Citizens.* Federal Emergency Management Agency, 1982.
19. Cummins RO, Eisenberg MD: Prehospital cardiopulmonary resuscitation: Is it effective? *JAMA* 1985; 253:2408-2412.
20. Fleiss JL: *Statistical Methods for Rates and Proportions*, ed 2. New York, John Wiley & Sons, 1981.
21. Landis JR, Koch GG: The measurement of observer agreement for categorical data. *Biometrics* 1977; 33:159-174.

Medical students' exposure to formaldehyde in gross anatomy laboratories

JAY M. FLEISHER

ABSTRACT. This study assessed the effect of formaldehyde exposure on medical students conducting dissections in the gross anatomy laboratory course of a major medical college in the Northeast. Self-administered questionnaires were designed to assess the frequency of occurrence of various symptoms indicating the acute effects of formaldehyde exposure. The questionnaires were given to a cohort of first-year medical students on completion of the gross anatomy laboratory course. Air sampling of formaldehyde levels in the anatomy laboratories was carried out on one day during the time in which these students were conducting dissections. An identical questionnaire was given to the same cohort of students after completion of the pathology and microbiology laboratory courses, which provided no exposure to formaldehyde. The results obtained from the pathology/microbiology questionnaires were used to assess the extent of reported symptoms associated with laboratory sessions of long duration and, thus, were used to control for this factor in the anatomy laboratories. Although the results of the air sampling showed formaldehyde levels to be well below current occupational standards, significant numbers of students reported experiencing symptoms associated with formaldehyde exposure. Estimates of the relative risk of experiencing formaldehyde-related symptoms in the anatomy laboratories compared to the control laboratories ranged from 2.0 to 19.0, depending on the particular symptom. In addition, it was found that female students were three times more likely to report formaldehyde-related symptoms than male students. The results of this study indicate the need for further investigation of formaldehyde exposure in gross anatomy laboratories.

(NY State J Med 1987; 87:385-388)

A variety of acute and chronic health effects resulting from exposure to formaldehyde have been reported. Symptoms associated with acute effects of formaldehyde exposure include eye, nose, and throat irritation; sneezing; shortness of breath; tightness in the chest; nausea; and excess phlegm production. Exposure to concentrations ranging from 0.01 to 5 ppm can cause eye irritation and irritation of the upper respiratory tract. Exposure to higher concentrations can produce coughing, tightness in the chest, headache, nausea, heart palpitations, and sleeplessness. Chronic effects of continued exposure to formaldehyde include chronic airway obstruction, dermatitis, asthma, bronchitis, rhinitis, pharyngitis, menstrual disorders, reproductive disorders, and possibly cancer.[1-4] The current standards for occupational exposure to formaldehyde are 3 ppm for an eight-hour time-weighted average, a 5 ppm acceptable ceiling, and a 10 ppm maximum 30-minute ceiling.[5]

Although the health effects of formaldehyde exposure have been studied in the occupational setting, recent interest has developed in the possible health effects of formaldehyde exposure in the gross anatomy laboratories of medical schools.[6-9] Human cadavers are preserved in a formalin solution, with phenols added as a bleach and germicide.[10,11] Medical students and faculty are therefore exposed to varying concentrations of formaldehyde during dissections.

This study was conducted at a major US medical school which has six gross anatomy laboratories. Each laboratory accommodates between six and ten cadavers, on which teams of first-year medical students (approximately four to a team) conduct dissections. During the fall 1983 semester, the State Division of Safety and Health was asked to monitor formaldehyde levels in all six gross anatomy laboratories. Subsequent to the air sampling, the entire class of first-year medical students who had been conduct-

---

Address correspondence to Mr Fleisher, Assistant Professor, Department of Preventive Medicine and Community Health, SUNY Health Science Center at Brooklyn, Box 43, 450 Clarkson Ave, Brooklyn, NY 11203.

This study was conducted while the author was a graduate student at Columbia University School of Public Health, New York, NY.

ing dissections during this time period was surveyed with regard to possible formaldehyde-related symptoms. The survey was conducted approximately one month after the students completed the gross anatomy course. The survey was designed to provide an estimate of the extent of formaldehyde-related symptoms suffered by the students during dissections. The results of both the air sampling and symptom survey are presented.

SUBJECTS AND METHODS

A self-administered questionnaire was provided to each first-year medical student to determine whether the students had experienced any of a variety of symptoms representative of the acute effects of formaldehyde exposure. The specific symptoms listed included eye irritation, nose irritation, sneezing, sleep disorders, tightness in the chest, headache, rash, itch, shortness of breath, nausea, cough, throat irritation, sinus problems, and heart palpitations. Students were asked to indicate the frequency with which each of the symptoms had occurred: all of the time; some of the time; rarely; or never. In addition, students were asked to indicate their names, sex, the average length of each laboratory session attended (0–1 hour; 1–2 hours; 2–3 hours; 3–4 hours; more than 4 hours), and the percentage of laboratory sessions they had attended during the semester (0–25%; 25–50%; 50–75%; 75–100%).

An identical questionnaire was administered to the same cohort of students after completion of the pathology and microbiology laboratory courses approximately six months later. The pathology and microbiology laboratory sessions combined comprise approximately the same number of hours per session as the gross anatomy laboratory course. Since the length of the laboratory sessions was similar, and the pathology and microbiology laboratories provided no exposure to formaldehyde, the latter were used as controls to establish a base-line of symptoms due to factors other than formaldehyde exposure. Students were asked to indicate their names on both questionnaires so that paired comparisons could be carried out on the responses of individuals completing both questionnaires. The questionnaires were placed in the students' mailboxes and the students were asked to return the completed questionnaires in an attached, pre-addressed envelope via intra-office mail.

Air quality measurements were carried out in each gross anatomy laboratory on one day during the semester to which the student questionnaires pertained. Three different methods were used for formaldehyde sampling. Direct-reading Drager tubes were used in laboratories 1 through 6. The range of measurement of this method is from 1 to 40 ppm. The National Institute for Occupational Safety and Health (NIOSH) method, P. CAM (125), 1% sodium bisulfite solution with single impinger, was used in three of the six anatomy laboratories. The range of measurement of this method is from 0.02 to 10 ppm. In order to evaluate personal breathing zone levels of formaldehyde, 3M Diffusional Monitors were placed on two instructors.

TABLE I. Air Quality Sampling Results

Laboratory	Formaldehyde Concentration in Parts per Million* (Length of Sample in Minutes): Direct Reading Drager-Tube Method†	NIOSH Method	3M Diffusional Monitors
1	<1	0.59 (222)	0.69 (180)
2	<1	0.03 (188)	—
3	<1	—	—
4	<1	—	—
5	<1	—	—
6	<1	0.02 (190)	0.18 (190)

* The maximum permissible eight-hour time weighted average concentration is 3 ppm under current standards governing occupational exposure to formaldehyde vapors.

† General area samples.

TABLE II. Distribution of Reported Formaldehyde-Related Symptoms in the Gross Anatomy Laboratories

Symptom	Percent Responding "Always" or "Sometimes": All Students (N = 89)	Men (N = 63)	Women (N = 26)
Eye irritation	72.7	66.1	88.5
Nose irritation	64.7	60.0	76.0
Sneezing	35.3	31.1	45.8
Sleep disorders	11.9	8.3	20.8
Tightness in chest	10.8	6.8	20.8
Headache	62.3	52.5	88.5
Rash	23.5	18.3	36.0
Itch	25.0	16.9	44.0
Shortness of breath	8.3	5.0	16.7
Nausea	23.8	15.0	45.8
Cough	25.0	18.0	43.5
Throat irritation	32.9	27.9	45.8
Sinus problems	28.6	23.3	41.7
Heart palpitations	2.5	1.7	16.7

Statistical Methods. The data were analyzed using two approaches. In the first approach, the four possible responses to each symptom were combined into two categories indicating the presence ("always" or "sometimes") or absence ("rarely" or "never") of symptoms. These two groupings were then used to derive various estimates of relative risk. The other approach was to assign a numerical value to each of the four possible responses, as follows: always = 1; sometimes = 2; rarely = 3; and never = 4. In this way an index of discomfort could be calculated for each respondent by summing these values over all symptoms, with lower scores indicating greater discomfort.

For the anatomy laboratory questionnaire, the presence of symptoms was examined for all respondents and for male and female respondents separately. Estimates of the relative risk of each symptom for women as compared to men were computed and tested for significance using the Pearson chi-square statistic. A t-test for the difference between mean discomfort scores over all symptoms for men versus women was also computed. A two-way analysis of variance procedure was carried out using the discomfort score as the outcome variable, and gender and average number of hours per laboratory session as the independent variables. This was done to control for differential attendance patterns between men and women. Since different cadavers could contain different levels of formaldehyde, the laboratory and bench assignments for all respondents were obtained and inspected for any signs of clustering within particular laboratories.

In order to assess symptoms caused by factors other than formaldehyde exposure, discomfort scores derived from the pathology/microbiology laboratory questionnaire were compared to those obtained from the anatomy laboratory questionnaire. T-test procedures were used to test whether mean discomfort scores in the anatomy laboratory were significantly different from

TABLE III. Two-Way Analysis of Variance Table of Discomfort Scores Using Gender and Average Number of Hours Spent at Each Anatomy Laboratory Session as Main Effects

Source	DF	Sums of Squares	Mean Square	F
Gender	1	600.8919	600.8919	11.15*
Hours†	2	33.9486	16.9743	0.31‡
Errors	74	3987.8774	53.8902	

* Indicates significance at $p < 0.01$.

† No respondent indicated spending less than two hours per laboratory session.

‡ Not significant.

TABLE IV. Estimates of Relative Risk to Women Over Men of Experiencing Formaldehyde-Related Symptoms in the Gross Anatomy Laboratories* (N = 89)

Symptom	Relative Risk	P-Value
Eye irritation	3.93	0.0319
Nose irritation	2.11	0.1596
Sneezing	1.87	0.2022
Sleep disorders	2.89	0.1100
Tightness in chest	3.62	0.0619
Headache	6.95	0.0014
Rash	2.51	0.0802
Itch	3.85	0.0088
Shortness of breath	3.80	0.0805
Nausea	4.97	0.0027
Cough	3.50	0.0163
Throat irritation	2.19	0.1127
Sinus problems	2.35	0.0929
Heart palpitations	2.43	0.5229

* Mantel-Haenszel Summary Odds Ratio = 3.00 ($p < 0.0001$).

mean discomfort scores in the pathology/microbiology laboratories, and whether sex-specific estimates of mean discomfort scores differed significantly in the pathology/microbiology laboratories. Estimates of the relative risk of experiencing each formaldehyde-related symptom in the anatomy laboratories relative to the pathology/microbiology laboratories were computed. All interlaboratory comparisons were analyzed twice. One set of analyses, appropriate for paired comparisons, was performed using the subset of students that responded to both questionnaires. The larger series of all responses to the anatomy and pathology/microbiology questionnaires was treated as two independent samples. This enabled all responses to each questionnaire to be included in the analysis. Analysis of the paired and unpaired data showed similar results. Only the results of the analysis of the paired data are presented. T-test procedures appropriate for paired samples and McNemar's test of symmetry were used to determine the statistical significance of the paired interlaboratory comparisons.

RESULTS

The results of the air sampling are shown in Table I. All readings are well below current occupational standards. The last two columns, however, show levels of formaldehyde sufficient to cause symptoms.[4]

Of the 204 first-year students surveyed, 89 completed the questionnaire pertaining to the anatomy laboratory, while 60 completed the questionnaire pertaining to the pathology/microbiology laboratories. Seventy-one percent of the students who completed the anatomy laboratory questionnaire were men and 29% were women. Seventy-two percent of the students who completed the pathology/microbiology laboratory questionnaire were men and 28% were women. Thus, the response rate for men and women was consistent for both questionnaires.

Table II shows the distribution of reported formaldehyde-related symptoms in the gross anatomy laboratories for all students combined and for men and women. A high proportion of all students suffered formaldehyde-related symptoms, and there was a clear excess of symptoms among female respondents. The results of the t-test for the difference between men and women in mean discomfort scores over all symptoms in the anatomy laboratories was significant at $p < 0.01$ (the mean discomfort score for women was 2.85 as compared to 3.29 for men). This indicated that, on average, female respondents reported more discomfort than males. Table III shows the results of the two-way analysis of variance procedure used to control for possible confounding due to different attendance patterns between men and women in the gross anatomy laboratories. There was no difference in discomfort scores by the number of hours spent per laboratory session. Since 98.9% of respondents attended between 75% and 100% of the anatomy laboratory sessions, the number of laboratory sessions attended was not included in the analysis of variance procedure. Table IV shows the estimates of relative risk for all formaldehyde-related symptoms for women as compared to men, as well as the Mantel-Haenszel summary odds ratio. Table V contrasts the overall and sex-specific distribution of symptoms experienced in the anatomy laboratories with the distribution of symptoms experienced in the pathology/microbiology laboratories. The table shows a striking decrease in the frequency of reported symptoms originating in the pathology/microbiology laboratories.

The results of the paired t-test showed higher levels of discomfort in the anatomy laboratories ($p < 0.00001$). Sex-specific mean discomfort scores for men versus women in the pathology/microbiology laboratories were not statistically significant ($p = 0.61$). Table VI shows the estimates of relative risk of formaldehyde-related symptoms between the anatomy and pathology/microbiology laboratories for both sexes combined.

TABLE V. Percent of Students Who Responded to Both Questionnaires Reporting Symptoms* in the Gross Anatomy Laboratories versus the Pathology/Microbiology Laboratories

	All Students (N=38)		Men (N=26)		Women (N=12)	
Symptoms	Anatomy	Path/Micro	Anatomy	Path/Micro	Anatomy	Path/Micro
Eye irritation	68.4	21.0	57.7	19.2	91.7	25.0
Nose irritation	61.1	13.1	50.0	11.5	83.3	16.7
Sneezing	37.8	15.8	36.0	15.4	41.7	16.7
Sleep disorders	11.1	5.4	8.3	8.0	16.7	0.0
Tightness in chest	11.1	2.6	8.3	3.8	16.7	0.0
Headache	64.9	31.5	60.0	38.5	75.0	16.7
Rash	19.4	0.0	16.7	0.0	25.0	0.0
Itch	19.4	0.0	12.5	0.0	33.3	0.0
Shortness of breath	8.3	0.0	8.3	0.0	8.3	0.0
Nausea	27.8	7.9	16.7	7.7	50.0	8.3
Cough	28.6	5.3	20.8	7.7	45.4	0.0
Throat irritation	38.9	7.9	29.2	11.5	58.3	0.0
Sinus problems	35.1	5.3	28.0	7.7	50.0	0.0
Heart palpitations	5.7	0.0	4.3	0.0	8.3	0.0

* Indicated by a response of "Always" or "Sometimes."

DISCUSSION

A significant number of medical students reported suffering formaldehyde-related symptoms in the gross anatomy laboratories. The acute symptoms suffered were indic-

TABLE VI. Combined (Men and Women) Estimates of Relative Risk of Formaldehyde-Related Symptoms Between the Anatomy and Pathology/Microbiology Laboratories for Students Responding to Both Questionnaires (N = 38)

Symptom	Relative Risk	P-Value
Eye irritation	19.0	0.0001
Nose irritation	18.0	0.0001
Sneezing	9.0	0.0114
Sleep disorders	2.0	0.4142
Tightness in chest	4.0	0.1797
Headache	7.0	0.0027
Rash	—*	—
Itch	—	—
Shortness of breath	—	—
Nausea	4.5	0.0348
Cough	9.0	0.0114
Throat irritation	6.5	0.0045
Sinus problems	7.0	0.0027
Heart palpitations	—	—

* Missing estimates of relative risk indicate that no respondent indicated that this symptom was experienced in the pathology/microbiology laboratories. Relative risk could not be computed.

ative of exposure to low and moderate levels of formaldehyde vapors. Although formaldehyde levels in the anatomy laboratories measured well below current occupational standards, the type and frequency of reported symptoms suggest significant exposure to formaldehyde. Possibly, ambient air samples taken over short periods of time are not valid indicators of overall formaldehyde exposure in gross anatomy laboratories. The contrast between the frequencies of reported symptoms in the anatomy versus the pathology/microbiology laboratories suggests that the excess number of symptoms reported in the anatomy laboratory was not a function of fatigue or other factors associated with laboratory sessions of long duration.

Differential attendance patterns between men and women or interlaboratory or intercadaver differences in formaldehyde levels did not account for the higher levels of discomfort reported by women. Whatever the underlying mechanism, the observed sex differences in sensitivity should be taken into account in studies dealing with the acute effects of formaldehyde and possibly other environmental contaminants. Greater sensitivity in women would, of course, be important in setting occupational or environmental standards governing formaldehyde exposure.

This study has several limitations, specifically low response rates, self-reporting of symptoms, and indirect measurements of exposure. Nevertheless, the large numbers of medical students who reported symptoms of formaldehyde exposure in the gross anatomy laboratories suggest that a problem exists. Even if it is assumed that all nonrespondents had no symptoms, 29% of all students surveyed would have experienced the three most frequently reported formaldehyde-related symptoms. How widespread such exposure is at other medical schools and the long-term effects of such exposure remain unknown. Further investigations are needed into formaldehyde exposure in gross anatomy laboratories and into the possible increased sensitivity to formaldehyde exposure among women.

REFERENCES

1. Griesemer RA, et al: Report of the federal panel on formaldehyde. *Environ Health Perspect* 1982; 43:139–168.
2. Weber-Tschopp A, Fischer T, Grandjean E: Irritating effects of formaldehyde on men. *Int Arch Occup Environ Health* 1977; 39:207–218.
3. Kerfoot EJ, Mooney TF: Formaldehyde and paraformaldehyde study in funeral homes. *Am Ind Hyg Assoc J* 1975; 36:533–537.
4. Harris JC, Rumack BH, Aldrich FD: Toxicology of urea formaldehyde and polyurethane foam insulation. *JAMA* 1981; 245:243-246.
5. NIOSH recommendations for occupational safety and health standards. *MMWR* 1985; 34 (1S):1–31 (suppl).
6. Formaldehyde exposure in a gross anatomy laboratory—Colorado. *MMWR* 1983; 31:698–700.
7. Howitt LY: Beware formaldehyde [letter]. *New Physician* 1983; 32:4.
8. Haglund K: The occupational hazards of a medical education. *New Physician* 1982; 31:18–22.
9. Smalky K, Schor E: Environmental hazard: Gross anatomy [letter]. *N Engl J Med* 1984; 310:531-532.
10. Spriggs AO: *The Art and Science of Embalming*. Springfield, Ill, Champion Publishing, 1971.
11. Williams TM, Levine RJ, Blunden PB: Exposure of embalmers to formaldehyde and other chemicals. *Am Ind Hyg Assoc J* 1984; 45:172–176.

REVIEW ARTICLE

Current management of tuberculosis

ALJE VENNEMA, MD

The article in this issue of the *Journal* on tuberculosis mimicking acute pyogenic bacterial pneumonia by Padmanabhan et al[1] is timely, because tuberculosis is often not considered in the differential diagnosis of pneumonia. A roentgenographic picture of pneumonia compatible with tuberculosis clearly warrants further investigation.

Treatment of tuberculosis can be divided into first-line and second-line drugs. The former are mostly bactericidal and extremely effective, while the latter are both bactericidal and bacteristatic. The bactericidal drugs include streptomycin, isoniazid, rifampin, and pyrazinamide. The second-line bactericidal and bacteristatic drugs are less effective, not as potent, produce more side effects than first-line drugs, and should only be used when there is evidence of resistant organisms, in retreatment situations, and when side effects develop. Among the second-line drugs are para-aminosalicylic acid, cycloserine, ethambutol, ethionamide, capreomycin, viomycin, kanamycin, thiacetazone, and amikacin.

With proper treatment, practically all patients can be cured. Good treatment requires proper diagnosis—confirmed by culture—and proper species identification. In addition, follow-up culture of sputum on a monthly basis until three consecutive negative cultures have been obtained is necessary to ensure cure. Drug sensitivity studies are not indicated in previously untreated cases. However, they are indicated in patients who were previously treated, patients suspected of having contracted tuberculosis from someone with resistant bacilli (primary resistance), and patients suspected of having disease caused by atypical mycobacteria. Second-line drugs are more difficult to use and should only be employed by an experienced practitioner when indicated. Prior to the commencement of treatment, a minimum of three induced sputum samples, and preferably six, should be obtained for culture. When sputum smears are negative and tuberculosis is suspected, more cultures are indicated. On obtaining these specimens, treatment should be instituted if the smears are positive, or if tuberculosis is suspected to be the cause of the illness. When culture results are available and are negative, treatment can be reassessed. If other mycobacteria are cultured, treatment should be adjusted according to species identification. Other principles of treatment include the following: The number of drugs to be given should be contingent on the bacillary load; the highest possible dosage of medications should be prescribed unless contraindicated (eg, in patients with liver disease); the initial phase of treatment (usually two months) is to be intense, with three or four medications used to quickly kill off as many bacilli as possible; no single medication should ever be used for the treatment of tuberculosis; a single drug is only to be used for preventive treatment; patients should receive treatment on a daily basis, unless there are compelling reasons to do otherwise; drugs should be given prior to meals, ie, on an empty stomach; and no single drug should be added to a failing regimen.[2]

If treatment is properly instituted and the patient complies, the patient's condition should improve. The treatment regimen and duration depend on the type of problem that has been diagnosed, the patient's immunocompetence, the bacillary load, and compliance with treatment. It is advisable to employ the classifications of the American Thoracic Society in determining the proper treatment protocol.

TREATMENT BY DIAGNOSTIC CLASS

Class O—No history of tuberculosis exposure, not infected. This class refers to people who need clearance for tuberculosis as a job requirement. They should receive a Mantoux purified protein derivative (PPD) test, which is read as negative. Hence they are class O; no treatment and no radiograph is indicated.

Class I—Tuberculosis exposure, no evidence of infection. These are patients who have been exposed to tuberculosis, but who test negative with the Mantoux PPD. They are treated with isoniazid (10 mg/kg) for three months and are tested again. No radiograph is indicated. If at three months their test results are significant (greater than 5 mm of induration), a chest radiograph is taken. If the chest film is negative, they continue to receive isoniazid for a total period of 6–12 months. If there is evidence of clinical disease on the radiograph, the patients will undergo sputum examination and should be treated as class III (see below). The duration of treatment for infection was recently changed from 12 to six months.[3]

Class II—Tuberculosis infection without disease. These are patients who have been exposed to tuberculosis and whose Mantoux PPD test results are significant, but whose chest films are negative. If these patients had recent

---

Address correspondence to Dr Vennema, Director, Bureau of Tuberculosis Control, New York City Department of Health, 125 Worth St, New York, NY 10013.

contact with a known case of tuberculosis, they should be treated with isoniazid for a minimum of 6–12 months. Patients who have not had recent contact but have a risk factor such as immunosuppression, diabetes mellitus, gastrectomy, a positive serologic test for human immunodeficiency virus (HIV), substance abuse, malignancies, chronic renal disease, or malnutrition, or who are on steroid therapy, should also receive a minimum of six months of treatment with isoniazid. Patients under 35 years of age should be monitored for isoniazid toxicity on a clinical basis. Those over 35 years of age should have liver function studies done prior to treatment and when indicated during treatment.

Class III—Current disease with positive cultures for *Mycobacterium tuberculosis.* In some cases cultures are negative, but there exists a clinical condition compatible with tuberculosis and a radiograph suggestive of tuberculosis. Standard treatment for patients in class III consists of isoniazid, 10 mg/kg, up to a maximum of 300 mg daily, and rifampin, 10 mg/kg, up to a maximum of 600 mg for nine months. This is a highly successful regimen if there is good compliance and if the organisms are sensitive. Pyrazinamide, 15–30 mg/kg, and/or streptomycin, 15–20 mg/kg, can be added in the initial phase (first two months) of treatment. Alternatively, patients can be treated daily for a period of six months, with three drugs used in the initial phase (two months): isoniazid, 10 mg/kg; rifampin, 10 mg/kg; and pyrazinamide, 15–30 mg/kg. Ethambutol, 15–25 mg/kg, should be added in the initial phase if isoniazid resistance is suspected. If the six-month regimen is selected, streptomycin and ethambutol should not be substituted for pyrazinamide. In the continuation phase of four months, patients should be treated with isoniazid and rifampin only, and during the initial period, monthly sputum samples should be obtained to monitor conversion. Lack of conversion after three months indicates the need for sensitivity studies and modification of the treatment according to sensitivity results. No single new drug should be added to a failing regimen. Two new drugs must always be added. If the cultures are sensitive to isoniazid and rifampin, the total treatment period should be extended by at least six months beyond the time of conversion.

Extrapulmonary tuberculosis should be treated with the same medication as pulmonary tuberculosis. Children should be treated in the same manner as adults. Pyrazinamide, 15–30 mg/kg, may be used for children when indicated for the treatment of meningitis.

Regimens shown to be effective when rifampin cannot be used for reasons of side effects or resistance include the following:

- isoniazid, 10 mg/kg, up to a maximum of 300 mg daily, plus ethambutol, 25 mg/kg daily, for a duration of 18 months, with one to two months of streptomycin, 15–20 mg/kg, initially when the bacterial population is large;
- isoniazid, 10 mg/kg, up to a maximum of 300 mg daily, plus streptomycin, 15–20 mg/kg, up to a maximum of 1 g intramuscularly daily for a duration of 18 months.

Regimens shown to be effective when isoniazid cannot be used include the following:

- rifampin, 10 mg/kg, up to a maximum of 600 mg, plus ethambutol, 25 mg/kg, for a duration of 18 months;
- rifampin, 10 mg/kg, pyrazinamide, 15–30 mg/kg, and ethambutol, 15–25 mg/kg, for two months, followed by ten months of rifampin and ethambutol;
- if pyrazinamide cannot be used, nine months of isoniazid, 10 mg/kg, and rifampin, 10 mg/kg, may be given.

After having been properly diagnosed, patients who have been treated previously or who come from areas where primary resistance is a problem should be started on isoniazid, 10 mg/kg, rifampin, 10 mg/kg, and ethambutol, 15–25 mg/kg, until the results of sensitivity tests are known. Most treatment problems stem from a poor understanding on the part of the patient of his or her condition and from improper education concerning the illness.

Class IV—Old healed disease. Abnormal, stable radiographic findings in persons who have a significant reaction to the Mantoux PPD test or who have a history of tuberculosis. If patients have a history of a complete course of therapy and have no recurrence of symptoms, they should not be started on therapy again. If they have not had previous therapy and are clinically well, but are considered to have fibrocalcific nodules or fibrosis, they should be treated for a year with isoniazid and followed accordingly (see class II). If patients are clinically ill and have had no previous treatment, they should be considered as class V (see below) and treated accordingly.

Class V—Suspects. These patients are suspected to have tuberculosis. The diagnosis is pending until the diagnostic work-up—history, physical examination, chest film, skin test, and induced sputum cultures—to rule out tuberculosis has been completed (class III). When a determination has been made, appropriate follow-up should be instituted. Patients should not remain in this class for more than three months. If a suspect patient has a history of previous treatment for tuberculosis, has been exposed to a case of resistant tuberculosis, or comes from a country with a high prevalence of resistance, he or she should undergo sensitivity studies.

REFERENCES

1. Padmanabhan K, Dhar SR, Bhatt B: Tuberculosis mimicking acute pyogenic bacterial pneumonia. *NY State J Med* 1987; 87:404-406.
2. Treatment of tuberculosis and other mycobacterial diseases, Joint Statement of the American Thoracic Society and the Centers for Disease Control. *Am Rev Respir Dis* 1983; 127:790-796.
3. Treatment of tuberculosis and tuberculous infection in adults and children. *Am Rev Respir Dis* 1986; 134:355-363.

Adult measles

CASE PRESENTATION

Jasminder Mand, MD (Medical Intern): A 41-year-old man was admitted to the hospital with fever of five days' duration and a skin rash of three days' duration.

Three months prior to admission, the patient had travelled to Japan and to California but had remained well in the interval since then. Three weeks before admission he had hiked in the Hudson River Valley, and vaguely recalled a bite on one finger, presumably by an insect, but without local inflammation. Seven days before admission, while in otherwise good health, headache, mild photophobia, generalized myalgia, and nasal congestion developed. Two days later, the patient experienced lethargy, a sore throat, a dry cough, chills, and fever to 104° F. His appetite, however, remained good. Three days before admission, he noticed an erythematous nonpruritic rash over the face. During the next two days, the rash spread to the chest and upper extremities. The fever persisted despite aspirin and acetaminophen. After being evaluated in the emergency department, the patient was admitted.

The patient denied recent vaccinations or contact with anyone with a similar illness, but he was uncertain about his own history of childhood illnesses and immunizations. A close friend had had "CMV infection" but had not been seen by the patient during the past year. The patient's family history and medical history were noncontributory. The review of systems was also negative. The patient was unmarried, lived alone, took no medications (except during the present illness), denied allergies and tobacco use, and drank alcohol only on social occasions.

On admission, the patient was well developed, well nourished, and in mild discomfort. Physical examination disclosed the following: blood pressure, 120/80 mm Hg; pulse rate, 84/min, respiratory rate, 18/min; and temperature, 102.4°F. Immediately evident was an erythematous maculopapular rash, confluent over the face, neck, and scalp, with more discrete lesions over the trunk, and sparsely scattered lesions over the arms, palms, lower half of the abdomen, lower extremities, and soles of the feet. Small areas of erythema were present on the oral mucosa near the upper molars, but were not interpreted as Koplik spots; no exudates were identified. Ear examination was negative. There was slight, soft, tender enlargement of the anterior cervical lymph nodes, but no other adenopathy. The neck was supple, and there was no increase in jugular venous pressure. The cardiac sounds were normal, without murmurs, and the lungs were clear to percussion and auscultation. Examination of the abdomen, back, and genitalia was normal. Rectal examination was deferred. The peripheral pulses were easily felt; the extremities otherwise disclosed no abnormalities. The findings on neurologic examination were within normal limits.

The hemoglobin was 11.2 g/dL; hematocrit, 35%; white blood cell count, 2,900/μL, with 30% mature polymorphonuclear leukocytes, 59% immature forms, 10% lymphocytes, and 1% monocytes. The platelet count was 153,000/μL; the erythrocyte sedimentation rate (ESR) was 6 mm/h. During the following two days, the only hematologic changes were a reduction in immature polymorphonuclear forms to 8% and an increase in the lymphocyte count to 52%.

The blood glucose level was 145 mg/dL; serum levels of electrolytes, calcium, phosphate, alkaline phosphatase, total protein, albumin/globulin ratio, serum bilirubin, serum amylase, and gamma glutamyl transpeptidase were all within normal limits.

Four blood cultures showed no growth. The Monospot® test for infectious mononucleosis and the VDRL test for syphilis were negative. Viral serologic tests for measles (rubeola) by the ELISA method were reported as 0.08, indicating no detectable antibody; serum test for rubella antibody detected a high level of antibody, greater than 6.5 ELISA index. Antibody titers were positive in 1:16 dilution for Rocky Mountain spotted fever and were positive in 1:128 dilution for murine typhus.

Initial treatment included intravenous hydration, acetaminophen for fever, and tetracycline, 500 mg orally every six hours. The patient remained comfortable with a good appetite.

Within a few hours of admission, the skin rash spread more extensively over the abdomen. Several hours later, the patient was transferred to a nursing area that permitted better respiratory isolation. During the next two days, his temperature slowly returned to normal; the rash gradually progressed caudally, but with resolution of the facial lesions and of the cervical lymphadenopathy. By the fourth day of hospitalization, the fever and skin rash had completely resolved; tetracycline therapy was discontinued, and the patient was discharged.

RADIOLOGIC PRESENTATION

George Braff, MD (Chief of Radiology): The chest film taken on the day of admission shows a normal cardiac sil-

---

Based on a clinical conference held at the New York Infirmary, Beekman Downtown Hospital, New York City, on August 1, 1986.

Editor: JOHN T. FLYNN, MD.

Address correspondence to Dr Flynn, Consulting Editor, *New York State Journal of Medicine*, PO Box 5404, 420 Lakeville Rd, Lake Success, NY 11042.

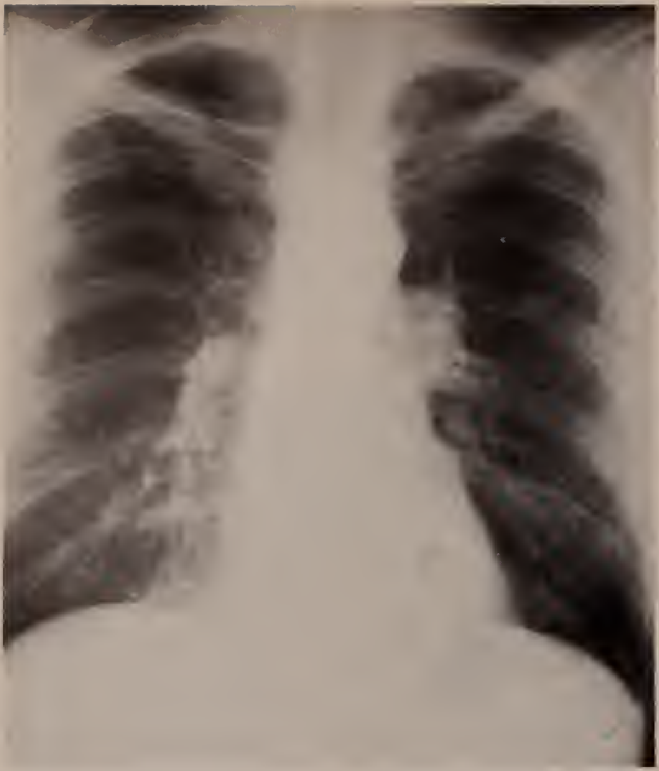


FIGURE 1. Postero-anterior chest film taken on admission showing increased fullness in the hilar regions, probably representing vascular shadows.

houette and clear lung fields (Figs 1, 2). There is a fullness to the hilar regions, suggesting either lymphadenopathy or prominent hilar vessels. It would be helpful to have earlier films for comparison. In their absence, I feel that these changes represent prominent hilar vessels.

In children with measles, an interstitial pulmonary infiltrate can develop, together with hilar adenopathy and perihilar interstitial disease. In adults, hilar adenopathy does not develop; the findings are confined to mixed interstitial patterns—perihilar and at the lung bases, or, rarely, extensive reticulonodular disease.

John T. Flynn, MD (Associate Chief of Medicine): Did this patient have any immunizations immediately prior to his trip to Japan?

James Malcolmson, MD (Medical Resident): No.

Dr Flynn: On admission here, was his sore throat impressive enough to suggest the need for throat cultures?

Dr Malcolmson: The patient said he had been coughing so much that as a result his throat was slightly sore.

Naveen Rao, MD (Medical Attending): Deferral of the rectal examination at the time of admission might lead you to overlook a primary lesion of syphilis.

Dr Malcolmson: We did inspect the anal area for any evidence of an inflammation or a chancre.

Hamid Sadaghdar, MD (Medical Resident): How sure can we be that the tetracycline helped to reduce his fever over the three or four days in the hospital?

Dr Malcolmson: We cannot be sure. He was receiving other antipyretics as well.

The Differential Diagnosis

Dr Malcolmson: In this patient, the most striking presentation was that of an acute exanthem. While the skin rash was obvious, its differential diagnosis in an adult is not simple.

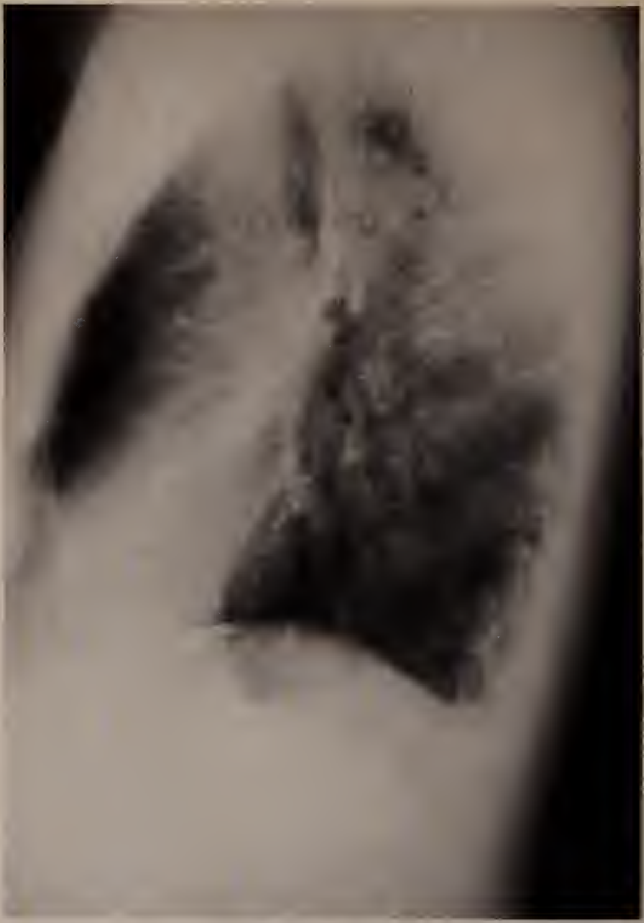


FIGURE 2. Lateral chest film showing linear markings in lower lung fields.

A feature common to exanthems is fever, which in this patient was present for three days prior to his admission. Other important clinical signs and symptoms were general myalgia, nasal congestion, and conjunctivitis—all nonspecific but important clues to the viral exanthems.

Common noninfectious causes of acute exanthematous reactions are drugs. The only medications that the patient had used were antipyretics, which were started after the onset of the fever, when the rash was already beginning. He had had no history of previous allergic reactions. Eosinophilia is often seen with drug eruptions, but this was not present in this case.

The history of a questionable insect bite during a hike in the Hudson River Valley three weeks prior to admission suggested to a physician at another hospital the possibility of Rocky Mountain spotted fever. This infection was originally encountered in the mountain states of Montana and Idaho, but is now more prevalent in the South Atlantic states than in the West. In the eastern United States, the condition is centered in Cape Cod and some adjacent islands, with foci on Long Island and in Florida. Almost half of the cases in the United States occur in Maryland, Virginia, North Carolina, and Georgia.[1-3]

This disease is also known as spotted fever and tick typhus, and is a mild to severe—sometimes fatal—acute infectious disease. It is endemic in the Western hemisphere, is caused by *Rickettsia rickettsii*, and is transmitted to man by several species of ticks. Patients experience the sudden onset of chills, headache, and fever of two to three days' duration. The rash appears on about the fourth day of the illness, first about the wrists and ankles, and rapidly spreading to all parts of the body, including the palms, soles, face, and occasionally the mucous membranes of the mouth and throat. At first the lesions are pink macules 2–5 mm in diameter, which blanch on pressure; in two to three days, they become fixed, darker red or purplish maculopapules. On about the fourth day, petechial hemorrhagic lesions may appear and coalesce. The rash begins to disappear as the fever subsides, but often leaves behind pigmented spots for many weeks.

In the patient under discussion, the rash followed the fever by only two days, and began on the face, did not become hemorrhagic, and was not followed by local pigmentation.

The rapid, severe course of Rocky Mountain spotted fever in some patients makes it imperative to regard any suspected case as a medical emergency. In untreated cases, the overall fatality rate is about 20%. The prognosis depends on the severity of the infection, the age of the patient, the presence of other diseases, and the interval between onset and the initiation of specific antirickettsial therapy. Prompt administration of tetracycline is the single most important therapeutic measure. Because specific etiologic diagnosis may be impossible during the first few days of the disease, any patient seriously considered to have Rocky Mountain spotted fever should be treated as such, while other diagnostic procedures are undertaken. This was one of the major reasons why this patient was admitted to the hospital and treated with tetracycline on the night of admission.

The two diseases that consistently cause the greatest difficulty in the diagnosis of exanthems are measles and meningococcemia. The lesions of meningococcemia can be maculopapular, but often are purpuric, which was not a finding in this patient. This patient's rash was maculopapular and even morbilliform, that is, measles-like. There was also a lack of meningeal signs. Blood cultures taken before antibiotic therapy was started were sterile.

Rubella, or German measles, is characterized by a three-day rash, generalized lymphadenopathy, and minimal or absent prodromal symptoms. The exanthem of rubella is usually apparent within 24 hours of the first symptoms as a faint macular erythema, which first involves the face and neck. It is frequently evanescent and tends to spread rapidly to the trunk and extremities, sometimes clearing at one site while reappearing at another. The pink macules that constitute the rash blanch with pressure and rarely stain the skin. The truncal rash may coalesce, but the lesions on the extremities remain discrete. The eruption vanishes by the third day. Rubella can be clinically diagnosed with assurance only during an epidemic. Distinction from measles may be made on the basis of fainter, nonstaining rash, a milder clinical course, and minimal or absent systemic complaints.

Another possibility on the list of morbilliform rashes is exanthem subitum, also known as roseola infantum, a rather common diagnosis in pediatric clinical practice. Rotavirus has been implicated as the etiologic agent. The white blood cell count is usually elevated initially; as the disease progresses, leukopenia develops, as was true in this patient. However, the rash is evanescent and characteristically of brief duration.

Other infections that must be considered are those due to echoviruses, a separate genus within the enterovirus family. The characteristics of the echovirus exanthems are not sufficiently distinctive for the establishment of an etiologic diagnosis on clinical grounds alone. The most common clinical presentation is a morbilliform rash, which is most frequently caused by echovirus IX and consists of fine discrete macules and/or papules primarily on the face and neck and, to a lesser extent, on the chest and extremities. This presentation is commonly confused with that of rubella, although posterior cervical and auricular lymphadenopathy are generally not present. Although minimal prodromata, lack of posterior cervical and auricular lymphadenopathy, and the distribution of the rash in the patient being discussed here could be consistent with echoviral infection, the timing of the fever and the overall presentation of the rash were not consistent with such a diagnosis.

Another condition that must be considered in the differential diagnosis is scarlet fever. This condition may present with a morbilliform rash, but in this patient, the absence of certain symptoms, such as sore throat, vomiting, classic "strawberry tongue," and tonsillitis, tended to rule out this possibility.

Infectious mononucleosis also can be associated with a maculopapular or occasionally petechial rash in less than 50% of cases; fever, lymphadenopathy, myalgias, photophobia, and sore throat are frequently present. Splenomegaly is present in 50% of cases. The absence of most of these symptoms and signs, together with a negative heterophil agglutination test (the Monospot® test), help to rule out this infection.

Always to be considered is the possibility of secondary syphilis, especially when there are lesions on the palms and the soles of the feet. A maculopapular rash is the most common skin lesion of secondary syphilis; fever and generalized lymphadenopathy are commonly present. However, in this patient, the soles of the feet were initially spared, but later were the site of lesions. There was no indication of a primary chancre in this patient, and the VDRL was reported as negative. Headache may occur in almost any of these exanthems, so it does not help in the differential diagnosis.

Yehuda Handelsman, MD **(Medical Resident):** What made you favor the diagnosis of measles, rather than rubella?

Dr Malcolmson: The rash was not the fine, pin-point type of lesion which one sees with rubella. The lymphadenopathy was evident in anterior-chain locations in the neck and not, as in rubella, in the postauricular areas. Admittedly, the lymphadenopathy of rubella can present in many ways.

The antibody titer for rubella was already elevated early in the course of this illness, at the time of presentation. This suggested that antibody was the result of a previous exposure to rubella, even though the patient could not recall such an event.

Dr Handelsman: With the lymph node enlargement, he might have had infectious mononucleosis, even though the skin rash was not typical.

Dr Malcolmson: Leukopenia can be consistent with many viral exanthems, as are coryza, conjunctivitis, and rash. The difficult question to answer was which viral exanthem we were dealing with.

At the other hospital, a physician suspected Rocky Mountain spotted fever because the patient had told the nurse about a possible tick bite. However, when we reviewed this in considerable detail with the patient, he could not recall whether the lesion was characteristic of a tick bite, with local swelling and tenderness about the bite site. But, admittedly, the tick bite of Rocky Mountain spotted fever is often forgotten by patients.

Dr Rao: The rash of measles is said to have a superficial texture somewhat like that of sandpaper.

Dr Flynn: Measles is a disease of the skin, as well as of many other organs; it is not solely a small-vessel vasculitis which disrupts the normal control of circulation, resulting in blotchy erythema. In measles, the dermal cells contain intranuclear and intracytoplasmic inclusions, as well as microtubular aggregates that are believed to be replicating viral particles.

Arturo Constantiner, MD (Director of Nephrology): How do we account for the anemia, the leukopenia, the many immature polymorphonuclear neutrophils on the peripheral blood smear, and the relatively low platelet count? Would you not consider the diagnosis of lymphoma in this patient?

Dr Flynn: Are you asking whether the entire clinical picture might be due to lymphoma, or whether the patient may have lymphoma and has incidentally contracted measles in a setting of immunocompromise?

Dr Constantiner: I am asking whether the entire clinical picture might be due to a lymphoma.

Dr Malcolmson: We did not seriously consider the possibility of lymphoma in this patient, in view of his previous good health up to the time of this illness. Perhaps we should have.

Review of Adult Measles

Dr Malcolmson: Measles, also known as rubeola, is an acute, highly contagious disease characterized by fever, cough, conjunctivitis, an enanthema, and an exanthem. The virus is an enveloped RNA paramyxovirus of the genus *Morbillivirus*.

Instead of being an inevitable illness of childhood, in recent years measles infection has become more common in adolescents and young adults who have remained susceptible due to lack of childhood immunization or to lack of exposure to natural infection.[4,5] However, throughout the world, measles is still a disease of children. Most adults acquire active immunity in childhood. Beyond the age of ten, 90% of the population have specific antibody. This patient had no detectable antibody by the ELISA method, which suggests absence of prior exposure.

Measles is one of the most highly contagious infections. The epidemiologic data indicate that the virus is disseminated and acquired via the respiratory tract. It can be demonstrated in nasal secretions during the prodromal, early febrile, and early skin rash stages of the disease. An attack of measles is usually followed by lifelong immunity.

In the prodromal stage—of one to four days' duration—petechial lesions of the palate and pharynx may appear; or tiny white spots on the buccal mucosa known as Koplik spots may herald the appearance of the skin rash. These mucosal lesions constitute a valuable if not a pathognomonic diagnostic sign. Koplik spots indicate inflammatory mononuclear cell infiltration in the buccal submucosal glands and necrosis of focal vesicular lesions of the mucosa.

The skin rash is the result of proliferation of capillary endothelial cells in the stratum corneum and the coexistent exudation of serum and occasionally erythrocytes into the epidermis. Simultaneous with the onset of rash, measles-specific antibody is detectable in serum and by immunofluorescence in the areas of the rash. A marked leukopenia is frequently observed throughout the febrile period, as was seen in this patient. The leukopenia is characterized by a decline in the percentage of lymphocytes during the first days of the illness.

It has long been known that cell-mediated immunity is impaired during measles. There is transient suppression of the tuberculin reaction, improvement of eczema and allergic asthma, delayed wound healing, and induction of remission of nephrosis. The incubation period averages 11 days, and then the infection becomes clinically manifested by prodromal symptoms of fever, malaise, myalgia, and headaches, all of which were present in this patient. Ocular symptoms of photophobia and burning pain are accompanied within a few hours by conjunctival injection, tearing, and exudate in the conjunctival sac. Concomitantly, or soon thereafter, catarrhal inflammation of the respiratory tract leads to sneezing, coughing, and nasal discharge. The rash of measles follows the prodromal symptoms by two to four days, occasionally as late as seven days.

There is no specific antiviral therapy for measles. In the absence of complications, bed rest is the essence of treatment of this usually benign, self-limited disease. The vaccine available for the prevention of measles is derived from the Edmonstron strain of virus, which was originally isolated in the laboratory by Dr John Enders. This vaccine should be administered to infants before the age of one year. The current vaccine is fully effective and considered safe to administer to susceptible adults.

A recent five-year survey of the transmission of measles in a medical setting has been reported.[6] Since 1981, there has been a general increase in the number of cases and in the percentage of total cases thought to have been acquired in medical settings. Factors that have been implicated in this phenomenon include missed or delayed diagnoses. Measles is now a relatively rare disease, and consequently awareness of the need for control measures may be delayed. Also, more persons with measles may seek medical attention and be hospitalized because of uncertainty as to the diagnosis, as was true with this patient. In addition, diminished frequency of transmission of the infection in recent decades has probably allowed some unvaccinated young adults to escape natural measles infection. A significant proportion of medical personnel may thus have escaped exposure to the virus. Medical personnel accounted for one quarter of the cases acquired in the medical setting; nurses and physicians constituted the majority of these.[7]

The current recommendations of the Immunization Practices Advisory Committee of the US Public Health Service assume that most people born before 1957 are immune because of natural infection in early life.[7] Only 5.2% of all reported measles cases in 1984 occurred in persons born before 1957, and the incidence rate for this group was less than 0.1 case per 100,000 population.[8] Thus, it appears that the birth years 1957 and thereafter should be appropriate time-indicators for routine measles vaccination. However, two thirds of the medical personnel with measles were born before 1957, which raises concern about small groups of susceptibles in the over-30-year age group.

Respiratory isolation is recommended for hospitalized patients throughout the infectious period, which is up to four days after the onset of rash; and continued isolation of all immunocompromised patients. Hospitals and all other medical facilities are urged to insure that personnel at increased risk of contact with patients with measles should be protected by vaccination. Adequate evidence of measles immunity is based on the following: vaccination with live measles virus vaccine on or after the first birthday; laboratory evidence of measles immunity; or history of physician-diagnosed measles.

Postexposure prophylaxis should be offered to susceptible individuals who have been recently exposed to measles. The vaccine, if given within 72 hours of exposure, may provide some protection. Immune globulin can be given within six days of exposure to prevent or modify the illness in a susceptible person.

Dr Rao: A good number of our house staff were born after 1957.

Dr Flynn: But that cut-off year does not seem to hold for this patient, who was born in 1945.

Dr Rao: What is more important is that between 1963 and 1967 a killed vaccine was sometimes used. Persons receiving this vaccine developed antibodies that then combined with residual viral antigen to create immune-complex disease in the recipients. This did not give them permanent immunity to measles. So when they were later exposed to measles, they developed so-called atypical measles, which was even a worse form of the disease, with many additional complications.[9] This patient could have been inoculated with the killed vaccine.

Dr Malcolmson: The patient insists that he never received any vaccination for measles at any time.

Chester Lerner, MD (Infectious Disease Consultant): The differential diagnosis of this patient on admission included many possibilities, but it was urgent that certain forms of therapy be considered. Also, the patient had to be told of the diagnosis, as a means of identifying other individuals at special risk with whom he might have had very recent contact. One such special risk might be a pregnant woman. Another important decision involved whether he needed to be in the hospital, and whether he had an illness that might be a hazard to the hospital staff and to other patients in the hospital.

By all accounts, this patient is a generally healthy man who had an acute illness, which might possibly have been an acute tick-related illness, an acute illness with headache, or an acute summertime viral illness. But the most prominent feature was the rash, which was, I must add, not entirely typical of measles.

This acute illness seemed not to explain his anemia. Further questioning elicited a history of acute and chronic hemorrhoidal bleeding, which has been under the care of a colon surgeon over the past two or three years, during which time the anemia was well recognized. That does not explain why he has not recovered from the anemia, but for the time being it would at least explain the anemia.

How did this patient contract measles? In fact, we are currently in the middle of a measles epidemic.[10] When measles was a frequent illness of children, the first pediatrician to see a case would say, "This could be measles," but when the tenth patient with the condition came into the office within the next three days, the diagnosis was only too obvious. None of us here have seen an adult epidemic of measles in recent years, because there have not been many cases in this country even in the last five years.

However, the illness in this adult patient, just as with the children in earlier times, indicates that we are currently in the middle of a measles epidemic, and that, to me, is the most important aspect of this case. There were about 100 cases of measles in New York City during the past three weeks, which is far more than the average weekly incidence during the last five years. In the midst of this epidemic, I would say that the patient most probably had measles.

To complicate matters, this patient was transferred here from another hospital emergency room with the possible diagnosis of Rocky Mountain spotted fever. It is always difficult to discard a diagnosis offered by another physician. The first question I was asked about this patient on a Sunday night was, "How do we treat Rocky Mountain spotted fever?" Rocky Mountain spotted fever is, first of all, still an uncommon disease; about 1,000 cases are reported per year in this country, of which about 25 are in New York State; and there are probably more that remain undiagnosed.[11,12] This infrequency is a factor on which I would not rely too heavily in trying to reach a correct diagnosis.

Furthermore, the rickettsial organism is present in the ticks along our seacoast, on Long Island, and in the offshore islands of Cape Cod, including Nantucket and Martha's Vineyard. But it is present to a much lesser degree in upstate New York or the Hudson River Valley. So Rocky Mountain spotted fever is not a frequent illness, and the patient's recent hiking trip did not involve a common location for acquiring this disease. In addition, when I examined him, I though he had Koplik spots, which start just before the rash appears. After the first days, these lesions become difficult to identify as Koplik spots. His mucosal lesions had a granular appearance around hypertrophied papillae near his molars, and they certainly were suggestive of Koplik spots.

Among the major problems in the differential diagnosis here was the awareness that he might have had an insect bite, which at the other hospital was initially suspected of being due to a tick. But we all know that there are a great many arthropods out there besides ticks. Furthermore, the rash of Rocky Mountain spotted fever begins on the wrists and ankles; this does not happen in measles, and this man clearly stated that such a sequence was not true in his case.

Still, the rash of Rocky Mounain spotted fever can easily be confused with the syndrome of atypical measles. The differential diagnosis with an initial peripheral rash would have been between spotted fever and aytpical measles—but not typical measles. However, this patient is too old to have been vaccinated during the three or four years in the 1960s which Dr Rao mentioned. The leukopenia, as mentioned, is not a finding in Rocky Mountain spotted fever, but is compatible with measles or the other viral infections that were mentioned. Cytomegaloviral mononucleosis, though it may produce a rash, does not light up the patient's face with such a fiery red color. Also, this rash had a texture such as you find in measles.

The patient was placed in respiratory isolation, because of the very high degree of contagiousness of the illness when present among susceptible persons. The Centers for Disease Control have spent a great deal of effort in controlling measles, in spite of the fact that most adults and many physicians tend to disregard the condition. Patients who are injured the most seriously by the disease are children with measles encephalitis and subsequent neurologic damage. In the prevaccine era, when there were more than one-half million cases of measles per year in this country, encephalitis occurred in one case per thousand cases, often among infected children. This resulted in residual central nervous system damage, which represented many ruined lives. The number of cases of mental retardation caused by measles has been greatly reduced.

Measles is a disease characterized by invasion of the respiratory epithelium, with a significant incidence of pneumonia. There have been several recent college outbreaks; during one such epidemic there were three deaths, which were apparently related to respiratory complications from measles.[13] There is no treatment for this form of pneumonia, as there might be for other forms of viral pneumonia, beyond general supportive measures. If a patient with measles cannot be put in respiratory isolation because of the bed situation in the hospital, then the patient should not be admitted to the hospital unless he or she is desperately ill. The virus can so easily be spread to susceptible persons.

Whether this patient's condition was due to measles or not, the rash to me was a viral exanthem. The suggestion of a tick bite was probably only a first-glance inspiration of the first physician who spoke to him. However, the patient was given tetracycline because, if he did have Rocky Mountain spotted fever and it was left untreated, that disease—which is a vasculitis—can affect the cerebral hemispheres. At this point, chloramphenicol can be administered; however, there is still a 30% mortality rate if severe nuerologic symptoms develop. In this patient, antibiotic therapy was discontinued after two days and was not continued after his discharge from the hospital.

Another major consideration at the time of his admission was acute meningococcemia, which is often a fatal disease if not treated. He was not treated for that condition, because his history of an illness for one week prior to the rash was not typical of acute meningococcemia, and neither was the rash. But the question was raised because of the involvement by the rash of the palms and soles, which narrows the field of likely illnesses to such other entities as secondary syphilis, measles, Rocky Mountain spotted fever, and drug reactions. Syphilis was looked for, but there was no corroborative history or supportive laboratory findings.

In the days before vaccination for measles, over 95% of adults were immune to the disease, so there was only a small chance that this man was not immune. If he had had previous immunity, then we could have immediately discarded a present diagnosis of measles; the antibody to measles persists for many years, and the immunity to measles appears to be life-long. This patient may have had measles as a child 35 or 40 years ago, but most patients would have developed antibody and be immune. His lack of measles antibody supports a current diagnosis of measles even more emphatically. For some reason, up to recently, he may have escaped measles exposure, as is true for less than 5% of adults. And now we have a local epidemic, and this time he presumably did not escape exposure, at work or on the subway, to someone with measles who coughed two or three times in his direction and passed on the virus. If he really had measles, he should have had an antibody response within two weeks.

The rubella titer was not originally tested for IgM antibody to German measles; if he had IgM antibody to German measles, then this recent illness could be rubella, though it would be very atypical. Rubella in adults is a mild illness; the rash is generally not so prominent as in this patient, but in some patients it can be. The higher titer of rubella antibody with the ELISA test seems to be present too early, if indeed this illness was rubella.

The agents of measles and rubella are the two leading causes of maculopapular viral exanthems; also common are the enteroviruses, of which there are about 70 serotypes, including the Coxsakie and the echoviruses. There are one or two types which can provoke a rash similar to that of measles, even to the production by echovirus IX of lesions that mimic Koplik spots. We will not be able to diagnose infection with all of these, since the necessary tests are not available. If the serologic tests for measles and rubella are not eventually indicative of a diagnosis, then this patient most probably had an enteroviral infection as the cause of his exanthem.

Incidentally, when the patient first visited the emergency room, one of the housekeepers on the midnight shift there happened to see the rash and asked why this patient with measles was being admitted to the hospital. (Addendum: Four weeks after discharge, repeat ELISA test for rubeola-measles antibody was reported as 0.2—an increase in serum antibody to a low-level positive result.)

Abdollah Naficy, MD (Medical Resident): At what point would you expect the antibody tests for measles to become positive after the illness?

Dr Lerner: Within two to three weeks at the outside, and perhaps within the first five days. If this illness was measles, his reaction was not positive, certainly within the first few days.

Dr Flynn: I gather that you have to be careful in giving the vaccine to people who are allergic to neomycin, because of the small amount of neomycin in the vaccine as a preservative.

Dr Malcolmson: The contraindications to measles vaccine are pregnancy, febrile illness, history of anaphylactic reactions to eggs or neomycin sulfate, and altered immunity.

Dr Flynn: A rather curious finding has turned up in studies on Paget disease of bone. Osteoclasts in the abnormal bone are loci of measles virus as indicated by indirect immunofluorescence[14]; also, respiratory syncytial virus has been identified in the same osteoclasts.[15] There does not seem to be any explanation or known significance of these findings up to the present.

REFERENCES

1. Rocky Mountain spotted fever—United States, 1984. *MMWR* 1985; 34:195–197.
2. Massachusetts Department of Public Health: On the alert for Rocky Mountain spotted fever. *N Engl J Med* 1975; 292:1127–1128.
3. Brusch JL: Rocky Mountain spotted fever. *Infect Dis Practice* 1985; 8:1–6.
4. Measles on college campuses—United States, 1985. *MMWR* 1985; 34:445–449.
5. Krause PJ, Cherry JD, Deseda-Tous J, et al: Epidemic measles in young adults. *Ann Intern Med* 1979; 90:873–876.
6. Davis RM, Orenstein WA, Frank JA Jr, et al: Transmission of measles in medical settings, 1980 through 1984. *JAMA* 1986; 255:1295–1298.
7. Measles prevention: Recommendations of the Immunization Practices Advisory Committee (ACIP). *MMWR* 1982; 31:229–231.
8. Measles—United States, 1984. *MMWR* 1985; 34:308–312.
9. Martin DB, Weiner LB, Nieburg PI, et al: Atypical measles in adolescents and young adults. *Ann Intern Med* 1979; 90:877–881.
10. Measles—United States, first 26 weeks, 1986. *JAMA* 1986; 256:2168, 2173.
11. Cases of specified notifiable diseases, United States. *MMWR* 1987; 35:801.
12. Rocky Mountain spotted fever—United States, 1981. *MMWR* 1982; 31:261–263.
13. Multiple measles outbreaks on college campuses—Ohio, Massachusetts, Illinois [Leads from the *MMWR*]. *JAMA* 1985; 253:1853–1857.
14. Mills BG, Singer FR, Weiner LP, et al: Cell cultures from bone affected by Paget's disease. *Arthritis Rheum* 1980; 23:1115–1120.
15. Mills BG, Singer FR, Weiner LP, et al: Immunohistological demonstration respiratory syncytial virus antigens in Paget's disease of bone. *Proc Natl Acad Sci USA* 1981; 78:1209–1213.

FROM THE LIBRARY

PROPHYLAXIS AND TREATMENT OF MEASLES, DIPHTHERIA, AND POLIOMYELITIS

Measles. The seroprophylaxis of measles has become rather difficult since convalescent serum has fallen into disfavor and gamma globulin has been withdrawn from the market. Gamma globulin is now used almost exclusively for the prevention of poliomyelitis. The small amount delegated to the prevention of measles and infectious hepatitis in New York City is distributed almost invariably in the case of measles to susceptible family contacts under five years of age. Gamma globulin is denied to all who are exposed under different conditions as well as to those over five years who are exposed at home. Specifically this means that infants exposed outside of their homes and older children exposed in schools, buses, play groups, parties, etc., regardless of age cannot get gamma globulin. One exception to these rules is the sick child of any age for whom gamma globulin is usually made available after special consideration. Each such case is a special situation which has to be discussed with the health authority who must be satisfied that the child is in urgent need of the gamma globulin before it is granted. Another exception is the hospital ward or nursery where measles is discovered, when gamma globulin is usually granted for all susceptible contacts.

In view of the fact that there are approximately 1,000,000 cases of measles in the United States per year and many more contacts and because of the severity of the unmodified disease, especially in infants and young children, the problem of prophylaxis is one of great importance and concern. Because gamma globulin is not available at the corner drug store, it is quite possible that many children who need prophylaxis will not get it. The recent experience with gamma globulin in the prophylaxis of poliomyelitis has generated a widespread feeling that gamma globulin should be returned to the open market, but there is little hope that this will be effected in the immediate future. . . .

SAMUEL KARELITZ, MD
(*NY State J Med* 1954; 54:2831–2836)

COMMITTEE REPORT

Ethical considerations of reproductive technologies[*]

FRED ROSNER, MD; ERIC J. CASSELL, MD; MICHAEL L. FRIEDLAND, MD; ALLISON B. LANDOLT, MD; LAURENCE LOEB, MD; PATRICIA J. NUMANN, MD; FERNANDO V. ORA, MD; HERMAN M. RISEMBERG, MD; PETER P. SORDILLO, MD

The custody battle in the state of New Jersey over "Baby M" generated nationwide debate on the ethical, moral, and legal aspects of surrogate motherhood. This is an arrangement whereby a woman agrees to be artificially inseminated and then to give up for adoption the child thus conceived to the biological father and his wife, whose infertility is generally the reason for the couple's seeking other means of obtaining a child. An attorney attends to the legal phases of the transaction, and draws up a contract between the adoptive parents and the surrogate mother to compensate the latter for the costs of her medical care, her living expenses, and any discomfort or inconvenience occasioned by the pregnancy.

The biological father in the "Baby M" case was William Stern, whose wife, Elizabeth, a pediatrician, had been advised against becoming pregnant to avoid exacerbating her mild case of multiple sclerosis. Mary Beth Whitehead agreed to a payment of $10,000 plus medical expenses to bear the baby for the Sterns, and was artificially inseminated with William Stern's sperm. After the child was born, however, Whitehead decided she wanted to keep the baby, and fled with her to Florida. She was finally found and returned to New Jersey where the battle for custody was fought in the courts.

Currently, no state has a law expressly forbidding or endorsing surrogate parenthood, although several states, including California and New York, are considering legislation to regulate this practice and to protect the best interests of all concerned parties, including the baby.

Artificial insemination of women has been successfully practiced for nearly a century to enable otherwise infertile couples to have children. Different legal, moral, ethical, and social questions are posed by the use of donor sperm versus the husband's sperm. Some of these questions are discussed below.

In 1978, Louise Brown, the world's first "test tube" baby, was born as a result of in vitro fertilization, the process of mixing an egg with some sperm in a petri dish to achieve fertilization, then transferring the early embryo to a woman's uterus with the hope that it would successfully implant and lead to the birth of a healthy child. In the case of Louise Brown, the sperm and egg used were those of Mr and Mrs Brown. Since 1978, there have been several hundred thousand births resulting from this technique. Often the embryo is implanted into the uterus of a woman other than the egg donor; the sperm utilized may also be obtained from a man other than the husband of the egg donor or the woman into whom the embryo is implanted. This technique is characterized by some as science at its best and by others as immoral meddling. Other possibilities suggested by this technology include the freezing and storage of eggs and embryos, the donation or sale of gametes or embryos, early gender selection, early diagnosis of genetic or chromosomal abnormalities, and embryo research.

Some of the many legal, moral, ethical, and social questions involved in these procedures of reproductive technology are the following:

Artificial insemination

Who is responsible if a defective child is born?

Should all sperm donors be screened for genetic defects?

What is the donor's responsibility for (knowingly?) giving defective sperm?

Should sperm from a donor with acquired immunodeficiency syndrome be automatically rejected?

Is the physician guilty of perjury when he or she signs the birth certificate knowing that the biological father is not the one named on the birth certificate?

Is the child considered legitimate?

What are the child's rights concerning inheritance, support, and custody?

Can the child sue for the donor's estate?

Can the mother sue the donor for support of the child?

Can the donor sue for custody of the child?

What is the donor's responsibility concerning the provision of support for his offspring?

Should the husband legally adopt the child when his wife gives birth?

Address correspondence to Dr Rosner, Director, Department of Medicine, Queens Hospital Center Affiliation of the Long Island Jewish Medical Center, 82-68 164th St, Jamaica, NY 11432.

[*] Prepared as an educational and informational report by the Committee on Bioethical Issues of the Medical Society of the State of New York.

How do adoption laws apply here, or do they apply at all?

Surrogate motherhood

What if the adoptive parents die or are divorced before the birth of the child, or decide they do not want the baby after all?

What if the child is born defective?

Is it proper for surrogates to have children to be turned over to single people, or to homosexual couples?

What if the surrogate mother decides to have an abortion or wishes to keep the baby?

If a surrogate mother receives a fee, is she in effect selling her baby?

Should the surrogate be married or single, have other children, or have no children?

Should the adoptive parents (including the biological father) meet the surrogate?

Should the child know about the surrogate arrangement when he or she grows up?

Is monetary compensation the real issue?

What kind of counseling should be done with all parties, and what records should be kept?

One underlying theme in considering these legal issues and moral dilemmas is the motivation behind the actions of the concerned parties. On one extreme are those sperm donors and surrogate mothers who are motivated purely by a desire for monetary gain. On the other are those couples that have been trying unsuccessfully for years to have a child and finally resort to one of these methods. Their motivation is pure and represents their burning desire to have a child. Many ethical and moral conflicts arise because the motivation of one party (the sperm donor or surrogate mother) is different from the motivation of the other (the husband and wife). Few ethical concerns are posed by the case of the husband and wife who resort to in vitro fertilization using their own egg and sperm, or that of the young woman with Hodgkin disease or other cancer, who cryopreserves her eggs for later use in having children with her own spouse. Such cases pose very few moral dilemmas other than those of the propriety or religious permissibility of artificial insemination and in vitro fertilization. The religious and theological questions pertaining to these reproductive technologies are numerous and are important to physicians of various religious backgrounds and practices, but these are beyond the scope of this paper.

THE WARNOCK COMMITTEE OF INQUIRY INTO HUMAN FERTILIZATION AND EMBRYOLOGY

In 1983, the British Parliament asked Dame Mary Warnock to chair a blue-ribbon commission to explore the ethical implications of advances in the treatment of human infertility. The Warnock report[1] was completed in 1984 and the recommendations were published and editorially and legally commented upon in *The Lancet*.[2-4] The report was optimistic in tone and sympathetic to the desires of infertile people. It stated that sperm and egg donations and in vitro fertilization are acceptable techniques for the treatment of infertility. It recommended that the effects of freezing of embryos continue to be explored, although it warned that frozen embryos should not be transplanted until it has been established that no unacceptable risk of abnormality is involved.

The Warnock committee also recommended strict legal controls on the provision of infertility services, on research on human embryos, and on surrogacy arrangements. Under its guidelines, a statutory licensing authority would be set up to regulate and monitor infertility services and research. All practitioners who use human gametes or embryos for clinical or research purposes would have to be licensed, as would the premises in which they work. Legal controls would apply to the collection of sperm, the storage of frozen eggs, and the treatment of infertile patients.

The committee took a pragmatic view of surrogate motherhood, based heavily on the fear of commercial exploitation, or "womb-leasing." A limited, non-profit-making surrogacy service, subject to licensing and inspection, was considered by the committee but condemned by the majority since it was felt it would encourage the growth of surrogacy. The majority also recommended that legislation be enacted to make surrogacy a criminal offense and that surrogacy contracts be considered illegal and unenforceable.[1]

On the other hand, the committee recommended that a child born following artificial insemination with donor semen should be recognized as legitimate and that the semen donor and the child should be considered as having no parental relationship.[1] The committee's position on who should have access to artificial insemination with donor sperm or to in vitro fertilization was rather liberal, but the committee also suggested that no more than ten children should be fathered by a single donor. With some reservations, the committee approved the use of stored human embryos as a treatment for infertility and advised that the child born by means of embryo donation should be recognized as the product of the nurturing, rather than the genetic, parents. The committee also suggested that no unimplanted human embryo be allowed to develop past the 14th day after fertilization. The committee was sharply divided on the issue of the legal status of the embryos and under what circumstances and rules research might be performed upon them.

In response to both the Warnock committee report and the hostile and disapproving mood of the British public towards commercial surrogacy, the British government passed the Surrogacy Arrangements Act of 1985 which makes it a criminal offense to benefit from commercial surrogacy.[5] Voluntary surrogacy, however, is still within the law.

THE AMERICAN MEDICAL ASSOCIATION'S JUDICIAL COUNCIL

Reports and opinions of the Judicial Council of the American Medical Association (AMA) on artificial insemination by donor, in vitro fertilization, and surrogate parenting were published in 1984 and 1985.[6,7] In 1986, the AMA House of Delegates adopted the reports containing the opinions of the AMA's Council on Ethical and Judicial Affairs on surrogate parenting, artificial insemination, embryo research, and fetal research.

In regard to surrogate motherhood, the council expressed concern about

> . . . the ethical, social and legal problems that may arise in an arrangement in which a woman agrees to become pregnant through artificial insemination, to carry to term and to give the child thus conceived to other persons to serve as adoptive parents. The welfare of the child should be a foremost consideration. In ordinary adoption proceedings an appropriate agency usually investigates prospective adoptive parents to determine their fitness as parents. This precaution is not always present in surrogate motherhood arrangements.

The Judicial Council was also concerned that, if there were a subsequent birth of a defective child, the prospective adoptive parents and the woman who gave birth to the child might not want to or would be unable to assume the responsibilities of parenthood. Many other ethical, social, and morally difficult situations were envisioned by the council. For example, the woman who has contracted to bear the child may decide to have an abortion or to refuse to give the child up for adoption. Another consideration which may be overlooked is the psychological impairment that may occur in a woman who deliberately conceives with the intention of bearing a child which she will give up. Therefore, the Judicial Council concluded that surrogate motherhood presents many ethical, legal, psychological, societal, and financial concerns and does not represent a satisfactory reproductive alternative for people who wish to become parents.

In regard to artificial insemination, the council advised that physicians should obtain the informed consent of both women seeking artificial insemination and their husbands. They should also inform them that children conceived through the process have all the rights of naturally conceived children. Physicians were urged to use the best available scientific techniques to screen semen donors for genetic defects, inheritable and infectious diseases, and other disorders that may affect fetuses. It was recommended that parents and children not be informed of the identity of donors. Physicians were also advised to avoid the frequent use of semen from the same donors.

The council commented that the technique of in vitro fertilization, combined with embryo transplantation, helps certain previously infertile couples to bear children, and that it is useful also in research aimed at understanding how genetic defects arise and are transmitted and how they might be prevented or treated.

Finally, in regard to embryo and fetal research, the council stated that all fertilized eggs that have the potential for human life and that will be implanted into the uteruses of women should not be subjected to laboratory research. All fertilized ova not used for implantation and that are maintained for research purposes should be handled with the strictest adherence to the Principles of Medical Ethics, the council's guidelines for research and medical practice contained in its fetal research opinion, and the highest standards of medical practice.

STATEMENT OF THE ETHICS COMMITTEE OF THE AMERICAN COLLEGE OF OBSTETRICIANS AND GYNECOLOGISTS

In 1984, the American College of Obstetricians and Gynecologists (ACOG) published a rather technical statement on in vitro fertilization, detailing the procedure involved, as well as some of its risks and benefits.[8] One short paragraph was devoted to "ethical considerations" which acknowledged that the procedure raised "many questions of ethical importance," and promised that ACOG would discuss some of these issues "in the near future."

On September 1, 1986, the ACOG released a statement prepared by its Committee on Ethics, addressing such issues as frozen embryos, donating or selling embryos, embryo research, gender selection, and insurance coverage.[9] The statement began by pointing out the support by a broad social consensus of the simplest case in which the gametes are provided by husband and wife and the pregnancy, if successfully initiated by in vitro fertilization and embryo placement, is carried by the wife. Ethical questions remain concerning the optimum number of eggs to be fertilized, the optimum number of embryos to be placed during one cycle, and the use or disposition of surplus embryos. Among the alternatives available are the freezing of embryos, the donation of embryos to another couple, donation of embryos for research, or the discarding of surplus embryos. The ACOG statement recommended that these options be discussed in advance with the gamete providers.

Regarding the use of artificial insemination or in vitro fertilization with embryo placement for single individuals, the ACOG statement suggested that physicians should handle requests from single persons within an overall policy regarding the screening of candidates for infertility services. The primary consideration should be the probable welfare of the resulting child. The statement also voiced concern about the quality standards of centers and practitioners offering these reproductive services.

The ACOG statement asserted that

> . . . embryo freezing raises almost unprecedented metaphysical, ethical, and legal problems. The metaphysical problem is what status to ascribe to an embryo that can be maintained for years, perhaps even decades, in a state of suspended development. The ethical and legal problem to be resolved is whether frozen embryos should be regarded, in some sense, as the responsibility of their progenitors. It is recommended that, when embryos are to be frozen, couples should be required to declare their wishes in advance, in writing, concerning the disposition of the embryos in the event of the death of a spouse or both members of a couple, the wife's incapacitation, or the dissolution of the marriage. For practical reasons, centers should also set policies on time limits for maintaining early embryos in a frozen state.

Artificial insemination by donor is considered ethically acceptable if the husband carries a serious genetic defect or if he cannot fertilize the eggs of his wife. In similar cases, egg donation would also seem to be ethically acceptable, although egg retrieval entails greater risk to the donor than does semen donation. The receipt of a donated embryo, according to the ACOG statement, "is analogous to adoption in the sense that neither adopting parent contributes genetically to the offspring" but the recipient couple has the advantage of experiencing the pregnancy and the delivery. Any donation program should include careful screening of donors for infectious diseases and genetic problems. Careful records should be kept and organized in a two-tiered system that protects the privacy of donors yet allows access to relevant medical information.

The sale of gametes is a serious ethical choice for our society. Commercial sperm banks already exist in the United States, and when the cryopreservation of eggs becomes technically feasible, such banks will probably also want to provide human eggs for a fee. The ACOG statement affirmed that a nonprofit system for gamete collection and distribution would be ethically preferable to the current system, in part because it would reduce the incentive for gamete donors to withhold vital medical information. Preimplantation gender selection to help couples avoid sex-linked genetic disorders is viewed by the ACOG as "ethically justifiable."

With regard to laboratory research on early human embryos, the ACOG statement took an intermediate view on the moral status of the human embryo.

> Our moral obligations to early human embryos [less than 15 days] may be outweighed by the duty to develop new and better methods for providing care to pregnant women, infertile couples, early embryos, and future children . . . In light of this conclusion, it is recommended that laboratory research with early human embryos proceed, guided by ethical standards and subject to prior review.

ETHICS COMMITTEE REPORT OF THE AMERICAN FERTILITY SOCIETY

One week after the ACOG released its statement on ethical issues related to in vitro fertilization and embryo placement and research, the American Fertility Society's Ethics Committee (AFSEC) published its report.[10] The report identified four major issues relating to the new reproductive technology: the degree of artificiality of the new reproductive technology, the moral status of the human preembryo, the role of the family or genetic lineage, and the appropriate role of government.

The AFSEC considers the following procedures ethically acceptable: the use of artificial insemination with husband's sperm for demonstrated medical indication; the use of artificial insemination with donor sperm; basic in vitro fertilization involving the use of donor sperm, donor eggs, and donor preembryos; the cryopreservation of sperm; and research on the preembryo.

The AFSEC considers the following as clinical experiments, the general application of which is premature: uterine lavage for preembryo transfer, the cryopreservation of unfertilized eggs, fertilized eggs, and preembryos; and surrogate motherhood. The AFSEC opposes the use of surrogate gestational mothers for nonmedical reasons but believes that there may be medical reasons to justify individual decisions. There are no adequate reasons, continues the AFSEC statement, to recommend legal prohibition of surrogate motherhood. Nevertheless, the AFSEC had serious ethical reservations about surrogacy and recommended that if it is pursued, it should be done as a clinical experiment but without general application of the procedure until more data is available about its risks and virtues.

Finally, the report expressed concern about quality assurance in the use of reproductive technologies and suggested that the American Fertility Society, in conjunction with other public and professional groups, take the initiative "in stimulating active training, in reviewing standards, in providing information, and in encouraging professional and public oversight of ongoing reproductive technologies." Several appendices to the report describe a minimal genetic screen for gamete donors, provide new guidelines for the use of semen for donor insemination, and cite minimal standards for programs of in vitro fertilization.

COMMENT

Artificial insemination, in vitro fertilization, surrogate motherhood, and cryopreservation of sperm, eggs, or fertilized zygotes for later use are strongly opposed by some people on moral and ethical grounds, and just as strongly justified by others. Human procreation should not be converted into the "manufacture" of progeny. The intimate love joining husband and wife together should not be broken by the "biologization" of family life.

On the other hand, if infertility is considered to be an illness, either physiological or emotional or both, the physician's duty and mandate is to heal illness and to overcome, if possible, both somatic and emotional strains related to that illness. To help a couple to have their own child through the modern technologies of artificial insemination or in vitro fertilization seems to be within the physician's purview and might even strengthen the bonds of the marriage and the family structure. The use of host or surrogate mothers for the convenience of couples able to conceive by normal coitus cannot be condoned. However, an infertile couple may have recourse to the new reproductive technologies including the use of a surrogate mother, in the absence of alternatives, in order to effect pregnancy and by so doing preserve their marriage and bring themselves happiness.

REFERENCES

1. *Report of the Committee of Inquiry into Human Fertilization and Embryology*. Cmnd 9314. London, H M Stationery Office, 1984.
2. Recommendations of the Warnock Committee. *Lancet* 1984; 2:217–218.
3. Human reproduction: Regulated progress or damned interference? [editorial]. *Lancet* 1984; 2:202–204.
4. Brahams D: Warnock report on human fertilisation and embryology. *Lancet* 1984; 2:238–239.
5. Brahams D: The hasty British ban on commercial surrogacy. *Hastings Center Rep* 1987; 17:16–19.
6 Recent opinions of the Judicial Council of the American Medical Association. *JAMA* 1984; 251:2078–2079.
7. Reports of the Judicial Council of the American Medical Association, December 1984. *JAMA* 1985; 253:2424–2425.
8. *Human In Vitro Fertilization and Embryo Placement* (ACOG Committee Statement). Washington, DC, American College of Obstetricians and Gynecologists, 1984.
9. *Ethical Issues in Human In Vitro Fertilization and Embryo Placement* (ACOG Committee Statement). Washington, DC, American College of Obstetricians and Gynecologists, 1986.
10. Ethics Committee of the American Fertility Society: Ethical considerations of the new reproductive technologies. *Fertility and Sterility* 1986; 46 (suppl 1):1-94S.

Polyarteritis nodosa presenting with migratory soft tissue swelling

GARY S. MEREDITH, MD; HAL J. MITNICK, MD; HARRIS E. BURSTIN, MD;
SOL S. ZIMMERMAN, MD

Polyarteritis (periarteritis) nodosa was initially described in 1866 by Kussmaul and Maier[1] as a systemic disease. Cutaneous features of the illness have long been recognized.[2-5] Polyarteritis nodosa limited to the skin has been considered a discrete entity by some authors and may be associated with minor systemic involvement and a better overall prognosis than its systemic counterpart.[6-10] The varied cutaneous manifestations include nodules, livedo reticularis, gangrene, and ulcers. We report a case of a patient with polyarteritis nodosa that presented with painful soft tissue swelling, evolved in a migratory pattern, and rapidly improved with therapy.

CASE REPORT

A 13-year-old boy was transferred to the pediatrics service at New York University Hospital for evaluation of fever and a painful left arm. The patient had previously been well and in excellent health until one month prior to admission, when he reported a painful and swollen left leg following minor trauma. These symptoms resolved with local therapy. Several days later, the patient noted a painful and edematous right upper extremity with erythema extending from the fingers to the shoulder. He had a temperature of 102°F and was admitted to another hospital with a diagnosis of cellulitis.

A laboratory evaluation at that time revealed a white blood cell (WBC) count of $>30,000/mm^3$ (84% neutrophils, 5% band forms, 7% lymphocytes, 3% monocytes, 1% eosinophils), with a hematocrit of 30%. The platelet count was consistently $>700,000/mm^3$, and the erythrocyte sedimentation rate (ESR) was >50 mm/hr. Urinalysis revealed blood by dipstick with 0–4 red blood cells per high power field. A test for myoglobinuria was negative.

Radiographs of the right arm and left leg were normal, as were radiographs of the chest and abdomen. An electrocardiogram revealed sinus tachycardia. Renal, liver, and spleen scans were unremarkable. A bone scan at the time of presentation revealed increased soft tissue activity in the right upper extremity thought to be consistent with the clinical diagnosis of cellulitis.

A course of intravenous antibiotics was administered, and the process slowly resolved, despite continued daily temperature spikes to 102°F. With resolution of the swelling, a similar process developed in the left upper extremity, as well as new symptoms of pain and tenderness in the left ankle and calf. A migratory pattern was apparent.

At the time of transfer, the patient was a well-developed boy in obvious pain. His blood pressure was 140/80 mm Hg; pulse rate, 100/min; respiratory rate, 20/min; and temperature, 101°F. Physical examination was remarkable for marked generalized swelling and tenderness of the left upper extremity without evidence of synovitis in the elbow or wrist. The skin was extremely tender to light touch. No focal nodules or rash was noted. There was pain on testing of range of motion of the right upper extremity without swelling or erythema. Pain with extension of the left knee and with dorsiflexion of the left ankle was also noted. Lymphadenopathy was absent.

Laboratory studies again revealed a WBC $>30,000/mm^3$. The hemoglobin level ranged from 8.7 to 10.4 g/dL, and the platelet count was now $1,000,000/mm^3$. The ESR was 100 mm/hr. Serum electrolyte, blood urea nitrogen, and creatinine levels were within normal limits. Rheumatoid factor was less than 1:20, and antinuclear antibody titers were negative. Circulating immune complexes measured by polyethylene glycol (PEG) precipitate were elevated at 0.07 (normal, <0.03), and immunoelectrophoresis revealed a polyoligoclonal immunoglobulin pattern. Cryoglobulins were absent. The serum C3 level was 156 mg/dL (normal range, 83–177 mg/dL), and the C4 level was 21 mg/dL (normal range, 15–45 mg/dL). Multiple sets of blood cultures were without growth.

The hospital course was notable for a progressive increase in the left arm edema, which subsequently spread to involve the chest wall on the left side. In addition, erythema and induration developed over the right anterior thigh overlying the hip.

A venogram of the left upper extremity was normal. A computed tomographic (CT) scan of the chest revealed diffuse soft tissue swelling of the left upper extremity and of the soft tissues of the left anterior chest wall and the periscapular region. The soft tissue swelling involved both the muscular and fatty tissue planes (Fig 1). A CT scan of the abdomen was unremarkable.

The patient underwent full thickness skin and muscle biopsy of the right anterior thigh in the region of erythema previously noted. Histopathologic studies revealed fibrinoid necrosis with focal arteritis involving the small and medium-sized muscular arteries in the septae of the subcutaneous tissue. Adjacent panniculitis was present (Fig 2). The skeletal muscle was without pathologic changes.

A diagnosis of polyarteritis nodosa was made. Oral prednisone therapy at 60 mg/day was started, and there was rapid clinical

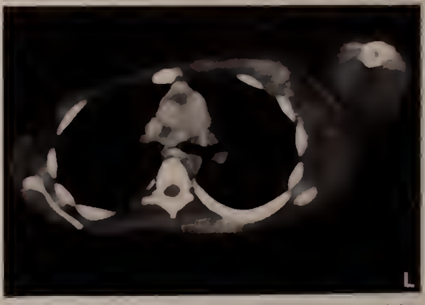


FIGURE 1. Computed tomographic scan of the chest demonstrating asymmetric soft tissue swelling of the left chest and upper extremity.

From the Division of Rheumatology of the Department of Medicine (Drs Meredith and Mitnick) and the Department of Pediatrics (Drs Burstin and Zimmerman), New York University Medical Center.

Address correspondence to Dr Meredith, Division of Rheumatology, Department of Medicine, New York University Medical Center, 550 First Ave, New York, NY 10016.

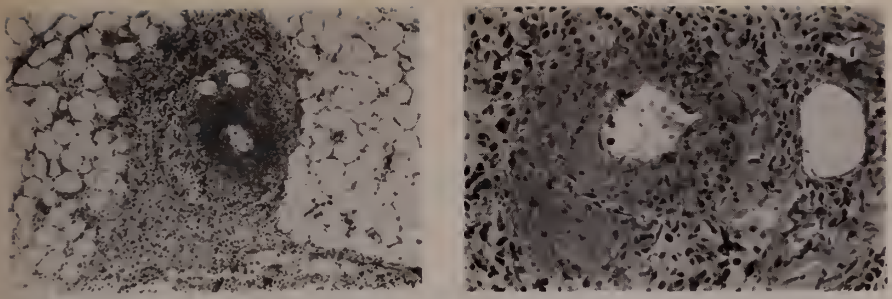


FIGURE 2. Tissue sections prepared from excisional biopsy of skin with underlying subcutaneous tissue. **Left**, vasculitis with panniculitis (hematoxylin-eosin stain, original magnification × 188); **Right**, muscular artery illustrating fibrinoid necrosis (hematoxylin-eosin stain, original magnification × 640).

improvement. Laboratory study results also showed improvement. Prednisone dosage was tapered over a six-week period. At the three-month follow-up visit, the WBC count had declined to 11,700/mm^3, the hematocrit had risen to 43.5%, the platelet count had declined to 279,000/mm^3, and the ESR was 10 mm/hr. The patient remained well at follow-up evaluation after six months with no medication.

DISCUSSION

Polyarteritis nodosa is one of several illnesses categorized under the broad classification of systemic necrotizing vasculitis. Clinically, it can be limited or diffuse and can range in severity from mild to a rapidly fatal disease. It can involve many organ systems including the kidneys, heart, gastrointestinal tract, nerves, muscles, skin, and joints. Morbidity and mortality are related to the type and degree of organ system involvement. Glomerulonephritis, renal vasculitis, hypertension, coronary arteritis, intestinal infarction, and mononeuritis multiplex are among the many clinical manifestations of the disease. Involvement of the kidneys and gastrointestinal tract is associated with a worse prognosis.

Polyarteritis nodosa limited to the skin has been described by many authors. A debate concerning whether or not cutaneous polyarteritis is a discrete entity has existed since Lindberg[2] described the cutaneous illness in 1931. Many believe that the cutaneous form is characterized by a benign course.[9,10] The cutaneous features are varied and have included livedo reticularis, nodules, gangrene, and ulcers. In addition to the diversity of skin lesions, a variety of clinical syndromes of cutaneous polyarteritis nodosa have been noted. These include polyarteritis nodosa presenting with leg pains resembling claudication,[11] and more recent reports of cases that simulate chronic recurrent thrombophlebitis.[12,13]

Despite a spectrum of clinical findings, histopathologic results consistently reveal focal inflammation of the skin and subcutaneous small and medium-sized muscular arteries. The dermis shows nonspecific lymphocytic perivascular infiltrates and is therefore nondiagnostic. Indeed, Diaz-Perez and Winkelmann[10] have cautioned against performing punch biopsy in these cases.

The case described here of a young boy with fever and soft tissue swelling illustrates what we believe to be a unique report of polyarteritis nodosa presenting with an acute migratory cutaneous pattern. The histopathologic findings from an excisional biopsy from an active skin lesion revealed a diagnostic, acute necrotizing arteritis with panniculitis. The presence of fever, leukocytosis, anemia, elevated ESR, and mild urinary abnormalities is not uncommon in cutaneous polyarteritis nodosa. The rapid response to corticosteroid therapy is typical of that previously observed in the majority of reported cases of polyarteritis limited to the integument, and supports the concept that cutaneous disease has a better prognosis.[9,10,12] Thus, although several of the clinical features noted are consistent with previously reported cases of cutaneous disease, we would emphasize that polyarteritis nodosa limited to the skin remains a controversial concept, and we did not rigorously exclude visceral involvement with biopsy or angiography.

Two aspects of this presentation are unusual. First, the acute migratory nature of the illness is notable and, to our knowledge, this feature has not previously been reported. The second point involves the diffuse nature of the cutaneous involvement. The marked swelling in the absence of focal cutaneous findings was clinically suggestive of thrombophlebitis. The painful swelling in association with the normal venogram clinically resembled the recently described syndrome of hyperalgesic pseudothrombophlebitis.[14] An explanation for the findings in the present case may relate to functional obstruction of small venules and lymphatic vessels as they course through inflamed panniculus in close proximity to the involved arteries.

The migratory nature of the illness in this patient was a clinical clue that an infectious cellulitis was unlikely. We suggest that a diagnosis of polyarteritis nodosa should be considered in patients with a predominantly cutaneous disease with laboratory evidence of inflammation in the absence of infection. Furthermore, consideration should be given to early excisional biopsy of affected skin.

Acknowledgments. The authors thank Dr Robert Schoentag for technical assistance with the photomicrographs, and Dr Gerald Weissmann for reviewing the manuscript.

REFERENCES

1. Kussmaul A, Maier R: Ueber eine bisher nicht beschriebene eigenthumliche Arterienerkrankung (Periarteritis Nodosa) die mit Morbus Brightii und rapid fortschreitender allgemeiner Muskellahmung einhergeht. *Dtsch Arch Klin Med* 1866; 1:484–518.
2. Lindberg K: Ein Beitrag zur Kenntnis der Periarteritis nodosa. *Acta Med Scand* 1931; 76:183–225.
3. Slinger WN, Starck V: Cutaneous form of polyarteritis nodosa: Report of case. *Arch Dermatol* 1951; 63:461–468.
4. Lyell A, Church R: Cutaneous manifestations of polyarteritis nodosa. *Br J Dermatol* 1954; 66:335–343.
5. Belisario JC: Cutaneous manifestations in polyarteritis (periarteritis) nodosa: Report of a case with livedo reticularis. *Arch Dermatol* 1960; 82:526–532.
6. Rötstein J, Good RA: Periarteritis nodosa, limited to skin and muscle: Report of a case with a discussion of the practical and theoretical implications. *Arthritis Rheum* 1958; 1:462–472.
7. Ruiter M: The so-called cutaneous type of periarteritis nodosa. *Br J Dermatol* 1958; 70:102–106.
8. Fisher I, Orkin M: Cutaneous form of periarteritis nodosa—an entity? *Arch Dermatol* 1964; 89:180–189.
9. Borrie P: Cutaneous polyarteritis nodosa. *Br J Dermatol* 1972; 87:87–95.
10. Diaz-Perez JL, Winkelmann RK: Cutaneous periarteritis nodosa. *Arch Dermatol* 1974; 110:407–414.
11. Golding DN: Polyarteritis presenting with leg pains. *Br Med J* 1970; 1:277–278.
12. Rawlinson T, Ball GV: Chronic cutaneous polyarteritis nodosa: A distinct subset of polyarteritis nodosa? [letter]. *Arch Intern Med* 1981; 141:961.
13. Zimran A, Shilo S, Dollberg L, et al: Chronic cutaneous polyarteritis nodosa simulating recurrent thrombophlebitis. *Isr J Med Sci* 1985; 21:154–156.
14. Abramson SB, Odajnyk CM, Grieco AJ, et al: Hyperalgesic pseudothrombophlebitis. New syndrome in male homosexuals. *Am J Med* 1985; 78:317–320.

See also p 381.

Tuberculosis mimicking acute pyogenic bacterial pneumonia

KRISHNAN PADMANABHAN, MD; SANTI R. DHAR, MD; B. BHATT, MD

Pulmonary tuberculosis in the adult can present with varied roentgenographic manifestations. The most common is an infiltrate or cavity in an upper lobe. Acute lobar consolidation or tuberculous pneumonia is a less well recognized mode of presentation. A review of a 12-month experience with 26 newly diagnosed cases of pulmonary tuberculosis in a community hospital revealed three patients (11.5%) with acute tuberculous pneumonia. Continued awareness that pulmonary tuberculosis can present with lobar consolidation mimicking acute pyogenic bacterial pneumonia is important, to avoid misdiagnosis or delay in diagnosis. Failure to recognize this presentation will lead to further dissemination of the disease and to exposure of hospital personnel to the risk of acquiring the infection.

CASE REPORTS

Case 1. A 43-year-old woman was admitted to the hospital with fever, night sweats, productive cough, and right-sided pleuritic chest pain of seven days' duration. Erythromycin therapy that had been started four days prior to admission, when the patient was seen on an outpatient basis, was of no benefit. Examination on admission revealed an acutely ill and febrile patient with a temperature of 40°C. Chest auscultation revealed rales and bronchial breathing anteriorly and posteriorly over the right upper chest, indicative of consolidation in the right upper lobe. The peripheral leukocyte count was 9,300/mm^3 with 70% polymorphonuclear leukocytes and 30% lymphocytes. A chest roentgenogram revealed consolidation of the right upper lobe, and an air bronchogram was visible (Fig 1). Gram's stain of sputum showed a mixed flora. Empirical therapy with ampicillin was instituted, with no improvement over the next two days. At this time, sputum cultures revealed no growth, but Ziehl-Neelsen stain of the sputum showed numerous acid-fast bacilli which, on culture, proved to be *Mycobacterium tuberculosis.* A skin test using purified protein derivative (PPD) (5Tu) was positive (20 mm).

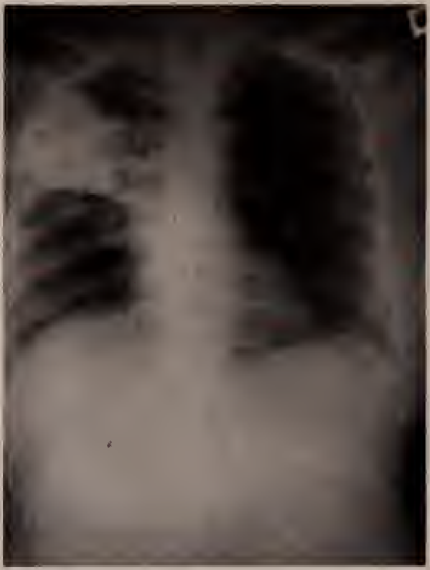


FIGURE 1. Posteroanterior roentgenogram showing consolidation with air bronchogram in right upper lobe (Case 1).

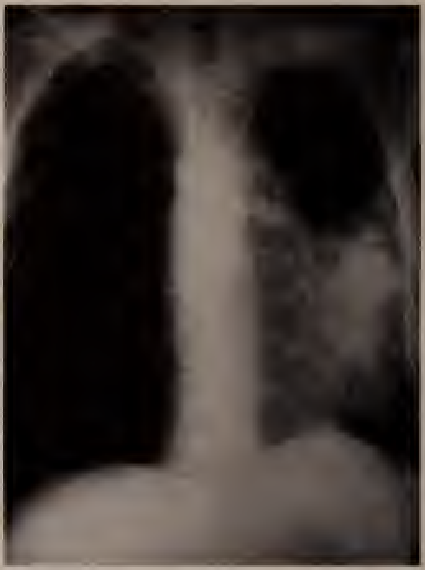


FIGURE 2. Posteroanterior roentgenogram showing consolidation in the lingula of left upper lobe and apical segment of left lower lobe (Case 2).

Treatment with isoniazid, rifampin, and ethambutol led to resolution of the fever in three days and complete resolution of the roentgenographic abnormality in three months.

Case 2. A 65-year-old man was admitted to the hospital with a nonproductive cough of two weeks' duration and fever associated with night sweats of one week's duration. Outpatient treatment with erythromycin was ineffective. On admission, he appeared acutely ill and was febrile, with a temperature of 38.8°C. Chest auscultation revealed rales and bronchial breathing in the left midzone anteriorly and posteriorly. A chest roentgenogram disclosed consolidation over the lingula and in the apical segment of the left lower lobe (Fig 2). The peripheral leukocyte count was 8,300/mm^3. Gram's stain of sputum revealed no organisms, and Ziehl-Neelsen stain revealed no acid-fast bacilli. There was no response to treatment with intravenous ampicillin therapy during the next two days. On the third day of hospitalization, fiberoptic bronchoscopy with transbronchial lung biopsy, brushing, and lavage of the lingular segments was performed. The biopsy specimen contained epithelioid granuloma, and bronchial washings and brushings revealed numerous acid-fast bacilli. Cultures from this tissue subsequently grew *M tuberculosis.* Treatment for tuberculosis was instituted, and further care was continued at another institution.

Case 3. A 30-year-old woman, seven months pregnant, was admitted to the hospital with fever, productive cough, and left-sided pleuritic chest pain of five days' duration. She had a history of intravenous heroin abuse in the distant past. She did not smoke, denied alcohol intake, and kept a healthy parakeet at home. On admission, she was febrile, with a temperature of 38.3°C. Chest auscultation revealed signs of consolidation with bronchial breathing and rales over the left base posteriorly. A chest roentgenogram disclosed consolidation over the left lower lobe, involving mainly the anterior and lateral basal segments, as well as prominence

---

From the Department of Medicine, Coney Island Hospital, Brooklyn, NY, and the Department of Medicine, State University of New York Health Science Center at Brooklyn.

Address correspondence to Dr Padmanabhan, Associate Chief, Department of Medicine, Coney Island Hospital, 2601 Ocean Pkwy, Brooklyn, NY 11235.

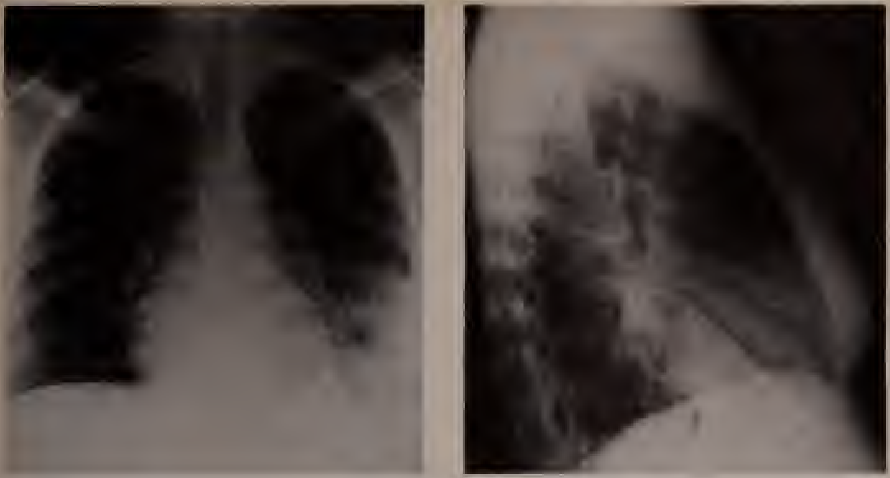


FIGURE 3. **Left**, posteroanterior roentgenogram showing consolidation in the left lower lobe; **Right**, lateral view showing involvement of anterior and lateral segments of left lower lobe (Case 3).

of the left hilum (Fig 3). The peripheral leukocyte count was 14,500/mm^3 with 80% polymorphonuclear leukocytes. Gram's stain and Ziehl-Neelsen stain of sputum revealed no organisms. A presumptive diagnosis of psittacosis was made, and intravenous erythromycin therapy was started.

During the subsequent four weeks, the fever gradually subsided, but the cough, left-sided pleuritic chest pain, signs of consolidation, and roentgenographic abnormalities persisted. During this time, sputum cultures grew *M tuberculosis*. A skin test using PPD (5Tu) was positive (15 mm). Treatment with isoniazid, rifampin, and ethambutol was started, with rapid resolution of symptoms. A computed tomographic scan of the thorax done postpartum revealed left hilar and subcarinal adenopathy as well as parenchymal disease in the left lower lobe. A repeat roentgenogram four months later revealed complete resolution of lower lobe consolidation and the left hilar adenopathy.

DISCUSSION

The incidence of tuberculosis has been declining in the United States over the last four decades.[1] In 1974, the number of new cases reported nationwide was 30,210—an incidence 46% lower than a decade before.[2] Figures from the Centers for Disease Control in Atlanta indicate that the number of new cases reported in 1985 in the United States declined by 27% to 21,801. A similar decline in the incidence of the disease was reported in New York City up to 1980.[3] In 1964, the number of new cases reported in the city was 4,207.[3] This number decreased to 2,022 new cases in 1974, and to 1,514 new cases in 1980.[3]

During the same time, responsibility for diagnosing and treating tuberculosis has shifted from the specialist in the sanitorium to the internist and family physician in the general hospital. As a result, there may be a degree of unfamiliarity with the disease among physicians that results in misdiagnosis and delayed diagnosis. Khan et al[4] emphasized this in their review of the spectrum of pulmonary tuberculosis in the adult. Supporting evidence was provided by MacGregor[5] in 1972, who found that in an urban teaching hospital, 44% of the patients with pulmonary tuberculosis were initially misdiagnosed in spite of the fact that 75% of these patients had the characteristic presentation of upper lobe infiltrative or cavitary disease. This delay in diagnosis led to an average of 35 hospital workers being exposed to active tuberculosis. In a community hospital in 1971, Jacobs and Greenberg[6] found that in 19 of the 20 patients with tuberculosis, the diagnosis was not suspected on admission. The possibility of misdiagnosis can be expected to increase several fold when the presentation is less characteristic.

Acute tuberculous pneumonia is an unusual form of pulmonary tuberculosis. It can easily be mistaken for acute pyogenic bacterial pneumonia, as the patients often have high fever and show lobar consolidation on chest roentgenograms.[7] In children, tuberculous pneumonia is usually secondary to overwhelming primary infection. Tuberculous pneumonia in the adult, secondary to both primary and reactivation disease, has been described during pregnancy, in the elderly, and in blacks, diabetics, and alcoholics. In a review of primary pulmonary tuberculosis in the adult, Choyke et al[8] noted that 25% of the patients had homogeneous air space opacities with air bronchograms on chest roentgenogram, while 7.7% of the patients presented with an acute consolidative process. Khan et al,[4] reviewing the spectrum of pulmonary tuberculosis in the adult, reported a 6.8% incidence of lower lobe tuberculosis. In most of these patients, the presentation was like that of acute tuberculous pneumonia.

A review of newly diagnosed cases of active pulmonary tuberculosis in adults in this hospital during a 12-month period from June 1984 to June 1985 identified 26 patients with tuberculosis which was confirmed by growth of *Mycobacterium tuberculosis* in sputum or biopsied lung tissue. Three patients had acute lobar consolidation. Eighteen patients had the characteristic presentation of infiltrate alone or infiltrate and cavitation in the upper lobes. Three patients had disseminated disease. It is of interest that two patients had acquired immunodeficiency syndrome (AIDS) and had a more atypical presentation. One patient had perihilar infiltrates, and the other had a normal chest roentgenogram. This pattern of presentation of tuberculosis in AIDS has recently been described.[9] The increasing frequency of AIDS has widened the spectrum of tuberculosis in the adult. Mediastinal adenopathy and perihilar infiltrates, not cavitary disease, are the most common signs of tuberculosis in this disease.[9]

Tuberculous pneumonia in the adult is secondary to the discharge of caseous material containing tubercle bacilli into a main bronchus. This can result from a caseous lymph node eroding into and perforating the bronchial wall, or secondary to aspiration of caseous material from a tuberculous cavity.[10] The response to this discharge of caseous material is most often an exudative hypersensitivity reaction to the tuberculoprotein, with rapid development of lobar consolidation.[10,11] Austrian and Willis[12] demonstrated this response in rabbits by intratracheal administration of acid-fast bacilli. Larson and Long[13] demonstrated the same response in guinea pigs. The pathologic picture is characterized by a florid histiocytic and neutrophilic exudate. Transalveolar spread of tuberculoprotein elicits more of this response, and rapid progression of the consolidative process can occur. This has been referred to as collateral pneumonia.[10]

Less often, unlike the inflammatory exudative response, a slower granulomatous response to the tuberculoprotein may be seen. The alveoli become filled with solid plugs of epithelioid and giant cells forming a tubercle that is not spherical, but rather takes the shape of the alveolar tree. This has been described as epithelioid cell pneumonia.[10] In both of these situations, organisms are not plentiful, and failure to demonstrate tubercle bacilli on sputum smear is not uncommon.

Endobronchial involvement is commonly seen in tuberculous pneumonia either due to the erosion of a lymph node into the bronchial wall, or as a result of direct extension from the pneumonic process. The high incidence of endobronchial involvement makes bronchoscopy very successful in the early diagnosis of tuberculous pneumonia. Early diagnosis is likely, however, only when tuberculosis is included in the initial differential diagnosis of the pulmonary disease. Otherwise, a diagnostic delay of two to six weeks has been noted.[8,14] This was the case in the three patients described here. The diagnosis of tuberculosis was made in two to four days in the first two cases, in which tuberculosis was part of the initial differential diagnosis. In the third case, psittacosis was the accepted diagnosis, and a delay of four weeks ensued before the correct diagnosis was made.

In acute lobar pneumonia, especially one that does not respond to antibiotics, one must maintain a high index of suspicion of tuberculosis. If sputum examination does not reveal the tubercle bacillus, more invasive procedures such as bronchoalveolar lavage, transbronchial lung biopsy, or percutaneous lung aspiration are indicated to establish the diagnosis.

The importance of early recognition of tuberculosis cannot be over-emphasized, especially in view of the disturbing upward trend in the incidence of tuberculosis in New York City since 1981.[3,15] In 1983, 1,651 new cases of tuberculosis were reported in the city.[3] Preliminary figures from the Centers for Disease Control indicate that 1,910 new cases of tuberculosis were reported in New York City in 1985. The potential relationship between the increase in the incidence of tuberculosis in New York City and the current epidemic of AIDS is presently being investigated by the New York City Health Department.[15]

REFERENCES

1. *Tuberculosis Programs 1972*, US Dept of Health, Education, and Welfare, publication No. (CDC) 74-8189. Public Health Service, 1973, p 1.
2. Table 2(A), Reported cases of specified notifiable diseases, US 1965–1974. *MMWR* 1974; 23:2.
3. Bureau of Tuberculosis of the New York City Department of Health: *Tuberculosis in New York City 1983*. New York, New York Lung Association, 1985, p 2.
4. Khan MA, Kovnat DM, Bachus B, et al: Clinical and roentgenographic spectrum of pulmonary tuberculosis in the adult. *Am J Med* 1977; 62:31–38.
5. MacGregor RR: A year's experience with tuberculosis in a private urban teaching hospital in the postsanitorium era. *Am J Med* 1975; 58:221–228.
6. Jacobs S, Greenberg HB: Diagnosis and treatment of 20 tuberculous patients who entered a community hospital. *Am Rev Resp Dis* 1972; 105:528–531.
7. Schwartz WS, Moyer RE: Management of massive tuberculous pneumonia. Therapeutic review of 75 cases. *Am Rev Tuberc* 1951; 64:41–49.
8. Choyke PL, Sostman HD, Curtis AM, et al: Adult-onset pulmonary tuberculosis. *Radiology* 1983; 148:357–362.
9. Pitchenik AE, Rubinson HA: The radiographic appearance of tuberculosis in patients with acquired immune deficiency syndrome (AIDS) and pre-AIDS. *Am Rev Respir Dis* 1985; 131:393–396.
10. Rich AR: *The Pathogenesis of Tuberculosis*. Springfield, Ill, Charles C. Thomas, 1944, pp 828–830.
11. Septiumus EJ, Awe RJ, Greenberg SD, et al: Acute tuberculous pneumonia. *Chest* 1977; 71:774–775.
12. Austrian CR, Willis HS: Pulmonary effects of intratracheal injections of tubercle bacilli and blood in rabbits. Experimental study of factors responsible for roentgenographic changes following haemoptysis in pulmonary tuberculosis. *Am Rev Tuberc* 1926; 14:306–315.
13. Larson A, Long ER: Experimental tuberculin pneumonia. *Am Rev Tuberc* 1931; 23:41–44.
14. Stack BH: Diagnosis of tuberculosis in a general hospital. *Br Med J* 1971; 4:610–612.
15. Bureau of Tuberculosis of the New York City Department of Health: *Special Supplement to Tuberculosis in New York City 1983*, New York City Department of Health, June 1985.

See also pp 389–390.

Volvulus of the descending colon

JOHN S. O'SHEA, MD; ROBERT ZUBOWSKI, MD; JAMES C. DILORENZO, MD

Volvulus is a minor cause of large bowel obstruction, occurring in 10–15% of cases.[1,2,3(p 1150),4] Regardless of the segment of bowel involved, the anatomic arrangement necessary for a volvulus to occur is a freely mobile segment of bowel and a fixed point about which the bowel can rotate.[1,2,5-8]

Although usually relatively fixed in position, the descending colon may have a persistent mesentery.[9] Rarely, the mesentery is of sufficient length to allow a freely mobile segment of descending colon to undergo torsion, causing acute or recurrent obstruction. We believe this to be the first English language report of a patient who required surgical intervention for a descending colon volvulus, with only one previous report appearing in the world literature, in French.[10]

CASE REPORT

A 28-year-old man presented with a five-year history of almost daily episodes of discomfort in the left upper quadrant of the abdomen, associated with audible bowel sounds. The patient stated that he would commonly massage this area of the abdomen, which would "break the gas into small bubbles," allowing it to pass through the rectum. This process would usually, but not always, relieve his symptoms. His bowel movements were regular and did not contain blood or mucus. His medical history was unremarkable.

An abdominal examination was noncontributory. He was seen by a gastroenterologist, who performed colonoscopy, but was able to advance only to the upper descending colon, where there appeared to be a redundant loop. No mucosal lesions were noted. A barium enema revealed a redundant "C" loop in the descending colon (Figs 1, 2). Computed tomography revealed no intra-abdominal disorders.

The patient underwent exploratory laparotomy, which disclosed a redundant loop of descending colon. Some filmy adhesions were noted between the apex of the redundant loop and the small bowel mesentery. The mesentery corresponding to the redundant large bowel was somewhat thickened

---

From the Department of Surgery, Our Lady of Mercy Medical Center, Bronx, NY.

Address correspondence to Dr O'Shea, 613 East 233rd St, Bronx, NY 10466.

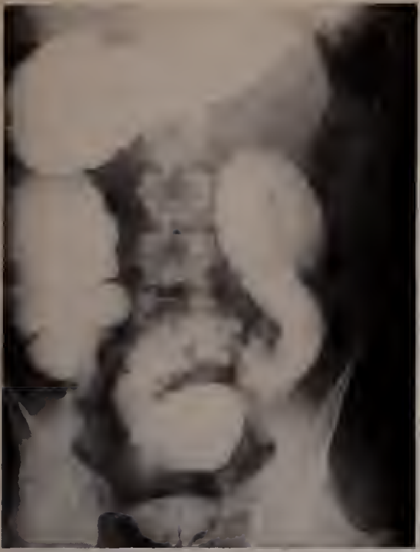


FIGURE 1. Barium enema examination showing redundant loop of descending colon.

and scarred, the intestine itself being grossly normal. A nodule adjacent to the bowel was identified by frozen section as fat necrosis. The rest of the abdominal exploration was within normal limits. The redundant loop of colon was mobilized and resected, with a primary end-to-end anastomosis being done between the proximal and distal descending colon. The patient was discharged from the hospital on the sixth day following surgery. Pathologic examination of the specimen revealed no gross abnormality of the bowel and, other than prominent submucosal lymphoid aggregates, the microscopic examination was unremarkable as well.

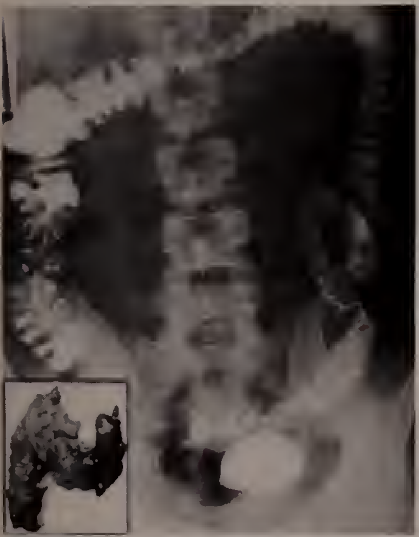


FIGURE 2. Post-evacuation barium enema showing persistent loop in descending colon. Insert, resected loop of descending colon corresponding to that seen on barium enema.

DISCUSSION

Volvulus of the colon accounts for approximately 5–10% of acute large bowel obstructions.[1–4] The usual site of colonic volvulus is the sigmoid colon. Less frequently involved areas are the cecum and ascending colon[3–5,11] and, rarely, the transverse colon or splenic flexure.[6–8]

In surveying the literature, we found only one previous case of a patient who underwent surgical treatment of a descending colon volvulus.[10] That patient was also a 28-year-old man, but unlike the patient presented here, he did not give a history of antecedent abdominal symptoms. He presented with an acute large bowel obstruction which was successfully managed by resection of the redundant segment of descending colon. In that same report, the authors presented two patients who underwent barium enema examinations as part of a work-up for vague abdominal complaints. Both patients were found to have radiologic evidence of a redundant loop in the descending colon, though neither patient had undergone surgery at the time of the report.

Nice and Puyau[12] mention the subject of descending colon volvulus and give as an example the findings of a barium enema that may represent a volvulus of the splenic flexure or upper descending colon. The authors provide no references or surgical follow-up, however.

The anatomic requirements that allow a volvulus to occur, regardless of location, are a redundant or freely mobile segment of bowel and a fixed base point or points about which the loop can rotate.[1,2,5–8]

The descending colon is usually invested anteriorly and laterally by peritoneum, with its posterior surface being relatively fixed in the retroperitoneum. However, some persistence of a descending mesocolon has been noted in as many as 36% of subjects studied,[9] though rarely is it of sufficient length to allow a freely mobile segment. Anderson[13] reported the unusual case of a patient who underwent nonoperative decompression of a volvulus which changed position on radiologic evaluation from splenic flexure to sigmoid, possibly related to a very mobile descending colon.

The patient described here did not present with an acute obstruction, but had a history consistent with intermittent volvulus. At surgery, he also had the gross findings of a redundant loop and a thickened, scarred, corresponding mesentery, which, when found in the sigmoid, has been felt to be characteristic of recurrent volvulus.[14–16] By massaging the abdomen, the patient may have been accomplishing nonoperative detorsion, thus preventing progression of the obstruction.

As with recurrent volvulus at other sites, resection of the involved segment is the recommended treatment.[3(p1153),14,15] This patient has done well following surgery.

REFERENCES

1. Kerry RL, Ransom HK: Volvulus of the colon. *Arch Surg* 1969; 99:215–222.
2. Arnold GJ, Nance FC: Volvulus of the sigmoid colon. *Ann Surg* 1973; 177:527–537.
3. Goligher J: *Surgery of the Anus, Rectum and Colon*. London, Balliere Tindall, 1984, p 1150.
4. Ballantyne GH: Review of sigmoid volvulus. History and results of treatment. *Dis Colon Rectum* 1982; 25:494–501.
5. Villet DD: Volvulus of the proximal colon. *Ann Surg* 1959; 150:1075–1085.
6. Gumbs MA, Kashan F, Shumofsky E, et al: Volvulus of the transverse colon. Reports of cases and review of the literature. *Dis Colon Rectum* 1983; 26:825–828.
7. Welch GH, Anderson JR: Volvulus of the splenic flexure of the colon. *Dis Colon Rectum* 1985; 28:592–593.
8. Goldberg M, Lernau OZ, Mogle P, et al: Volvulus of the splenic flexure of the colon. *Am J Gastroenterol* 1984; 79:693–694.
9. Treves VF: Anatomy of intestinal canal and peritoneum in man. *Brit Med J* 1965; 1:580–583.
10. Couinaud C, Fabre X, de Margerie V: Une urgence exceptionelle: Le volvulus du colon descendant. *Chirurgie* 1979; 105:672–674.
11. Peterson HI: Volvulus of the cecum. *Ann Surg* 1967; 166:296–299.
12. Nice CM, Puyau FA: The large bowel, part I: Nonneoplastic lesions, in Teplich JG, Haskin ME (eds): *Surgical Radiology*. Philadelphia, WB Saunders Co, 1981, pp 663–666.
13. Anderson MF, Pickney L: The traveling volvulus. *Dis Colon Rectum* 1979; 22:276–278.
14. Smith RB, Kettlewell MG, Gough MH: Intermittent sigmoid volvulus in the younger age groups. *Br J Surg* 1977; 64:406–409.
15. Sturzaker HG, Lawrie RS, Joiner CL: Recurrent sigmoid volvulus in young people: A missed diagnosis. *Br Med J* 1975; 4:338–339.

Endotracheal hamartoma

JENNIFER E. ALEXANDER, MD, RICHARD BRODMAN, MD

Primary tracheal tumors in general and tracheal hamartomas in particular are rare. Our literature search has identified only four published case reports of tracheal hamartomas. We describe here a patient who presented with upper airway obstruction that was subsequently diagnosed as an endotracheal hamartoma. The literature on tracheal hamartomas is reviewed.

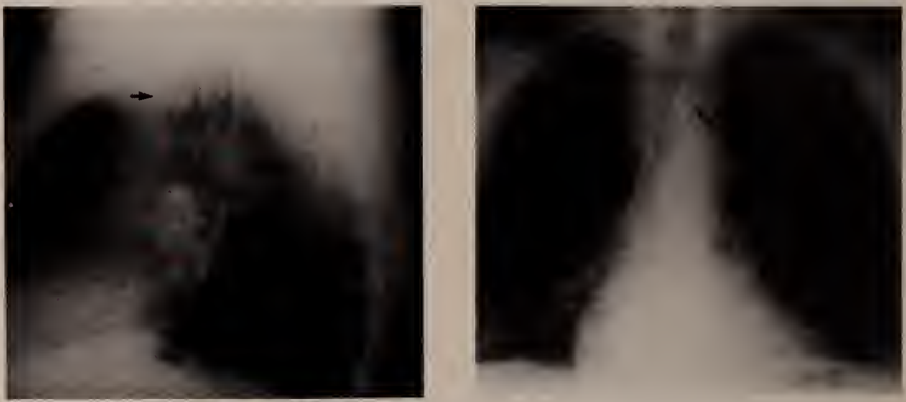


FIGURE 1. **Left,** lateral chest film; **Right,** posteroanterior chest film; arrows show tracheal mass.

CASE REPORT

A 48-year-old man was admitted to Montefiore Medical Center with a nine-month history of shortness of breath and a productive cough. There was no associated fever, pleuritic pain, hemoptysis, or weight loss. The patient had a 42-pack/year smoking history, but an otherwise unremarkable medical history. He had been seen one month prior to this admission at another hospital. At that time, chest roentgenograms showed a nodular density in the left side of the trachea (Fig 1). Tomograms defined an oval-shaped nodule on the anterolateral aspect of the trachea, 4 cm proximal to the carina (Fig 2). No mass was noted in the lung parenchyma. A computerized axial tomographic (CAT) scan was also obtained (Fig 3). Flexible bronchoscopy at the other institution revealed a polypoid tumor mass occluding one half of the trachea. The biopsy was superficial and consisted of only inflamed, hyperplastic, and metaplastic respiratory mucosa.

The patient was subsequently referred to Montefiore Medical Center, where an attempt at fulguration of the mass with a CO_2 laser was unsuccessful because the tumor was relatively firm. A surgical approach was then used. Through a right thoracotomy, the mediastinal trachea was easily mobilized around the palpable intratracheal mass. The trachea was opened, and a section of contiguous trachea containing a tracheal ring was resected with the broad-based tumor mass, avoiding morcellation of the large mass and enabling assessment by the pathologist of the extent of submucosal extension. A diagnosis of hamartoma was made by frozen section. Further tracheal resection was unnecessary.

Pathologic Findings. Gross examination of the hamartomatous specimen disclosed a ring of trachea containing a submucosal, broad-based polypoid mass measuring 3 × 2 × 2 cm (Fig 4). The cut surface revealed tan colored, multilobulated, mucoid-appearing tissue with foci of calcification. The lesion was discrete, did not arise from the subjacent tracheal ring, and appeared to be completely excised. On microscopic evaluation, the lesion was covered by intact hyperplastic respiratory epithelium, exhibiting focal squamous metaplasia. The lesion was not encapsulated and was composed of numerous irregular lobules of hyaline cartilage in various stages of maturation, mixed with loose, primitive-appearing mesenchymal tissue and adipose tissue. A gradual transition of the primitive mesenchymal tissue into adipose tissue, as well as some lobules of cartilage, were observed. A solitary focus of metaplastic bone formation was also noted (Fig 5).

The patient made an uneventful recovery and is without evidence of tumor after nearly two years.

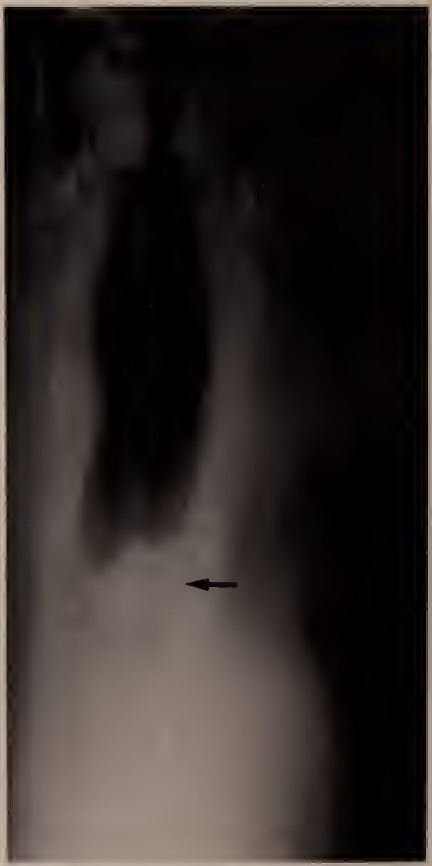


FIGURE 2. Tomogram showing tracheal mass.

DISCUSSION

Benign tumors of the trachea include papillomas, inflammatory fibrous pol-

---

From the Departments of Pathology and Surgery, Cardiothoracic Surgery Division, Montefiore Medical Center and The Albert Einstein College of Medicine, Bronx, NY.

Address correspondence to Dr Alexander, Department of Pathology, Montefiore Hospital and Medical Center, 111 E 210th St, Bronx, NY 10467.

TABLE I. Reported Cases of Tracheal Hamartoma

Sex	Age (yr)	Clinical Symptoms	Clinical Diagnosis	Diagnostic Aids	Treatment	Follow-up Without Recurrence	Author
M	48	Cough, expectoration	—	Chest film, tomograms, CAT scan, bronchoscopy	CO_2 laser, surgical resection	22 mos	Present case
M	51	Cough, expectoration, dyspnea	Asthma	Bronchoscopy	Surgical resection	4 yrs	Engelking
M	75	Cough, fever, expectoration	Chronic obstructive pulmonary disease	Lateral film, bronchoscopy	Surgical resection	5 mos	Hurst & Nelson
M	34	Cough	Bronchitis	Chest film, tomograms, bronchoscopy	Cautery	9 mos	Kaneko et al
M	47	Dyspnea	Asthma	Lateral film, tomograms, bronchoscopy	Multiple punch	2½ yrs	Kim et al

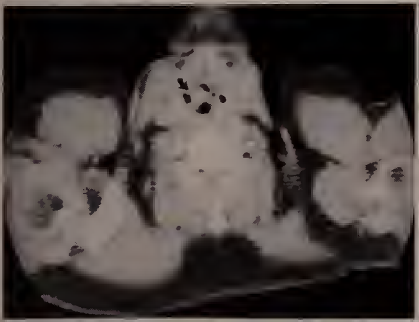


FIGURE 3. CAT scan showing tracheal mass.

yps, and hamartomas.[1] By definition, hamartomas are abnormal configurations of a variety of benign tissues, presumably derived from primitive connective tissue. The tumor reported above fulfills these criteria. We are aware of only four cases of tracheal hamartoma reported in the English literature. Table I summarizes the published cases, including the present one.

The first case of tracheal hamartoma reported in the English language literature was published by Engelking,[2] who described a 51-year-old man with a long history of asthma, who eventually was shown to have a tracheal hamartoma. This patient's pseudoasthmatic condition improved after surgical resection of the lesion. Similarly, subsequently reported cases were all men with varying degrees of upper airway obstruction manifesting clinically as chronic cough, shortness of breath, and other symptoms of bronchitis and/or asthma.[3-5] These patients received medical treatment for some time, until the correct diagnosis was made.

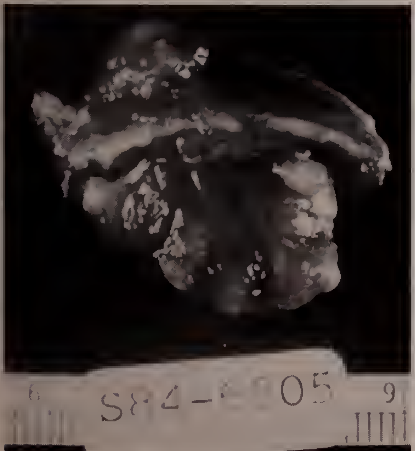


FIGURE 4. Gross surgical specimen.

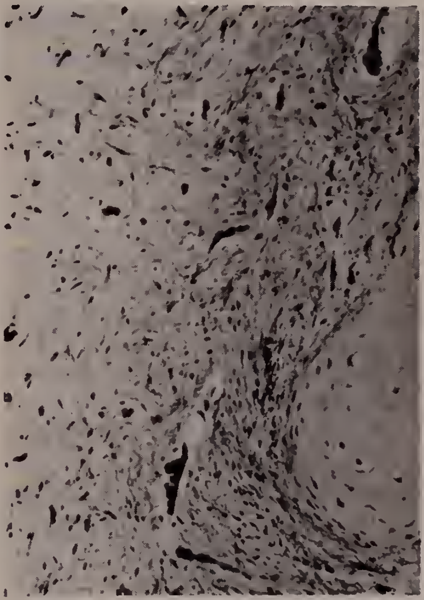


FIGURE 5. Photomicrograph showing metaplastic bone formation (lower right); original magnification × 125, hematoxylin-eosin stain.

Most symptoms of tracheal hamartoma are due to mechanical obstruction, and because of the slow growth of the tumors, symptoms are usually present for many months. The earliest presenting symptom may be dyspnea,[2,5] which may be constant or paroxysmal. The most common symptoms, however, were chronic cough[1-5] with expectoration, which may eventually progress to subtotal upper airway obstruction and chronic obstructive pulmonary disease. An early and correct diagnosis requires a high index of suspicion in patients with histories suggestive of chronic bronchitis, tracheitis, or asthma, but whose conditions are relatively refractory to medical treatment. Under these circumstances, when a tracheal mass is diagnosed the differential diagnosis includes malignant neoplasms and benign obstructing lesions. An adequate biopsy may be helpful in planning surgery. When the tumor appears resectable, biopsy is best done at bronchoscopy under the general anesthesia used for the definitive resection.[6] In the present case, inadequate sampling precluded an accurate preoperative diagnosis.

All reported patients completely recovered after surgery without recurrence of their tumors. Of some interest is the failure of a CO_2 laser to fulgurate this tumor. No report of a previous CAT scan of an endotracheal hamartoma was found in the literature.

Acknowledgments. The authors thank A. Foster, N. Sanders, and the staff at the Department of Pathology, Montefiore Hospital.

REFERENCES

1. Caldarola VT, Harrison EG Jr, Clagett OT, et al: Benign tumors and tumorlike conditions of the trachea and bronchi. *Ann Otol Rhinol Laryngol* 1964; 73:1042-1061.
2. Engelking CF: Hamartoma of trachea. Report of a case, with a review of the literature of benign trachea neoplasms. *Laryngoscope* 1959; 69:1278-1286.
3. Hurst IJ Jr, Nelson KG: Tracheal hamartoma. *Chest* 1977; 72:661-662.
4. Kaneko H, Uemura T, Kobayashi R, et al: A case of tracheal hamartoma. *Otologia Fukuoka* 1978; 24(suppl 2):723-727.
5. Kim SK, Cho BK, Park CI, et al: Tracheal hamartoma causing unique stridor and a review of the literature. *Yonsei Med J* 1982; 23:153-158.
6. Grillo HC: Management of tracheal tumors. *Am J Surg* 1982; 143:697-700.

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Medicaid recordkeeping requirements and professional misconduct

TO THE EDITOR: In February 1982, "Dr S" signed a stipulation with the Department of Social Services under which he agreed to reimburse the Department $14,638 in disallowed Medicaid payments and to refrain from future participation in the Medicaid program. The basis of the payment disallowance was "insufficient documentation to fully disclose the extent of services and care provided."[1] "Dr S" thought his legal problems were over with his agreement to the stipulation and the payment of the fine. Unfortunately, his problems had just begun.

In 1986, the stipulation was directly referred to a Regents Review Committee of the New York State Board of Regents pursuant to Public Health Law §230(10)(m)(iv). This section, which was enacted in 1986, provides that in cases of professional misconduct solely based upon the conviction of a crime, or upon a finding of a violation of a statute or regulation pursuant to a final determination of an administrative agency, and where the violation resulting in the final agency determination would constitute professional misconduct, charges may be prepared and the matter referred directly to the Regents Review Committee. That committee's function is to review the prior findings and determinations of guilt, and to recommend the measure of discipline to be imposed. In a hearing before the Regents Review Committee, only evidence and testimony relating to the nature and severity of the penalty to be imposed on the licensee may be offered. Following the Regents Review Committee's recommendation, final action is taken by the Board of Regents.

This procedure contrasts with the procedures generally followed in professional misconduct proceedings, which involve an investigation by the director of the Office of Professional Medical Conduct, a hearing before a committee of the board for professional medical conduct, a review and recommendation by the Commissioner of Health, and a review and final decision by the New York State Board of Regents.[2] Apparently, the rationale for the attenuated procedures followed in the case of convictions or final agency determinations of violations of statutes or regulations is that since the underlying conduct constituting the crime or violation has already been established, the professional misconduct proceeding need not again address the matter, but may proceed directly to recommending the appropriate measure of discipline to be imposed.

In the instant case, the direct referral to the Regents Review Committee was based on the contention that the stipulation that there was insufficient documentation to entitle the physician to Medicaid reimbursement was equivalent to a stipulation that he had violated a rule or regulation, which also constitutes professional misconduct. A brief filed with the Department of Health by the attorney for "Dr S" contended that the direct referral to the Regents Review Committee pursuant to Public Health Law §230(10)(m)(iv) was improper in that the statute should not apply to an administrative proceeding which occurred prior to the effective date of the statute. It also contended that even if the statute were found to have retroactive effect, a referral to the Regents Review Committee would be improper because there are no facts in the stipulation that could be construed as constituting professional misconduct.

As of the date of this writing, the case of "Dr S" remains pending. Regardless of the outcome of this case, however,

TABLE I. New York State Department of Social Services Regulations Concerning Minimum Criteria for Medical Records

. . . In the case of bills for physician services, physicians are required to maintain complete, legible records in English for each patient treated. Medical records shall include at a minimum, but not be limited to, the following:

(i) the full name, address and medical assistance program identification of each patient examined and/or treated in the office for which a bill is presented;

(ii) the date of each patient visit;

(iii) the patient's chief complaint or reason for each visit;

(iv) the patient's pertinent medical history as appropriate to each visit, and findings obtained from any physical examination conducted that day;

(v) any diagnostic impressions made for each visit;

(vi) a recording of any progress of a patient, including patient response to treatment;

(vii) a notation of all medication dispensed, administered or prescribed, with the precise dosage and drug regimen for each medication dispensed or prescribed;

(viii) a description of any X-rays, laboratory tests, electrocardiograms or other diagnostic tests ordered or performed, and a notation of the results thereof;

(ix) a notation as to any referral for consultation to another provider or practitioner, a statement as to the reason for, and the results of such consultations;

(x) a statement as to whether or not the patient is expected to return for further treatment, the treatment planned, and the time frames for return appointments;

(xi) a chart entry giving the medical necessity for any ancillary diagnostic procedure; and

(xii) all other books, records and other documents as are necessary to fully disclose the extent of the care, services and supplies provided.

this experience demonstrates how insufficient documentation of the extent of services provided to Medicaid patients may not only involve financial sanctions and possible exclusion from participation in the Medicaid program, but may also later result in a referral to a professional misconduct proceeding involving possible licensing sanctions.

Physicians should keep detailed medical records relating to patient care in all cases. With specific regard to Medicaid cases, the Department of Social Services has promulgated a regulation, 10 NYCRR §540.7(a)(10)(see Table I), which outlines the minimum criteria for medical records. Such records should be kept for a period of not less than six years.[3] Keeping in mind the possible sanctions for inadequate records, the regulation should be carefully considered.

DONALD R. MOY, JD
General Counsel
Medical Society of the
State of New York
420 Lakeville Rd
Lake Success, NY 11042

---

1. 18 NYCRR §515.2(b)(ii); 18 NYCRR §540.7-(a)(8).
2. Public Health Law §230(10).
3. 18 NYCRR §540.7(a)(8); 8 NYCRR §29.2-(a)(3).

Homelessness in Paris

TO THE EDITOR: We read with great interest the research paper and commentaries on the homeless in New York City published in the January issue of the *Journal*.[1-3] Some of the issues covered concerning homelessness are similar to those in Paris, although each city has its unique features. In all major cities the plight of the homeless gets worse during the winter months. By definition the homeless are hard to census, but one can come up with rough estimates. The Paris figure for this winter (1986-1987) stood at about 12,000 out of a population of 2.5 million, representing 4.8 homeless per 1,000 population, compared to 42,500 in New York City, representing 6.0 homeless per 1,000 population.[4,5] Paris' homeless population is primarily made up of individuals suffering from alcoholism who are usually unemployed. The rise in unemployment is a prime factor in increasing the incidence of homelessness in Paris. The percentage of drug addicts is climbing proportionally, but the level remains relatively low. The percentage of homeless suffering from severe and persistent mental disorders has remained stable over the last ten years or so (under 20%). This greatly differs from New York City, where the proportion of the homeless suffering from chronic mental disorders is growing. That figure stands at 50% (as opposed to 40% alcoholics and 10% drug addicts).[2] In Paris, the majority of the mentally ill are cared for in mental hospitals.

Where do the homeless in Paris find refuge? Public and private welfare institutions such as the Salvation Army, the Red Cross, the EMMAUS Association, and the Nicolas Flamel Center have opened up emergency aid centers since the start of the winter. They provide some 3,000 beds for the homeless. In addition to this, two Metro stations stay open all night, and ten churches have opened their doors to the homeless. These premises are in fact not overcrowded. Many homeless actually remain without a roof over their heads, living hidden away, apart from any social structure—their numbers figments of statistical guesswork. This is partly because these individuals are not reached by the media and thus remain unaware of the existence of the new shelters available to them. Also, some hold back from using this kind of refuge—out of a sense of dignity, or reluctance to take a shower, or through fear of the violence that sometimes occurs in such places. Others are afraid of losing their own private shelters in some empty garret, cellar, corner of the subway, or hideaway in an unused railway siding or subway depot, etc. All these nooks and crannies represent uncharted places of refuge in Paris, where thousands of unknown inhabitants dwell in silence, out of the way of the media.

The characteristics of the homeless in Paris and New York City differ considerably, and city-specific solutions will be required to solve the problems. Meanwhile, the problem of the homeless in both cities is similar in that permanent solutions have yet to be found.

JEAN-MARC NORES, MD
University of Paris
Department of Internal Medicine
Hopital Raymond Poincaré
Garches 92380, France

JEAN DALAYEUN, MD
University of Paris
Hopital Suisse de Paris
Issy-Les-Moulineaux 92130, France

---

1. Chavkin W, Kristal A, Seabron C, et al: The reproductive experience of women living in hotels for the homeless in New York City. *NY State J Med* 1987; 87:10-13.
2. Imperato PJ: New York's homeless. *NY State J Med* 1987; 87:1-3.
3. Vermund SH, Belmar R, Drucker E: Homelessness in New York City: The youngest victims. *NY State J Med* 1987; 87:3-5.
4. *The World Almanac and Book of Facts 1987*. New York, World Almanac 1987, pp 247, 570.
5. *Homelessness in New York State*. Albany, New York State Dept of Social Services, 1984, vol 1, pp 6-8, 10.

Continuing use of x-ray pelvimetry for pregnant women

TO THE EDITOR: Some physicians in New York State still employ x-ray pelvimetry of pregnant women, despite serious questions about the efficacy and safety of the procedure.

Although the reported use of x-ray pelvimetry declined sharply in New York State in 1982-1983, reports to the Statewide Planning and Research Cooperative System (SPARCS) suggest that the decline has now halted and may actually have reversed. We believe that the continuing—and perhaps increasing—use of this controversial procedure merits the attention of the medical community.

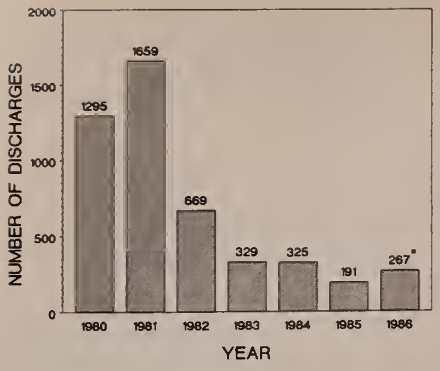
The SPARCS data bank, from which our information is drawn, contains data on essentially all stays in acute care hospitals in New York State, other than psychiatric and federal government hospitals. Approximately 2.8 million discharge data abstracts are collected each year. The collections for 1980-1985 are complete; the collection for 1986 is about 67% complete.

X-ray pelvimetry was identified on the discharge data abstracts by the ICD-9-CM procedure code 88.25. Where this code appeared, we assumed that the procedure had been performed. No hospital records were audited to verify that fact.

In each year more than 90% of the reported x-ray pelvimetries were performed on pregnant women, and approximately 60% of the procedures were associated with cesarean sections.

The trend in the reported use of x-ray pelvimetry in New York State since 1980 is illustrated in Figure 1. Sharp declines in 1982 and 1983 probably reflect a position statement developed in 1979 and issued formally in 1980 by an expert panel of obstetricians and radiologists convened by the US Food and Drug Administration (FDA).[1] When the statement was publicized in 1981 by a notice in the *FDA Drug Bulletin*,[2] it elicited a widespread response.

The FDA panel cautioned that x-ray pelvimetry "is not usually necessary or helpful in making the decision to perform a Cesarean section." In the "few instances" where the physician deter-



* Partial data; hospital reports to SPARCS for 1986 are about 67% complete at the time of this writing.

FIGURE 1. Number of New York State hospital discharge data abstracts including diagnostic x-ray pelvimetry reported to SPARCS, 1980–1986.

mines that the procedure is appropriate, "the reason for requesting the pelvimetry should be written on the patient's chart."

The FDA statement was endorsed in 1979 by the American College of Radiology,[3] and a similar statement was issued by the American College of Obstetricians and Gynecologists:[4] "X-ray pelvimetry provides limited additional information to physicians involved in the management of labor and delivery. It should not be a prerequisite to clinical decisions concerning obstetrical management. Reasons for requesting x-ray pelvimetry should be individually established."

These statements and the attendant publicity focused attention on an increasing body of literature on the risks and limitations of x-ray pelvimetry. Most, though not all, of these reports indicated that fetuses exposed to radiation are at increased risk for leukemia and other malignancies, and that no significant correlation exists between measurements of cephalopelvic disproportion and the need for cesarean section.

More widespread awareness of these reports, in addition to the FDA panel's statement, may account substantially for the sharp decline in reported procedures in New York State in 1982 and 1983. By 1984 and 1985, however, this decline was tapering off, and in 1986 it appears to have been reversed. SPARCS data for approximately two-thirds of the 1986 hospital discharges (all that are now available) show reports of 267 discharges with pelvimetry, sharply exceeding the total for 1985. The extrapolated total of about 400 reports for 1986 would exceed the total for any previous year since 1982.

The x-ray pelvimetries in the SPARCS data bank are a minimum count and demonstrate the fact, rather than the magnitude, of continuing use of this controversial procedure. The discharge data abstracts submitted by the hospitals to SPARCS are prepared under Uniform Hospital Discharge Data Set (UHDDS) guidelines, which until 1986 stated explicitly that reporting of x-ray pelvimetry was optional, not required. Changes in these guidelines effective on January 1, 1986,[5] removed this explicit exemption, but the impact on reportability is not yet clear.

We recognize that there may be several explanations for the apparent rise in the use of x-ray pelvimetry in 1986. The shift in UHDDS guidelines may have led to increased reporting of the procedure during 1986, independent of any change in its frequency of use. Alternatively, the lapse of time since the FDA panel's statement may have made the statement less familiar and therefore less widely effective.

The FDA statement itself has not been uniformly accepted, nor have the studies on which that statement was based. Klapholz,[6] among others, has interpreted the earlier data in ways more favorable to third-trimester x-ray pelvimetry, although he concludes that its application should be "judicious and infrequent." Morgan et al[7] have found that x-ray pelvimetry is valuable in assessing cephalopelvic disproportion when used in association with ultrasound measurement of the fetal abdomen (as an indicator of fetal weight). The influence of such reports on physicians' decisions to use x-ray pelvimetry is unknown.

Meanwhile, most research studies and clinical observations continue to support the FDA panel's position. Harvey et al,[8] for example, reported in 1985 that in a case-control study of more than 32,000 twins, "when birth weight was adjusted for, twins in whom leukemia or other childhood cancer developed were twice as likely to have been exposed to x-rays in utero as twins who were free of disease."

On the issue of efficacy, Anderson[9] showed in 1983 that "even in a select group of patients in abnormal labor with the highest probability of CPD—a condition it was believed x-ray pelvimetry could diagnose—no significant difference in bony pelvic dimensions existed between vaginal and cesarean delivered infants."

In any case, he added, "the diagnosis of CPD can be confirmed only when the fetal head fails to descend despite effective uterine action. Thus, an adequate trial of good-quality labor and the lack of progress of cervical dilatation and fetal descent are the requirements for the diagnosis. Short of these observations, the diagnosis of CPD is only an assumption."

Johnson[10] in 1986, reviewing other studies and the advent of new diagnostic modalities with no risk of ionizing radiation, concluded that "pelvimetry in the management of labor is occasionally useful. However, in the face of apparent broad clinical consensus, routine pelvimetry by any technique no longer should be considered a 'standard of care' in the management of labor and delivery."

We believe that 30 years of cautionary reports on in utero exposure to x-rays, beginning with the major study by Stewart et al[11] in 1958, should not be disregarded. In the absence of clearly contradictory data, the continued—and perhaps increasing—use of this procedure is a matter of medical concern. In any case where the benefit to the patient is felt to clearly outweigh the risk, an explanatory note justifying the use of x-ray pelvimetry should be entered on the patient's chart.

Acknowledgment. The bar chart was prepared by Mr Barry Krawchuk.

ALFRED GELLHORN, MD
ALAN LEVENSOHN, PhD
GERALD KAUFMAN, PhD
LAURA IWAN, MS
New York State
Department of Health
Albany, NY 12237

1. US Dept of Health and Human Services: *The Selection of Patients for X-ray Examinations: The Pelvimetry Examination.* Washington, DC, 1980.
2. Statement on use of pelvimetry x-ray examination. *FDA Drug Bull* 1981; 11:22.
3. *ACR Bulletin* 1979; 35(10):2.
4. *ACOG Newsletter* 1979; 23(10-11):1.
5. Health Information Policy Council: 1984 Revision of the Uniform Hospital Discharge Data Set. *Federal Register* 1985; 50(31 July):31038-31040.
6. Klapholz H: Evaluation of fetopelvic relationships, in Cohen WR, Friedman EA (eds): *Management of Labor.* Baltimore, University Park Press, 1983, pp 25-40.
7. Morgan MA, Thurnau GR, Fishburne JI Jr: The fetal-pelvic index as an indicator of fetal-pelvic disproportion: A preliminary report. *Am J Obstet Gynecol* 1986; 155:608-613.
8. Harvey EB, Boice JD Jr, Honeyman M, et al: Prenatal x-ray exposure and childhood cancer in twins. *N Engl J Med* 1985; 312:541-545.
9. Anderson N: X-ray pelvimetry: helpful or harmful? *J Fam Pract* 1983; 17:405-412.
10. Johnson GC: Pelvimetry revisited. *Am J Radiol* 1986; 147:409-411.
11. Stewart A, Webb J, Hewitt D: A survey of childhood malignancies. *Brit Med J* 1958; 1:1495-1508.

Massive pulmonary embolism with right atrial thrombus

TO THE EDITOR: Right atrial clots have been classified as being either immobile or mobile.[1,2] The former originate in the atrium, whereas the latter probably originate in the peripheral veins and get lodged in the right atrium or ventricle.[2] We report a case of a mobile atrial thrombus, demonstrated by echocardiography, in a setting of pulmonary thromboembolism.

Case Report. A 64-year-old man was admitted to the hospital for a left total knee replacement. His medical history was significant for mild hypertension that had been treated and a 30-pack/year history of cigarette smoking. On the second day after surgery, hematemesis developed, and endoscopy showed an actively bleeding prepyloric ulcer. On the ninth day after surgery, the patient complained of dyspnea. Significant physical findings at this time included a heart rate of 96 beats/min with premature beats; blood pressure, 120/90 mm Hg; and respiratory rate, 24/min. There was a grade II/VI systolic murmur at the left sternal border, a right-sided S4 gallop, and accentuated P2 sounds. The lungs were clear to auscultation. There was a 1+ right ankle edema. An electrocardiogram showed sinus tachycardia.

Arterial blood gas studies at that time disclosed a Po_2 of 49 mm Hg; pH, 7.47; and Pco_2, 32 mm Hg. A ventilation perfusion lung scan showed a high probability of pulmonary embolism.

Venograms demonstrated filling defects in both lower legs. The patient was started on heparin on the 11th day after surgery. That evening, atrial fibrillation developed, with a rapid ventricular response which converted to normal sinus rhythm following intravenous digoxin and verapamil therapy. The next day, a two-dimensional echocardiogram showed a right atrial mass of irregular echoes, with no attachment to the interatrial septum, which had prolapsed through the tricuspid valve (Fig 1).

On the 13th day after surgery, the patient became confused, the dyspnea worsened, and brief episodes of apnea occurred. Repeat arterial blood gas studies showed increasing hypoxemia. Adequate anticoagulation had not been achieved. The patient was transferred to another institution for possible thrombectomy and inferior vena cava umbrella placement. Because of his critical condition, he was observed for six days and placed on intravenous heparin. His condition improved. A repeat ventilation perfusion scan showed some clearing of the defects, but the right atrial thrombus remained visible on echocardiogram. An ultrasound examination of the inferior vena cava was normal. Ten days after the initial pulmonary embolism, sudden dyspnea developed, and the patient suffered fatal cardiac arrest. A limited autopsy showed a saddle embolus of the main pulmonary artery having the "configuration of a leg vein." No clots or tumors were seen either near the inferior vena cava or attached to the interatrial septum.

Discussion. The patient described here died despite ten days of heparin therapy with adequate anticoagulation and objective signs of improvement. Previously published reports[2-15] also indicate that pulmonary emboli due to right atrial thrombi carry a poor prognosis. For cases such as these, two-dimensional echocardiography is an accurate, noninvasive test. In deciding who should have the test, signs of right-sided heart failure or tricuspid insufficiency can be important clues.[2,3]

Different approaches have been taken for treating right atrial thrombi. In one case,[4] there was an unsuccessful attempt at removing the clot using a Greenfield pulmonary embolectomy catheter. In other cases, surgery was accomplished with generally good results.[2,5-7] A recent review by Armstrong[8] identified 20 cases of right atrial thrombus and pulmonary embolism. Of these 20 patients, five died without benefit of specific therapy; of eight patients treated medically, four survived. The remaining seven patients underwent open heart surgery, and all but one survived. If one examines the eight medically treated cases, three of five given heparin or coumadin survived, whereas only one of three given streptokinase survived. Some authors even suggest that thrombolytic agents may cause harm by partially lysing the clot.[2,7,9] In another study, Felner et al[2] reported on six cases, including four with congestive cardiomyopathy and/or chronic cor pulmonale. Of three patients who had surgery, one died (of progressive right heart failure). The medically treated patients all survived (including one who recieved no therapy).

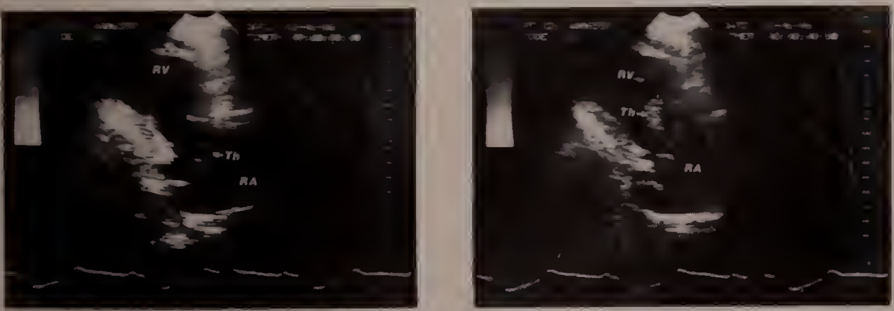


FIGURE 1. Images obtained with ATL MK 300c using a 3 megahertz transducer showing two long axis tricuspid veiws. **Left,** view during systole showing the clot in right atrium. **Right,** clot prolapsing into the right ventricle during diastole. RA, right atrium; RV, right ventricle; TH, thrombus.

Considering the general trend, it would appear that surgical removal of right atrial thrombi may be the treatment of choice for patients capable of undergoing thoracotomy. Further studies may better differentiate the benefits and risks of heparin versus streptokinase therapy for other cases. Patients undergoing inferior vena cava umbrella insertion should have echocardiograms beforehand to rule out the presence of a right atrial thrombus.

Acknowledgments. The authors thank Debbie Glanville and Anne Lagelante for their technical assistance in preparing this paper. Special thanks is due to Dr Gary Hoffman for his critical appraisal of our work.

NASIRUDDIN M. JAMAL, MD
Clinical Associate Professor of Medicine
SUNY Health Science Center
at Syracuse
766 Irving Ave
Syracuse, NY 13210

GEORGE M. GHATTAS, MD
Medical Resident
Wilson Memorial Hospital
33-57 Harrison St
Johnson City, NY 13790

1. Panidis IP, Kotler MN, Mintz GS, et al: Clinical and echocardiographic features of right atrial masses. *Am Heart J* 1984; 107:745-758.
2. Felner JM, Churchwell AL, Murphy DA: Right atrial thromboemboli: Clinical, echocardiographic and pathophysiologic manifestations. *J Am Coll Cardiol* 1984; 4:1041-1051.
3. Ouyang P, Camara EJ, Jain A, et al: Intracavitary thrombi in the right heart associated with multiple pulmonary emboli. Report of two patients. *Chest* 1983; 84:296-299.
4. Malloy PC, Gore JM, Rippe JM, et al: Transient right atrial thrombus resulting in pulmonary embolism detected by two-dimensional echocardiography. *Am Heart J* 1984; 108:1047-1049.
5. Spirito P, Bellotti P, Chiarella F, et al: Right atrial thrombus detected by two-dimensional echocardiography after acute pulmonary embolism. *Am J Cardiol* 1984; 54:467.
6. Bergman G, Monaghan M, Daly K, et al: Right atrial embolus. Echocardiographic features. *Br Heart J* 1985; 53:552-555.
7. Nestico PF, Panidis IP, Kotler MN, et al: Surgical removal of right atrial thromboembolus detected by two-dimensional echocardiography in pulmonary embolism. *Am Heart J* 1984; 107:1278-1280.
8. Armstrong WF, Feigenbaum H, Dillon JC:

Echocardiographic detection of right atrial thromboembolism. *Chest* 1985; 87:801-806.

9. Starkey IR, deBono DP: Echocardiographic identification of right-sided cardiac intracavitary thromboembolus in massive pulmonary embolism. *Circulation* 1982; 66:1322-1325.

10. Goldberg SM, Pizzarello RA, Goldman MA, et al: Echocardiographic diagnosis of right atrial thromboembolism resulting in massive pulmonary embolization. *Am Heart J* 1984; 108:1371-1372.

11. Van Kuyk M, Mols P, Englert M: Right atrial thrombus leading to pulmonary embolism. *Br Heart J* 1984; 51:462-464.

12. Lim SP, Hakim SZ, van der Bel-Kahn JM: Two-dimensional echocardiography for detection of primary right atrial thrombus in pulmonary embolism. *Am Heart J* 1984; 108:1546-1547.

13. Rosenzweig MS, Nanda NC: Two-dimensional echocardiographic detection of circulating right atrial thrombi. *Am Heart J* 1982; 103:435-436.

14. Manno BV, Panidis IP, Kotler MN, et al: Two-dimensional echocardiographic detection of right atrial thrombi. *Am J Cardiol* 1983; 51:615-616.

15. Come PC: Transient right atrial thrombus during acute myocardial infarction. Diagnosis by echocardiography. *Am J Cardiol* 1983; 51:1228-1229.

Obturator hernias

TO THE EDITOR: Obturator hernias pass through the obturator foramen below the superior ramus of the pubic bone. They account for less than 1% of all mechanical small bowel obstructions and are infrequently diagnosed prior to surgery. The following case is unusual in that the preoperative diagnosis of an obturator hernia was demonstrated with the aid of an upper gastrointestinal series.

Case Report. An 86-year-old woman with no history of abdominal surgery was admitted because of obstipation and mild nausea. She had had a long history of constipation and had lost 40 lb during the past three months. Her condition had worsened, with the development of abdominal distention and diffuse lower abdominal pains.

A plain film was consistent with a small bowel obstruction. A small bowel series was performed and showed a pattern consistent with an incarcerated obturator hernia (Fig 1). Operative findings included a left-sided obturator hernia with a 6-cm loop of strangulated distal ileum. The hernia was reduced, and a small bowel resection was performed. Closure of the hernia was then carried out by suturing pectineal muscle to the periosteum of the obturator canal, effectively obliterating the fascial defect. Subsequently, a superficial wound infection was successfully treated with local wound care, and the patient was discharged on the 12th day after surgery.

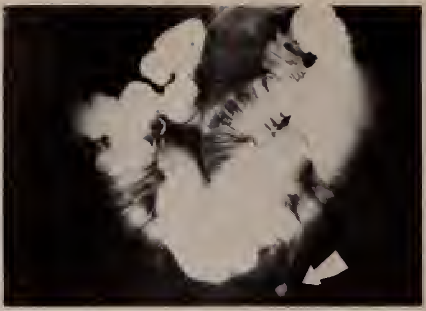


FIGURE 1. Radiographic film shows a pattern consistent with an incarcerated obturator hernia.

Discussion. The earliest known description of an obturator hernia was in 1722 by Roland Arnaude de Ronsil, and the first successful operation on an obturator hernia was performed by Henry Obre in 1851. By 1980, the number of reported cases in the English literature totalled 541.[1] Obturator hernias are correctly diagnosed prior to surgery in only 31% of cases and are associated with a mortality rate that ranges from 13% to 40%.[2] These hernias are responsible for about 0.4% of all mechanical small bowel obstructions.[3] A female predominance of 6:1 is thought to be due to the anatomic differences in the obturator canal in women; multiparity has also been incriminated. These hernias most commonly occur in the seventh and eighth decades of life and are associated with conditions of increased intra-abdominal pressure such as kyphoscoliosis, chronic respiratory disease, and chronic constipation.

The clinical presentation of an incarcerated obturator hernia is that of small bowel obstruction. In the early stages, however, the obstruction is incomplete, less dramatic, and therefore difficult to diagnose. The classic Howship-Romberg sign, or obturator neuralgia, consisting of pain in the anteromedial aspect of the thigh when the hip joint is extended, adducted, or inwardly rotated, is present in only 50% of cases.

A palpable mass in the thigh or on rectal or vaginal examination is rarely present. A correct preoperative diagnosis requires a high level of clinical suspicion, awareness of the Howship-Romberg sign, absence of an adductor reflex, and supportive radiographic evidence.[4] A retropubic or inguinal operative approach may be used, but the abdominal approach is recommended for four reasons: it establishes the diagnosis; exposure of the obturator ring is excellent; it is the method least dangerous to the obturator vessels; and it permits intestinal resection, which is required in 50% of cases.[5]

A variety of techniques have been described for closure of the hernia. They include the use of synthetic plugs such as tantalum gauze, Marlex mesh, and Teflon cloth, as well as the use of omental or costal cartilage plugs. Suturing the pectineal muscle to the periosteum of the obturator canal or using bladder wall or uterine fundus also repairs the defect.[6]

The operative mortality rate has been recorded as 10–50%. This high rate is due to delayed presentation, delayed operation, poor overall condition, advanced age of the patient, and rupture of gangrenous bowel at operation leading to septicemia. A correct preoperative diagnosis, whenever possible, decreases the likelihood of morbidity and mortality and allows for a better directed surgical approach.

DENNIS WADLER, MD
RICHARD H. ROSE, MD
Department of Surgery
Beth Israel Medical Center
10 Nathan Perlman Place
New York, NY 10003

1. Sinha SN, Decosta AE: Obturator hernia. *Aust NZ J Surg* 1983; 53:349-351.

2. Olivier J, Noyez D, KUL: Hernia obturatoris; Casuistiek en vergelijkende dissertatie. *Acta Chir Belg* 1973; 72:116-129.

3. Rogers FA: Strangulated obturator hernia. *Surgery* 1960; 48:394-403.

4. Hannington-Kiff JG: Absent thigh adductor reflex in obturator hernia. *Lancet* 1980; 1:180.

5. Gray SW, Skandalakis JE, Soria RE, et al: Strangulated obturator hernia. *Surgery* 1974; 75:20-27.

6. Gray SW, Skandalakis JE: Strangulated obturator hernia, in Nyhus LM, Condon RE (eds): *Hernia*. Philadelphia, Lippincott & Co, 1978, pp 427-440.

THE INTERNATIONAL TEXTBOOK OF CARDIOLOGY

Edited by Tsung O. Cheng, MD. 1297 pp, illustrated. Elmsford, NY, Pergamon Press Inc, 1986. $140.00 (hardcover)

Any cardiology text that has a picture of Paul Dudley White on its frontispiece and a foreword by his wife deserves respect and careful inspection. Dr Cheng worked with Dr White at Massachusetts General Hospital in 1957, during which time he had a son. He thought so highly of Dr White that he named his son Mark Dudley Cheng.

In the foreword, Mrs White reminds us of Dr White's philosophy of medicine as it pertained to bringing peoples of the world together. She says, "Physicians have 'no axe to grind,' their sole purpose being to work and study together to help solve the multiplicity of problems that face them for the betterment of all people."

Dr Cheng has literally brought together the works of distinguished cardiologists from all parts of the world in his *International Textbook of Cardiology*. Through his editing, this tremendous undertaking has been authored by 136 internationally known cardiologists. Their countries of origin stretch around the world. The very magnitude of the work reminds one of Dr White's expedition to record the electrocardiogram of the whale. To have amassed such a vast amount of material and yet have a timely publication represents a major endeavor.

In addition to the expected chapters on various types of heart disease, there are some unusual ones on such topics as geographic cardiology, heart diseases in the tropics and parasitic heart diseases, cardiovascular pharmacology, antibiotics in cardiology, geriatric cardiology, computers and cardiovascular practice, preventive cardiology, new cardiovascular drug development, and medical communications in cardiology. In the chapter on geographic cardiology, there is an interesting section on the changing incidence of heart disease in modern China. Among other risk factors, the levels of serum cholesterol and cigarette smoking have risen appreciably.

The text is clearly written and the print is easy to read, with double columns on each page. There is a topic outline at the beginning of each chapter. The illustrations, graphs, and tables are profuse and clearly identified. References are appropriately marked in the text and appear at the end of each chapter. References are dated as recently as 1986, and the information in the text gives every indication of being current.

The approach to some cardiac problems is somewhat different in this text, as compared to others. For instance, the chapter on cardiac pacing is short and to the point, leaving out extensive discussions of the types of pacemakers, their radiological appearance, and their technical abilities, although reference to such material is present. It seems evident that the increased number of topics has somewhat necessitated the abbreviation of the amount of material in each chapter. Such a process tends to indicate a more succinct discussion of the topics at hand.

The text provides a very comprehensive approach to cardiology for the student and the practicing family physician or general internist, and it serves as an excellent reference for the cardiologist.

JOHN N. EDSON, MD
The Long Island College Hospital
Brooklyn, NY 11201

CONSENT AND CONFIDENTIALITY IN THE HEALTH CARE OF CHILDREN AND ADOLESCENTS. A LEGAL GUIDE

By James M. Morrissey, Adele D. Hofmann, and Jeffrey C. Thrope. 273 pages. New York, The Free Press, 1986. $30.00 (hardcover)

Legal and ethical issues in the provision of medical care to children and adolescents are important concerns for physicians and other health care personnel. Can an adolescent be treated without parental consent? Can an adolescent be treated with parental consent, or must the adolescent agree to treatment as well? Are the issues legal ones or ethical ones? What constitutes the "law"? These types of questions are addressed with skill and expertise by the authors of this excellent, readable text.

The authors—Adele Hofmann, MD, formerly president of the Society for Adolescent Medicine, and lawyers James M. Morrissey and Jeffrey C. Thrope, who have practiced in the area of health law, rights of minors, and youth advocacy—are eminently qualified to collaborate on such an effort. The text is divided into six chapters and an extremely useful appendix which details for each state in capsule form such legal definitions as age of consent, mature minor statutes, emergency treatment rules, rules governing provision of contraceptive services, etc. I have had occasion to refer to this section several times in the short time that I have had the book available. The index easily allows the reader to more fully explore a specific item or situation. The legal and ethical issues are discussed according to patient status as well as patient need. Many special situations are also discussed, such as the use of minors as research subjects, parental refusal of recommended treatment, care of handicapped newborns, and parent/child conflict. The book uses appropriate case material to illustrate or clarify specific points in the law or ethical issues.

This book should be useful for health care practitioners and anyone interested in the complex issues which are discussed. The reader will develop an appreciation of the many factors involved in decision making in regard to consent and confidentiality issues. The book will be a valuable reference for every physician's library.

I. RONALD SHENKER, MD
Schneider Childrens Hospital
of the Long Island Jewish-
Hillside Medical Center
New Hyde Park, NY 11042

BLACK ADOLESCENT PREGNANCY. PREVENTION AND MANAGEMENT

Edited by Norma J. Goodwin, MD, 74 pp, illustrated. New York, Human Sciences Press, Inc, 1986. $9.95 (softcover)

The theme of this book is that unplanned adolescent pregnancy is a general societal problem. Its purpose is to describe how to prevent unplanned pregnancy from occurring, if possible, and to instruct physicians in the management of those pregnancies in which prevention has failed. The editor, Norma I. Goodwin, MD, has been intensely interested in this subject for many years and has assembled an impressive array of experts to present their findings. The book is a timely addition to the medical literature and assumes even greater significance given the American Medical Association's emphasis on the care of adolescents and the programs of the American College of Obstetricians and Gynecologists aimed at preventing teenage pregnancy.

The editorial by Dr Goodwin draws attention to concerns about high rates of premarital teen pregnancies, and notes that only 9% of teen fathers receive prior contraception advice. She stresses the need to give this problem a high priority to ensure a better world for all our adolescents. The book itself consists of 12 articles, beginning with a discussion of prevention and management and ending with a discussion of family life education in the English-speaking Caribbean.

Randolph and Gesche note that each day 180 teenagers become pregnant in New York State, and cite the immediate and long range consequences of these pregnancies. They discuss New York State's Teen Pregnancy Initiative which envisions the availability and promotion of alternatives to child bearing. Moran points out that the basis of contraceptive or abortion services for adolescents was the professional judgment of the physician as to the maturity of the person seeking care. He emphasizes the importance of the leadership role of black physicians in informing their communities about the risks and complications of teen pregnancies and parenting. Clarek compares the rate of teen pregnancy in Japan, 5/1,000, to that of the United States, and stresses the importance of prevention and early prenatal care, citing increased mortality and morbidity in very young teen pregnant patients. Harris reviews the experience of the Bronx Committee for the Community Health and describes their curriculum for 10-13-year-olds. Johnson focuses on meeting the total needs of adolescents of both sexes. Height notes the large numbers of black children born to single teenage mothers. She makes the point that few adolescent mothers turn to the fathers of their children for help. (This was graphically illustrated by Bill Moyers' recent award winning television program devoted to teenage mothers in Newark, New Jersey.) Pitt describes the adolescent male and the National Urban League initiative in developing a media campaign to create in young black males an awareness of their responsibilities in sexual relations and parenting. Matin describes the experience of the Sisterhood of Black Single Mothers in helping adolescent mothers through a series of projects. Shecter reviews the New York City School System's Family Life Education program which is designed to help youngsters reduce their anxieties and develop positive attitudes towards their own bodies. Canada comments on the Brooklyn Teen Pregnancy Network which serves as an information and resource sharing agency devoted to the needs of pregnant teenagers. Edelman and Pittman point out that the United States leads nearly all other developed nations of the world in rates of teenage pregnancy, abortion, and childbearing, even though it has comparable rates of sexual activity. They declare that a shift in focus is required from preventing sexual activity to preventing pregnancy and that all our youth should be prepared for adulthood. Epps et al describe a series of family life education programs funded by the United Nations for Caribbean countries using specially trained indigenous personnel.

The book is a clear and reasoned exposition of a social dilemma. It is well organized, with print that is easy to read and graphs and tables that complement the written material. The book should reach a wide audience since it is reasonably priced and compares favorably with similar publications. Again and again the authors reiterate that this is not an ethnic problem, but one that confronts the entire adolescent population of our society.

RANDALL BLOOMFIELD, MD
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

1986 YEAR BOOK OF FAMILY PRACTICE

Edited by Robert E. Rakel, MD, 602 pp, illustrated. Chicago, Year Book Medical Publishers, 1986. $42.95 (hardcover)

Dr Robert E. Rakel, the editor of this volume, is well known to physicians in family practice and is highly qualified to edit this annual yearbook of family practice. The editorial goal of this work was "to select 400 articles that are of the greatest value to the practicing family physician" from the 20,000 that were reviewed. The selections were culled from 121 different periodicals.

In fact, the articles are presented as summaries that average about one page in length. These seem to have been written by the editors themselves or their assistants; they do not appear to be summaries or abstracts taken from the originals. Chapters are arranged according to medical specialties and bodily systems. Most have several sections that deal with specific topics. With the table of contents organized according to this format, particular areas of interest can be located quickly. The index lists reference for the past five years, as well as the contents of the current volume.

The major function of this yearbook is to provide a rapid review of the staggering volume of medical literature published each year. The summaries are slightly longer and more thorough than the typical summaries or abstracts found in the original articles. Some articles have additional comments by the editor responsible for that topic. These comments are useful in that they give the reader another perspective, in agreement or disagreement with the original citation.

The more thorough sections include those on neoplasms, the circulatory and respiratory sytstems, gastroenterology, and gynecology and obstetrics. The best section is that covering psychiatry, which includes frequent and lengthy comments by Dr Phillip G. Couchman. Most sections include material on the "cutting edge" of modern medical terminology and practice. For example, genetic mapping and engineering are reviewed. Their roles in the diagnosis and treatment of disease and use in the increased production of some drugs that previously were only available in small quantities at great cost are also discussed.

Although there are some articles on sports medicine, this topic is generally not well represented. Family problems seemed "tacked on" at the end of the book because it is supposed to be about *family* practice. Discussion of the role of religious beliefs is entirely absent, as though it is never a meaningful factor in either the medical or psychological problems of families. Similarly, medical ethics is not addressed, although the family physician is deeply involved in issues of abortion, tube feeding, respirator control, medical economics, etc.

Any collated volume will be highly selective and reflect the editors' biases, yet this book seems to maintain a reasonable balance in the quality and breadth of its articles. It is not an exhaustive review of a particular subject. However, it is a means by which a vast literature can be reviewed in a brief manner. As such, and in spite of its several deficiencies, it is a valuable addition to the family physician's library and to the medical literature. This reviewer learned much from it.

FRANKLIN E. PAYNE, MD
Associate Professor
Medical College of Georgia
Augusta, GA 30912-5470

RABIES, THE FACTS

By Colin Kaplan, G. S. Turner, and D. A. Warrell. 126 pp, illustrated. New York, Oxford University Press, 1986. $13.95 (hardcover)

This volume is an excellent, well-referenced monograph dealing with many of the important aspects of the rabies virus, including basic science, clinical veterinary, epidemiologic, preventive medicine, and historical aspects. The authors, Colin Kaplan, profesor of microbiology at the University of Reading; G. S. Turner, former virologist at the Lister Institute and Blood Products Laboratory; and D. A. Warrell, director of the Oxford Tropical Medicine Programme, at the Wellcome Mahidol University, Bangkok, relate their extensive personal experience with the many aspects of the rabies virus in a concise, easy-to-read, and quite informative format. These authors capture the reader's interest with graphic case descriptions of the spectrum of clinical infection due to the rabies virus in man and animals. They point out issues not widely appreciated, such as how certain zoonotic viruses serologically distinct from the rabies virus (Mokola and Duvenhage viruses) occasionally may cause human infection which is clinically indistinguishable from rabies encephalitis, but apparently is not preventable by current rabies virus vaccines.

Rabies virus vaccine development is described in an interesting manner and provides a good understanding of the relative advantages and disadvantages of each vaccine within a framework of a fascinating historical perspective dating back to Pasteur's development of the first animal-passaged vaccine in 1885. Chapters dealing with the epidemiology of rabies, the ecology of the rabies virus, and the control of rabies in domesticated and nondomesticated animals round out the background material presented in this monograph.

The authors' comments are consistent with those of most other authorities. One significant difference of opinion should be noted, however. The authors recommend the current World Health Organization (WHO) program for post-exposure prophylaxis which consists of the administration of human rabies immune globulin (HRIG) in conjunction with six intramuscular doses of human diploid cell vaccine (HDCV). The Advisory Committee on Immunization Practices (ACIP) in the United States currently recommends HRIG followed by HDCV on days 0, 3, 7, 14, and 28 (a five-dose regimen excluding the sixth, 90-day, dose of the WHO regimen). No cases of human rabies have been reported when the ACIP recommendations have been properly followed (*MMWR* 1984; 33:393).

This monograph is accurate and well organized, and is appealing to readers of varied backgrounds. The authors attempt to define certain technical terms for the benefit of those with a more limited scientific background. These efforts will at times distract some readers, while at other times fall short of clarifying some technical terminology for the lay reader. Despite these minor criticisms, this excellent, concise monograph is a welcome addition to the libraries of health care providers and public health workers alike and is highly recommended as a convenient resource.

GERALD GORDON, MD
THOMAS BALZ, MD
Geisinger Medical Center
Danville, PA 17822

THE ANTIMICROBIAL DRUGS

By William B. Pratt, MD, and Robert Fekety, MD. 501 pp, illustrated. New York, Oxford University Press, 1986. $27.95 (hardcover)

This book, written by two well-known authorities from the University of Michigan, is a clinical expansion of Pratt's earlier books on antimicrobial agents. The authors have set about to integrate the field in a comprehensive but easily usable form. They succeed in a way that makes this moderately priced volume useful to both the generalist and subspecialist as well as to both the house officer and student.

As with most textbooks on antimicrobial chemotherapy, the major portion of the book deals with medications used in the management of bacterial infections. These chapters are grouped according to the mechanism of action of the agents. There are also well written and extensively referenced chapters on the treatment of mycobacterial infections, antifungal agents, and viral chemotherapy, and chapters on the treatment of malaria, protozoa, and helminths. These chapters are all authoritative, covering mechanisms of action, pharmacology, metabolism, toxicity, and the clinical utility of each agent discussed. The authors have included three chapters for clinicians in which they outline general principles regarding the choice of antibiotics in office practice and for treating severe bacterial infections in hospitalized patients.

The text is relatively up to date in its discussion of chemotherapy. However, as in any rapidly expanding field, a number of recently released agents are not included. Among these are a second generation cephalosporin, cefotetan, a third generation cephalosporin, ceftazidime, and the first released carbapenem, imipenem. In addition, the newest antiviral agent, ribavirin, used as an aerosol for respiratory syncytial virus, is not discussed. It would have been helpful if the authors had included some discussion of these antimicrobials as well as of agents such as azthreonam (the first monobactam) and the quinolones, norfloxacin and ciprofloxacin, which will be released shortly.

The early clinical chapters will appeal to practitioners. They contain a great deal of concentrated information. Some of the tables, however, are either too detailed or overly simplified.

This book will be an appropriate and welcome addition to any medical library. It contains a wealth of information in concise form which makes it extremely useful for practicing physicians.

LARRY I. LUTWICK, MD
Maimonides Medical Center
Brooklyn, NY 11219

THE PERSISTING OSLER. SELECTED TRANSACTIONS OF THE FIRST TEN YEARS OF THE AMERICAN OSLER SOCIETY

Edited by Jeremiah A. Barondess, MD, John P. McGovern, MD, and Charles G. Roland, MD. 318 pp, illustrated. Baltimore, Md, University Park Press, 1985. $45.00 (hardcover)

It is fashionable today to characterize some physicians as incompetent, poor communicators, and devoid of humanism. As a result, medical schools are searching for superior role models to teach students. The subject of this book, Sir William Osler, in his day was the best.

The editors and other contributors, all of whom are outstanding physicians, expertly employ a select series of essays to weave for the reader an intimate view of the personal and professional life of this remarkable man. Sir William loved people and books. He was an enthusiastic teacher, dedicated to the care of the sick and loyal to the institutions he served. He was kind and considerate. He spoke and wrote very well and was correctly considered the father of bedside teaching.

A teacher can be judged by the accomplishments of his students and using that as a yardstick, Osler was a "superstar." Amazingly, he even made an excellent choice in picking a mate. He was a devoted husband and loving father who never recovered from the pain he felt when a horrible war prematurely ended his only son's life.

There is no greater tribute one can pay to a man than recording the accolades written by his colleagues and students who refuse to allow him to die. McGill, the University of Pennsylvania, Johns Hopkins, and Oxford were fortunate to have had Osler and we thank the American Osler Society for affording us the opportunity to share selections from their transactions.

Students, educators, and medical history buffs will enjoy reading this marvelous book.

JULIUS E. STOLFI, MD
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

COMMON SPORTS INJURIES IN YOUNGSTERS

By Richard B. Birrer, MD, and David B. Brecher, MD. 148 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $19.95 (softcover)

Written by two family practitioners, this book provides a description of the injuries sustained as a result of sports participation. In addition to the six chapters organized around specific anatomic regions, there are sections dealing with the preseason examination, onfield management of injuries, the handicapped athlete, nutritional considerations, and rehabilitation techniques. There is a table with an excellent list of the supplies and equipment that should be available for athletic event coverage.

The strength of the book lies in its description of a large number of sports-related injuries. The weakness, a significant one, is that, based on the information provided, virtually none of the injuries can be adequately treated. The reliance on RICE (Rest, Ice, Compression, and Elevation) as "treatment" for nearly every injury ignores the almost universal need for specific rehabilitation programs when dealing with sports injuries.

Although the book bears a 1987 copyright, current knowledge regarding specific injuries is all but ignored. Examples of these omissions include the emphasis on hyperflexion as the major cause of (catastrophic) neck injuries and reliance on the anterior drawer test to "confirm" the diagnosis of a torn anterior cruciate ligament. In these and other instances, the information is not merely incomplete, but wrong.

For the practitioner seeking a brief description of the many injuries associated with athletic participation, this book would be of value. For those wanting to diagnose and treat sports injuries, the American Academy of Orthopaedic Surgeons' book, *Athletic Training and Sports Medicine*, would be much more appropriate.

JAMES G. GARRICK, MD
Director, Center for Sports Medicine
Saint Francis Memorial Hospital
San Francisco, CA 94109

Readership Survey

The *Journal* undertook a readership survey of a selected sample of 1,550 readers in June. The sample was proportionally and randomly chosen from among the five broad specialty categories of physicians who receive the *Journal*. The survey, which was conducted for the *Journal* by Health Industries Research of Stamford, Connecticut, elicited opinions about content and format and invited suggestions for future improvements. The results of the survey will be published in a future issue of the *Journal*. The last readership survey was conducted in 1978. Comments and suggestions are always welcome, and readers are cordially invited to send them to the attention of the editor.

New glue could do job of stitches

Researchers at the University of Pennsylvania Hospital in Philadelphia have developed a unique type of glue that may replace stitches in delicate operations, such as corneal transplants and surgery of the inner ear. The glue is composed of thrombin, a component of blood that is often used during surgery to promote clotting, and fibrinogen, another blood-based clotting agent. What differentiates this new adhesive from a similar product developed in Europe is that the components are derived from a patient's own blood. This eliminates the risk of transmitting disease through contaminated blood products.

Dr Robert Weisman, who developed the glue, is testing it on patients with hearing loss due to inner ear damage. The glue, which is injected through a thin needle, could prove useful for delicate procedures in which sutures are not appropriate.

Laser device is first approved for treatment of vascular disorder

The Food and Drug Administration (FDA) granted approval of a laser device to treat obstructed arteries in the leg. Current treatments for blocked arteries include surgery and the less invasive and less costly procedure, balloon angioplasty. In the latter procedure, a small balloon attached to a fiberoptic probe is inserted into an artery and inflated to compress the fatty material obstructing the artery. But balloon angioplasty is ineffective when the artery is completely blocked and when calcium deposits contribute to the blockage. In such cases, a laser can be used to create a hole large enough to allow passage of the balloon probe.

This new device (made by Trimedyne, Inc, Santa Ana, Calif) contains an argon laser encased in a metal-tipped fiberoptic probe. The probe is introduced into the artery to the site of the blockage. Activation of the laser heats the metal tip to about 400°C, which then vaporizes the fatty deposits. Because the laser beam is used as a heat source and does not come in direct contact with the tissue, there is little risk of accidentally burning the artery itself.

The laser probe has been shown to be effective in early studies on 207 patients. Indications for FDA-approved use of the device are limited to treating obstructed arteries in the leg that cannot be cleared using balloon angioplasty alone.

Vancomycin resistance documented

Antibiotic resistance among pathogenic strains of bacteria is increasing, and the implications of this phenomenon are frightening. Vancomycin is the antibiotic of choice for staphylococcal infections that are resistant to penicillin. As such, vancomycin has become a sort of "fail safe" antibiotic. Its use to treat infections caused by gram-positive bacteria had not led to the development of vancomycin-resistant organisms. However, in the April 9, 1987, issue of the *New England Journal of Medicine* (316:927–931), Schwalbe et al describe a case in which increasing vancomycin resistance is evident in successive staphylococcal isolates.

The patient was a 37-year-old man with diabetes, who was on continuous ambulatory peritoneal dialysis for end-stage renal disease. He sought treatment for nausea, vomiting, and abdominal pain. The peritoneal fluid was cloudy and contained coagulase-negative staphylococci. These organisms have been implicated in several types of infection, including nosocomial bacteremia and peritonitis; the latter being responsible for the symptoms experienced by this patient.

The patient was taking cefazolin for streptococcal peritonitis at the time of presentation, and he had previously completed at least three courses of vancomycin for staphylococcal infections. When treatment with intraperitoneal cefazolin and tobramycin failed, and methicillin-resistant coagulase-negative staphylococci—identified as *Staphylococcus haemolyticus*—were isolated from two specimens, intravenous vancomycin therapy, 1 g/week, was instituted. Subsequent isolation of coagulase-negative staphylococci led to a four-week course of treatment with an increased dose of vancomycin. When coagulase-negative staphylococci were again recovered, additional therapy with rifampin and tobramycin was needed to control the infection.

The authors report that the coagulase-negative staphylococcal isolates showed a stepwise increase in vancomycin resistance from a minimal inhibitory concentration of 2 μg/mL to 8 μg/mL during the 88 days of vancomycin therapy. "Our investigations strongly suggest that coagulase-negative staphylococci have the ability to acquire vancomycin resistance," they conclude.

Although this resistant strain of staphylococcus is probably rare, it is possible that other strains, such as *S aureus*, which is highly virulent, could also develop resistance. As Schwalbe et al note, "the current therapeutic alternatives to vancomycin are limited. For this reason, it would seem prudent to identify alternative antibiotics for treating these infections [and] . . . to use vancomycin sparingly as a prophylactic agent."

Protecting healthy cells from chemotherapeutic drugs

Radioprotectors are compounds that shield healthy cells from the toxic effects of chemotherapeutic agents. In the process of destroying cancerous cells, these agents can produce mutations in the genetic material of healthy cells, causing them to become cancerous five to 20 years later. Radioprotectors, now being studied at the Department of Energy's Argonne National Laboratory (Argonne, Ill), make normal cells as much as seven times more resistant to genetic alterations caused by certain chemotherapeutic drugs (eg, *cis*-platinum).

Radioprotectors are believed to exert this protective effect through two mechanisms: by lengthening the cell cycle and by inhibiting free radicals. A longer time period between cell divisions gives the cells a greater opportunity to repair DNA that may have been damaged during chemotherapy, thus lessening the chance that genetic muta-

---

NEWS BRIEFS are compiled and written by Vicki Glaser.

tions will be passed on to future cells. Free radicals, or atoms that have an extra electron, are chemical byproducts of cell metabolism that have been implicated in the initiation of cancer. Beneficial effects are achieved as healthy cells, which receive a better blood supply, are afforded more protection than cancerous cells, according to a spokesperson at Argonne National Laboratory.

Radioprotectors were shown to be most effective when given before exposure to chemotherapeutic drugs, although, in some cases, a protective effect was apparent when radioprotectors were given up to three hours after exposure. Radioprotectors are still in the early stages of experimentation, and they will not be available for clinical use in cancer therapy in the near future.

The importance of mandatory mumps vaccination

In the fall of 1983, a 40% increase in mumps cases was reported in a single county in New Jersey, as compared to the total number of cases in 1982 among schoolchildren statewide. Chaiken et al (*JAMA* 1987; 257:2455–2458) investigated the outbreak in the hopes of determining its cause and controlling the spread of the disease.

The mumps vaccine received approval for licensure in 1967. Reported cases of mumps declined by 98% between 1967 and 1983. A combined vaccine for measles, mumps, and rubella is routinely administered to children at 15 months of age. Mumps vaccination was first required for school entry in New Jersey in 1978. Therefore, by the fall of 1983, children in kindergarten through grade 5 should all have received mumps vaccine.

Seventy-six percent (48) of cases occurred in one of the six affected schools in New Jersey. Of those 48 cases, 25 (52%) occurred among students in the sixth grade, who were not covered by the vaccination law. These students were almost seven times more likely to be susceptible to the mumps than students in kindergarten through grade 5. In general, "grade-specific susceptibility rates paralleled grade-specific attack rates."

The authors reviewed the immunization records of all children in the school district, recording evidence of mumps vaccination or mumps disease. Of the 13 students in the fifth grade (of the one school mentioned previously) who got the mumps, 12 had been vaccinated. This finding prompted the authors to undertake a vaccine efficacy study, using this one school's sixth graders as the test population. The vaccine efficacy was estimated at 91% for sixth graders, with an attack rate of 4% among vaccinated students. The reason for the low vaccine efficacy rate among the fifth graders was not determined.

Chaiken et al determined the financial toll of this mumps outbreak—which affected 46 households—for a total cost of $4,687, or a mean cost of $102/household. These figures include the cost of workdays lost by parents, day-care costs, physician visits, emergency room visits, and medication.

The mumps vaccine is safe, effective, and cost effective. In the study population described here, there was a 95% rate of compliance with mandatory school vaccination laws. The majority of cases occurred in students not covered by the New Jersey law. Currently, 30 states require proof of mumps immunity for school entry. The authors conclude that "considerable medical and economic savings can be realized by including mumps immunization as part of state compulsory school immunization."

News in brief

- The congressional Office of Technology Assessment is predicting a five-fold increase in the number of victims of Alzheimer disease by the year 2040, assuming that no preventive or curative treatment is discovered before that time. The main cause of this anticipated increase—with 7.4 million Americans expected to have the disease by 2040—is the advancing age of the "baby boom" segment of the population. Although federal funding of research on all forms of dementia rose from $4 million in 1976 to $67 million in 1987, much of the financial toll of caring for a patient with Alzheimer disease rests on the families. As the number of victims climbs, one out of three families in the US will soon be affected.
- The American Medical Association (AMA), in association with the CBS and ABC television networks, has produced public service announcements that confront the issue of "safe sex." Aimed at adolescent viewers, the broadcasts feature teen idol actor Tony Danza urging teens to postpone having sex or to use condoms. This effort by the AMA is aimed at preventing the spread of AIDS among sexually active adolescents.
- Defects in the enamel of baby teeth may correlate with hearing loss, according to researchers at Case Western Reserve University (Cleveland, Ohio). The results of a pilot study (*Dentistry Today* 1987; 6(3):1, 38) suggest that enamel defects which manifest as a line or arc across the teeth may be a sign of hearing loss as well as of other neurologic disorders.

The two abnormalities—hearing loss and the enamel defect—are both thought to occur due to oxygen deprivation sometime during the pregnancy. The ears and the teeth develop at about the same time during gestation. Furthermore, the location of the enamel defect on the tooth can be used to determine how early in the pregnancy oxygen deprivation occurred and how extensive the hearing loss might be.

It is important to note that enamel abnormalities can occur in the absence of any other disorders. But the high correlation noted in this study suggests an easily identifiable biological marker for hearing loss.

- Results of a multicenter study coordinated by the Centers for Disease Control in Atlanta confirm a link between Reye syndrome—a rare form of encephalopathy in children, which can be fatal—and the use of aspirin to treat children with chicken pox, respiratory infections, or gastrointestinal illnesses. The investigators reported that more than 90% of the test group with Reye syndrome had recently received aspirin for a variety of illnesses. The data compiled for this controlled study—published in the *Journal of the American Medical Association* (1987; 14:1941)—indicate that the use of aspirin among children is declining.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Louis Brockman, MD, New York. Died January 16, 1987; age 82. Dr Brockman was a 1932 graduate of State University of New York Health Science Center at Brooklyn. He was a Fellow of the American Academy of Family Practice and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

George A. Bullwinkel, MD, Rye. Died April 17, 1987; age 85. He was a 1928 graduate of Cornell University Medical College, New York. Dr Bullwinkel was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

F. Vincent Burke, MD, Brooklyn. Died December 2, 1986; age 78. He was a 1935 graduate of the University of Vermont College of Medicine, Burlington. Dr Burke was a Fellow of the American College of Chest Physicians and the American College of Angiology. He was a member of the New York Cardiological Society, the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Donald Jack Casper, MD, New Hyde Park. Died April 8, 1987; age 55. He was a 1956 graduate of New York University College of Medicine, New York. Dr Casper was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Morris Harris Cohan, MD, Carmichael, California. Died February 28, 1987; age 84. Dr Cohan was a 1928 graduate of State University of New York Health Science Center at Brooklyn. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Robert Joseph Collins, MD, Syracuse. Died April 10, 1987; age 72. He was a 1942 graduate of State University of New York Health Science Center at Syracuse. Dr Collins was a member of the Academy of Medicine, the American Geriatrics Society, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Albert Cornell, MD, New York. Died April 11, 1987; age 77. He was a 1934 graduate of New York University College of Medicine, New York. Dr Cornell was a Fellow of the American College of Gastroenterology and a Diplomate of the American Board of Internal Medicine. His memberships included the New York Gastroenterological Society, the American Gastro-Enterological Association, the New York County Medical Society, and the Medical Society of the State of New York.

Frank Joseph DeLuca, MD, Lakehurst, New Jersey. Died January 21, 1987; age 77. Dr DeLuca was a 1934 graduate of Facolta di Medicina e Chirurgia dell'Universita di Bologna. He was a Fellow of the American Academy of Family Practice and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

John Eder, MD, Manhasset. Died April 6, 1987; age 77. He was a 1936 graduate of Akademia Medyczna, Krakow. Dr Eder was a member of the American Academy of Family Practice, the Nassau County Medical Society, and the Medical Society of the State of New York.

Fred Feldman, MD, Oneida. Died March 11, 1987; age 83. Dr Feldman was a 1930 graduate of Charles Universita Fakolta of Ped-Gen Medicine, Praha. He was a Fellow of the American Academy of Family Practice. His memberships included the Academy of Medicine, the American Society of Clinical Hypnosis, the Medical Society of the County of Madison, and the Medical Society of the State of New York.

Gerald Feuerstein, MD, Smithtown. Died March 28, 1987; age 61. Dr Feuerstein was a 1958 graduate of Faculteit der Geneeskunde Universiteit van Amsterdam. He was a member of the American Geriatrics Society, the Academy of Psychosomatic Medicine, the Suffolk County Medical Society, and the Medical Society of the State of New York.

E. L. Fronczak-Bukowska, MD, Buffalo. Died April 15, 1987; age 84. Dr Fronczak-Bukowska was a 1928 graduate of State University of New York at Buffalo School of Medicine, Buffalo. Dr Fronczak-Bukowska was a member of the American Academy of Family Practice, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Leopold Goldfarb, MD, Amityville. Died March 3, 1987; age 88. He was a 1925 graduate of Universitaet Wien Medizinische Fakultaet, Wien. Dr Goldfarb was a member of the American Psychiatric Association, the American Geriatrics Society, the Nassau County Medical Society, and the Medical Society of the State of New York.

Stefan Herz, MD, Brooklyn. Died January 29, 1987; age 86. Dr Herz was a 1927 graduate of Akademia Medyczna, Krakow. He was a member of the American Academy of Family Practice, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

George J. Kantor, MD, Ronkonkoma. Died April 18, 1987; age 80. He was a 1930 graduate of State University of New York Health Science Center at Brooklyn. Dr Kantor was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Milton Kissin, MD, New York. Died April 17, 1987; age 83. Dr Kissin was a 1928 graduate of Cornell University Medical College, New York. He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the Academy of Medicine, the Society for Experimental Biology and Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Hilmi I. Kodaman, MD, Brooklyn. Died January 30, 1987; age 62. Dr Kodaman was a 1951 graduate of Bursa Tip Fakultesi Istanbul Universitesi, Bursa. Dr Kodaman was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Danica N. Kovachev, MD, New York. Died April 20, 1987; age 68. Dr Kovachev was a 1947 graduate of Medicinski Fakultet Univerziteta u Beogradu, Beograd. Dr Kovachev was a Diplomate of the American Board of Plastic Surgery and a member of the American Association of Plastic Surgeons, the New York County Medical Society, and the Medical Society of the State of New York.

Dolor John Lauer, MD, Larchmont. Died April 2, 1987; age 75. He was a 1938 graduate of University of Minnesota Medical School, Minneapolis. Dr Lauer was a Fellow of the American College of Preventive Medicine and a Diplomate of the American Board of Preventive Medicine, Inc. His memberships included the Academy of Medicine, the American Academy of Occupational Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Garra Levant Lester, MD, Chautauqua. Died April 7, 1987; age 81. He was a 1929 graduate of State University of New York at Buffalo School of Medicine. Dr Lester was a Fellow of the American Academy of Family Practice and a member of the Academy of Medicine, the American Geriatrics Society, the Medical Society of the County of Chautauqua, and the Medical Society of the State of New York.

Donald Rich Lyon, MD, Middleburg. Died March 21, 1987; age 84. Dr Lyon was a 1938 graduate of Albany Medical College of Union University, Albany. He was a member of the Medical Society of the County of Schoharie and the Medical Society of the State of New York.

Frederick M. Marek, MD, New York. Died April 10, 1987; age 77. He was a 1934 graduate of Universitaet Wien Medizinische Fakultaet, Wien. Dr Marek was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Orthopedic Surgery. His memberships included the Academy of Medicine, the American Academy of Orthopaedic Surgeons, the American Academy of Compensation Medicine, Inc, the American Trauma Society, the New York County Medical Society, and the Medical Society of the State of New York.

David Mezz, MD, Hollywood, Florida. Died December 28, 1986; age 87. Dr Mezz was a 1928 graduate of New York University College of Medicine, New York. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Paul Colhoun Morton, MD, Washington Depot, Connecticut. Died March 8, 1987; age 90. He was a 1922 graduate of Harvard Medical School, Boston. Dr Morton was a Fellow of the American College of Surgeons, a Diplomate of the American Board of Surgery, and a member of the Academy of Medicine, the New York State Surgical Society, the New York County Medical Society, and the Medical Society of the State of New York.

Savas T. Nittis, MD, Lodi, Wisconsin. Died March 28, 1987; age 92. Dr Nittis was a 1922 graduate of Faculty of Medicine National University of Athens. He was a member of the Academy of Medicine, the International Society of Hematology, the New York County Medical Society, and the Medical Society of the State of New York.

Milton Alwood Palmer, MD, Lancaster. Died March 30, 1987; age 85. He was a 1927 graduate of State University of New York at Buffalo School of Medicine. Dr Palmer was a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Ophthalmology. His memberships included the New York Ophthalmology Society, the American Academy of Ophthalmology and Otolaryngology, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

George James Paul, MD, Rochester. Died March 28, 1987; age 49. Dr Paul was a 1964 graduate of State University of New York Health Science Center at Syracuse. He was a Diplomate of the American Board of Radiology and a member of the Academy of Medicine, the Radiological Society of North America, Inc, the American Roentgen Ray Society, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Howard G. Rapaport, MD, Scarsdale. Died March 30, 1987; age 79. Dr Rapaport was a 1934 graduate of Medizinische Fakultaet Der Universitaet Bern. He was a Fellow of the American College of Allergists and a Diplomate of the American Board of Pediatrics and the American Board of Allergy and Immunology. Dr Rapaport was a member of the American Academy of Pediatrics, the American Academy of Allergy, the New York County Medical Society, and the Medical Society of the State of New York.

Hymen Rogachefsky, MD, Rochester. Died March 15, 1987; age 67. Dr Rogachefsky was a 1951 graduate of State University of New York Health Science Center at Syracuse. He was a member of the Academy of Medicine, the American Academy of Dermatology, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Edwin Philip Russell, MD, Rome. Died April 10, 1987; age 90. He was a 1920 graduate of Cornell University Medical College, New York. Dr Russell was a Fellow of the American College of Physicians and a member of the Academy of Medicine, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Edward Aaron Saltzman, MD, Miami Beach, Florida. Died January 11, 1987; age 97. Dr Saltzman was a 1914 graduate of New York Medical College, New York. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Helga Amalie Schwarz, MD, Jamaica. Died April 5, 1987; age 64. Dr Schwarz was a 1949 graduate of Medizinische Fakultaet der Ludwig Maximilians Universitaet, Muenchen, Bayern. Dr Schwarz was a Diplomate of the American Board of Internal Medicine and a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Jane Henny Sichell, MD, New York. Died July 2, 1986; age 90. Dr Sichell was a 1927 graduate of Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Berlin. She was a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Herman Bernard Snow, MD, Hyde Park. Died April 12, 1987; age 78. He was a 1933 graduate of State University of New York Health Science Center at Syracuse. Dr Snow was a Fellow of the American Psychiatric Association and a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the Dutchess County Medical Society and the Medical Society of the State of New York.

Anthony J. Stalkus, MD, Forest Hills. Died April 20, 1987; age 77. Dr Stalkus was a 1935 graduate of Georgetown University School of Medicine, Washington, DC. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Frederick Engels Stone, MD, New York. Died August 1, 1986; age 89. Dr Stone was a 1921 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Otolaryngology and a member of the New York County Medical Society and the Medical Society of the State of New York.

Gaetano Thomas A. Viti, MD, Pelham Manor. Died April 20, 1987; age 52. Dr Viti was a 1963 graduate of Facolta di Medicina e Chirurgia dell'Universita di Padova. He was a Qualified Fellow of the International College of Surgeons. His memberships included the New York State Surgical Society, the American Society of Abdominal Surgeons, the Bronx County Medical Society, and the Medical Society of the State of New York.

Frederick Weintraub, MD, Brooklyn. Died January 21, 1987; age 92. He was a 1917 graduate of New York University College of Medicine, New York. Dr Weintraub was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, a Qualified Fellow of the International College of Surgeons, and a Diplomate of the American Board of Gynecology. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1983; 83:1089–1094). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith[2] reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

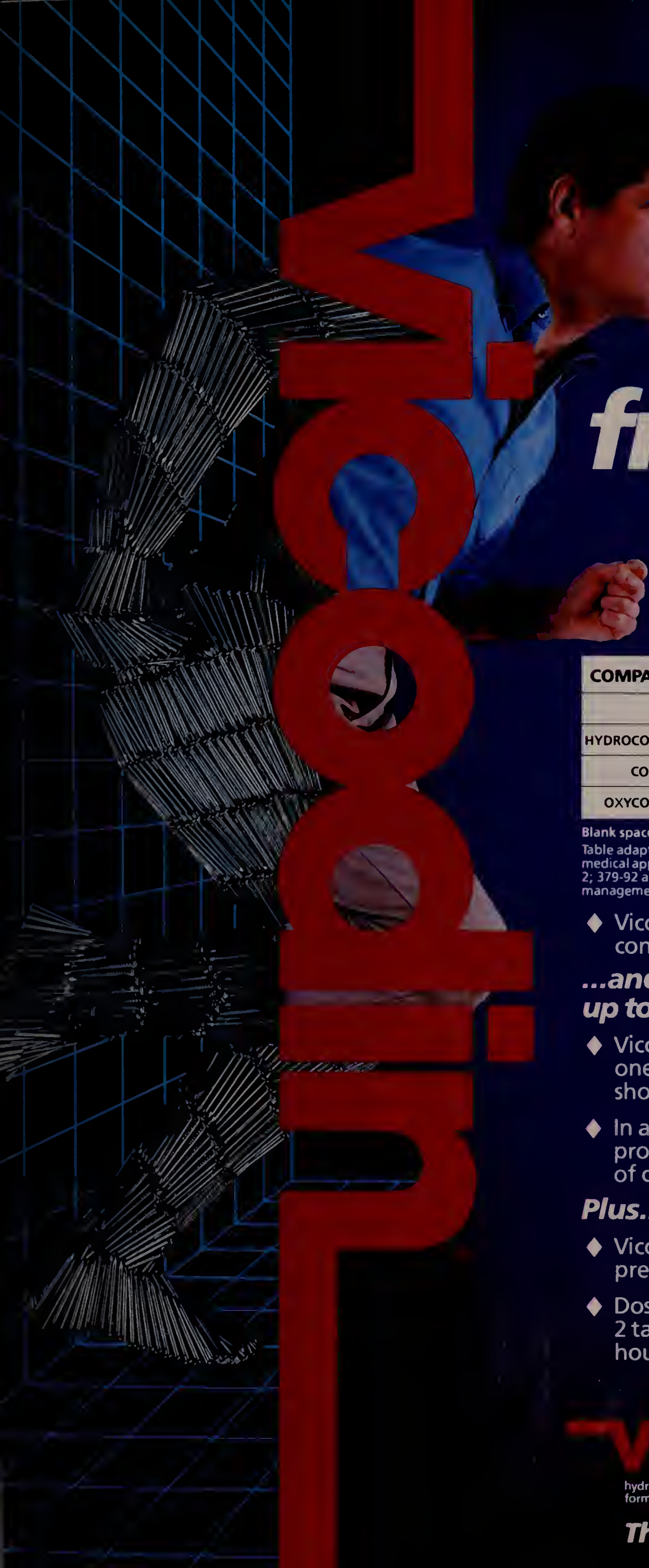
Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83: 925–927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337–338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, P.O. Box 5404, 420 Lakeville Road, Lake Success, NY 11042.



Freedom from pain

Just one part of pain relief therapy.

Vicodin® provides greater patient acceptance

COMPARATIVE PHARMACOLOGY OF THREE ANALGESICS

	CONSTIPATION	RESPIRATORY DEPRESSION	SEDATION	EMESIS	PHYSICAL DEPENDENCE
HYDROCODONE		X			X
CODEINE	X	X	X	X	X
OXYCODONE	XX	XX	XX	XX	XX

Blank space indicates that no such activity has been reported.

Table adapted from Facts and Comparisons (Nov.) 1984 and Catalano RB. The medical approach to management of pain caused by cancer. "Semin Oncol" 1975; 2; 379-92 and Reuler JB, et. al. The chronic pain syndrome: misconceptions and management. "Ann Intern Med" 1980; 93; 588-96.

- Vicodin offers: less nausea, less sedation, less constipation.

...and longer lasting pain relief— up to 6 hours.

- Vicodin contains hydrocodone not codeine. In one study, 10 mg. of hydrocodone alone was shown to be as effective as 60 mg. of codeine.[1]
- In a double-blind study, Vicodin (2 tablets), provided longer lasting pain relief than 60 mg. of codeine.[2]

Plus...

- Vicodin offers the convenience of CIII prescribing.
- Dosage flexibility—1 tablet every 6 hours or 2 tablets every 6 hours (up to 8 tablets in 24 hours).

1. Hopkinson JH III: *Curr Ther Res* 24: 503-516, 1978
2. Beaver, WT *Arch Intern Med,* 141:293-300, 1981.

vicodin®

hydrocodone bitartrate 5 mg. (Warning: May be habit forming) with acetaminophen 500 mg.

The original hydrocodone analgesic

vicodin ©

hydrocodone bitartrate 5mg (Warning May be habit forming). and acetaminophen 500mg

INDICATIONS AND USAGE: For the relief of moderate to moderately severe pain.
CONTRAINDICATIONS: Hypersensitivity to acetaminophen or hydrocodone.
WARNINGS
Drug Abuse and Dependence: VICODIN is subject to the Federal Controlled Substances Act (Schedule III) Psychic dependence, physical dependence and tolerance may develop upon repeated administration of narcotics; therefore, VICODIN should be prescribed and administered with the same caution appropriate to the use of other oral-narcotic-containing medications.
Respiratory Depression: At high doses or in sensitive patients, hydrocodone may produce dose-related respiratory depression by acting directly on brain stem respiratory centers Hydrocodone also affects centers that control respiratory rhythm, and may produce irregular and periodic breathing.
Head Injury and Increased Intracranial Pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a preexisting increase in intracranial pressure Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.
Acute Abdominal Conditions: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions.
PRECAUTIONS
Special Risk Patients: VICODIN should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture.
Information for Patients: VICODIN, like all narcotics, may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery; patients should be cautioned accordingly
Cough Reflex: Hydrocodone suppresses the cough reflex; caution should be exercised when VICODIN is used postoperatively and in patients with pulmonary disease.
Drug Interactions: The CNS-depressant effects of VICODIN may be additive with that of other CNS depressants When combined therapy is contemplated, the dose of one or both agents should be reduced The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone. The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus.
Usage in Pregnancy: Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose There are no adequate and well-controlled studies in pregnant women. VICODIN should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.
Nonteratogenic Effects: Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent. The intensity of the syndrome does not always correlate with the duration of maternal opioid use or dose.
Labor and Delivery: Administration of VICODIN to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used
Nursing Mothers: It is not known whether this drug is excreted in human milk; therefore, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.
Pediatric Use: Safety and effectiveness in children have not been established
ADVERSE REACTIONS
Central Nervous System: Sedation, drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, dizziness, psychic dependence, mood changes.
Gastrointestinal System: Nausea and vomiting may occur, they are more frequent in ambulatory than in recumbent patients Prolonged administration of VICODIN may produce constipation.
Genitourinary System: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported
Respiratory Depression: (See WARNINGS.)
DOSAGE AND ADMINISTRATION: Dosage should be adjusted according to the severity of the pain and the response of the patient However, tolerance to hydrocodone can develop with continued use, and the incidence of untoward effects is dose related The usual dose is one tablet every six hours as needed for pain (If necessary, this dose may be repeated at four-hour intervals) In cases of more severe pain, two tablets every six hours (up to eight tablets in 24 hours) may be required

Revised January 1987 MR 1987 5786

Knoll Pharmaceuticals
A Unit of BASF K&F Corporation
Whippany New Jersey 07981

BASF Group

knoll®

COMMERCIAL CONDOMINIUM
consultant
Paul A. Gellert
155 West 68th St. • NY, NY 10023
(212) 724-7900

REAL ESTATE FOR SALE OR RENT—CONT'D

OFFICE SPACE AVAILABLE: Throgs Neck section of Bronx. Modern medical complex, 1500 square feet, part time/full time. Call (212) 409-9494.

PROFESSIONAL CO-OP APARTMENT FOR SALE. Prime location in the heart of Forest Hills, suitable for any medical specialty. No doctor in 562 apartment building, ground floor, central air conditioning, garage available. One waiting, three examination and one reception rooms. Inquire 70-25 Yellowstone Blvd., Apt. 1-Z, Forest Hills, NY, 1-6 P.M. weekdays.

REGO PARK medical suite for rent in new high-rise condo building, near Alexander's Department Store on Queens Blvd., by owner. Future purchase possible. Call (718) 275-2907.

MISCELLANEOUS

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta, GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuarial certification and employee statements of participation—partnership agreements and professionals incorporation—No insurance required—references upon request—Wachstock and Dienstag Attorneys at Law, 122 Cutter Mill Road, Great Neck, NY 11021 (516) 773-3322.

FIRM OF GERALD GOLDBERG, CPA-ATTORNEY, formerly with IRS, announces his availability to prepare taxes and plan under changing tax environment, accounting, tax appeals, legal representation or practice transfers. (212) 406-1156 and (516) 565-6260.

MISCELLANEOUS—CONT'D

HAVE YOU RECEIVED ANY COMMUNICATION FROM THE OFFICE OF PROFESSIONAL MEDICAL CONDUCT? If affirmative, contact Susan Kaplan, Attorney-at-Law, (212) 877-5998. Practice limited to the crucial legal problems directly affecting your license to practice medicine—with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professions), and as an Assistant District Attorney in Nassau County. Susan Kaplan, Esq., 165 West End Avenue, Suite 27P, New York, N.Y. 10023.

TUNA THERAPY SEMINARS: Does your brain need a rest, your body need exercise, are your lungs in need of fresh air? Giant tuna fishing from Montauk Point, New York, aboard Montauk's finest charter boat "Dawn". Charters available for 1 to 4 serious medical anglers. Capt. Greg Beacher, RD-2, Box 16, Benson Drive, Montauk, NY 11954, (516) 668-9056.

COMPUTERIZE YOUR BILLING—Remarkably easy-to-use software automatically prints bills, statements, insurance forms. Menu-driven. Reports ageing balances, referral sources, income by time period, procedure & more. Installs automatically. IBM and compatibles. Solo practice, only $399. Group practice, only $499. Free information. Demonstration disk with complete manual, $19 plus $3 shipping, credited to purchase. Call or write REM Systems, Inc., Dept. NY6, 281 Dug Hill Road, Brownsboro, AL 35741 (205) 534-3457. VISA, MC Accepted.

PROFESSIONAL MISCONDUCT ATTORNEYS. William L. Wood Jr., formerly Executive Director of the New York State Office of Professional Discipline and Anthony Z. Scher, formerly Director of Prosecutions, announce the opening of their law office, Wood & Scher. Our recent tenure as chief enforcement officers has given us experience which will allow us to represent physicians in professional misconduct proceedings, malpractice licensure, license restoration, controlled drug proceedings, reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practitioners. Wood & Scher, Attorneys at Law, One Chase Road, Scarsdale, New York 10583. Telephone 914-723-3500.

SIXTH ANNUAL ELLA T. GRASSO MEMORIAL CONFERENCE, November 18, 1987, The Park Plaza Hotel, New Haven, Conn. For registration and further information (after August 1, 1987): Yale University School of Medicine, Office of Graduate and Continuing Education, 333 Cedar Street, P.O. Box 3333, New Haven, CT 06510, (203) 785-4578.

ADVICE, COUNSEL & REPRESENTATION of physicians and other health care professionals in licensure and professional conduct matters, reimbursement issues and practice-related litigation. David E. Ruck, Esq., former Chief, Criminal Division, Office of the New York State Special Prosecutor for Medicaid Fraud Control and Alain M. Bourgeois, Esq., former Acting Justice, Supreme Court of the State of New York, announce the opening of their offices for the practice of law under the name of Bourgeois & Ruck, 101 Park Avenue, New York, New York 10178. Telephone 212-661-8070.

A Clinical Opportunity for Smoking Intervention

You can play a special role in reaching smokers. Encouraging parents not to smoke can improve the health of the entire family.

Take a few minutes to explain that children of parents who smoke are often more prone to respiratory infections. Tell them that young children of parents who smoke are more likely to become smokers themselves.

The minutes you spend can make a difference now, and in the years ahead.

For a free copy of *Clinical Opportunities for Smoking Intervention: A Guide for the Busy Physician*, complete the form below.

Mail to:
The National Heart, Lung,
and Blood Institute
Smoking Education Progra
National Institutes of Healt
Building 31, Room 4A 18
Department P-26
Bethesda, MD 20892

Name

Specialty

Address

City

Classified Advertising

New York State Journal of Medicine

GENERAL INFORMATION

All classified advertisements are payable in advance, except for government agencies and advertising agencies; a 15% commission to recognized advertising agencies.

Frequency: Monthly
Mailing date: 7th of the month
Closing date: 30 days before mailing date

Circulation: 27,000 (mainly physicians & medical libraries)

DISPLAY ADVERTISING

	1 time	*6 time*	*12 time*	*Width*		*Depth*
1/4-page	$540	$480	$420	3-1/2″	×	4-7/8″
1/6-page	252	228	204	2-1/8″	×	4-7/8″
1/8-page	228	204	180	3-1/2″	×	2-3/8″
1/16-page	114	102	90	3-1/2″	×	1-1/8″
1/18-page	84	72	60	2-1/8″	×	1-1/2″

PARAGRAPH ADVERTISING

The first several words are set in bold face caps.

Rates: $35.00 per insertion for 50 words or less; additional words are 40 cents each. On request, Dept. numbers will be assigned by the Journal for replies to be forwarded to the advertiser. Dept. numbers are 50 cents per insertion. There is no frequency discount for paragraph ads.

Counting Words: Two initials, each abbreviation, figures consisting of 5 numerals or less, are counted as separate words. For replies, your name and address, or telephone number should appear at the end of the ad and be counted in the total number of words.

For further information, please call the Advertising Department at 516-488-6100.

CLASSIFIED ADVERTISING ORDER FORM

New York State Journal of Medicine
Attn: Classified Advertising
420 Lakeville Rd., P.O. Box 5404
Lake Success, N.Y. 11042

Date____________________

Name____________________________________

Address__________________________________

City/State/Zip____________________________

Payment enclosed: $____________

Number of Insertions: __________

Assign Dept. Number: __________

Please make check payable to New York State Journal of Medicine.

Some resocialization facilities are a last resort.
Ours are a first step.

Patients with significant psycho-social deficits need more than a place to spend their time. They need care that combines skilled nursing, social and behavioral treatment. The kind of care New Medico can provide.

Our Resocialization Programs are designed for patients requiring behavior-oriented treatment, as well as skilled nursing care. As part of a community, patients learn to cope with the challenges of daily living. Adaptive behaviors are formed, while inappropriate behaviors are overcome. Together, teams of specialists work to return residents to the highest level of wellness through long-term care within the facility, or eventual discharge and return to society.

New Medico's Resocialization Programs accommodate patients with histories of social and/or behavioral problems. Integrated into personalized, constantly updated treatment plans, our services include evaluation, behavioral management, resocialization, community re-entry and vocational rehabilitation. Today our experienced staffs are serving the behaviorally impaired through these highly supportive and innovative programs.

To find out more about New Medico, call us toll-free at 1-800-343-1238. Or write us at 113 Broad Street, Lynn, MA 01902. Your psycho-social patients need help on the road to recovery. And we can help them take the first step.

New Medico *Resocialization Programs*

GIVE ADDED CHANCE TO YOUR RESPIRATOR DEPENDENT PATIENT TO IMPROVE DIFFUSION AND GET OFF THE RESPIRATOR

with
Effective Lobe to Lobe Aspirations with the Rockey-Thompson Bronchia
Suction Catheters will help.

AVAILABLE IN SETS OF 4:
F14, F16, F18, F20

For more information call or write:
from:
R.E.P. DIVISION PECK PACKAGING COR
528 Carter Street, New Canaan,
CONN. 06840 Tel. (203) 972-0119

MAINE

August 20–22. **Controversies in Obstetrics and Gynecology.** 16 Cat I Credits. Sonesta Hotel, Portland. Contact: Registrar, The American College of Obstetricians and Gynecologists, 600 Maryland Ave, SW, Suite 300 East, Washington, DC 20024-2588. Tel: 1-800-633-8811.

MASSACHUSETTS

August 7–9. **The Cape Cod Conference on Pediatrics.** The Dunfey Hyannis Hotel, Hyannis, Cape Cod. Contact: Rayna Saville, Coordinator, Continuing Medical Education, Children's Hospital, 219 Bryant St, Buffalo, NY 14222-2099. Tel: (716) 878-7630.

SOUTH CAROLINA

August 6–9. **Critical Care Pediatrics for the Primary Care Physician.** Mariner's Inn, Hilton Head Island. Contact: Debbie Shealy, South Carolina Chapter American Academy of Pediatrics, PO Box 11188, Columbia, SC 29211. Tel: (803) 798-6207.

VIRGINA

August 6–9. **Summer Retreat: Practical Issues in Primary Care.** The Cavalier Resort Hotel, Virginia Beach. *August 14–16.* **Primary Care of the Female Patient.** The Cavalier Resort Hotel, Virginia Beach. Contact: Kathy Martin, Office of Continuing Medical Education, Medical College of Virginia, Box 48, MCV Station, Richmond, VA 23298-0001. Tel: (814) 786-0494.

AROUND THE WORLD

CANADA

August 6–8. **Update in Obstetrics.** 16 Cat 1 Credits. Four Seasons Hotel, Vancouver, British Columbia. Contact: Registrar, American College of Obstetricians and Gynecologists, 600 Maryland Ave, SW, Suite 300 East, Washington, DC 20024. Tel: 1-800-533-8811.

August 21–23. **Advances in Pediatric Gastroenterology and Nutrition.** The Sheraton Centre, Toronto, Ontario. Contact: Rayna Saville, Coordinator, Continuing Medical Education, Children's Hospital, 219 Bryant Street, Buffalo, NY 14222-2099. Tel: (716) 878-7630.

ENGLAND

August 1–9. **The Best of Two Worlds in Family Medicine.** 26 Cat 1 Credits. London. Contact: Dawne Ryals, Ryals and Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

FRANCE

August 31–September 3. **Second International Symposium on Flavonoids in Biology and Medicine.** Contact: Elliott Middleton, Jr, MD, State University of New York at Buffalo, Department of Medicine, Buffalo General Hospital, 100 High St, Buffalo, NY 14203. Tel: (716) 845-2985. □

Cover to cover . . . . always good reading.

NEW YORK STATE JOURNAL OF MEDICINE

MEDICAL MALPRACTICE
AN OVERVIEW

Contents

NEW YORK STATE JOURNAL OF MEDICINE

CIGARETTE SMOKING, TOBACCO, AND HEALTH
INTERNATIONAL PERSPECTIVES

Contents

NEW YORK STATE JOURNAL OF MEDICINE

Contents

NEW YORK STATE JOURNAL OF MEDICINE

Contents

1987 CME Assembly Preliminary Program
See Page 13A

In 1987, the *New York State Journal of Medicine* promises to provide timely and stimulating commentary, challenging research papers, analysis of clinical trends, debate on topical issues, and in-depth coverage of medical news. The journal will make a welcome gift to a colleague or student in the United States or abroad.

A free copy of the July 1985 issue, a special issue on smoking, will be included with each subscription while the supply lasts.

SUBSCRIPTION ORDER FORM Clip and mail

Circulation Department
New York State Journal of Medicine
420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042

Name______________________________

Address______________________________

____

	1 Year	2 Years	3 Years
U.S. & Canada	☐ $25	☐ $45	☐ $60
Foreign	☐ $30	☐ $55	☐ $75

Payment required with order.

Medical Society of the State of New York
ANNUAL CME ASSEMBLY
APRIL 22-24, 1988
The New York Hilton, New York City
Combined With The Annual Meeting of
The House of Delegates
APRIL 21-24

"SELLING PRACTICES IS OUR BUSINESS"

Want to maximize the return on your investment? Before you buy or sell, call for an appraisal to assure the best financial and transfer terms. We are a full service practice broker, not simply a listing service. Since 1981, Countrywide has been instrumental in helping hundreds of doctors like yourself buy and sell their practices. We guide you through the entire sales process from initial meeting to closing. Our offices serve New York, New Jersey and New England.

Countrywide Business Brokerage, Inc.
319 East 24th Street, Suite 23-G, New York, NY 10010
(212) 686-7902 (203) 868-3666 (617) 232-1075

It's Here At Last...
A Choice of Two Low-Cost Practice Evaluations.

Basic Practice Evaluation
Provides a benchmark for your practice.

Expanded Option Evaluation
Provides 15 different option categories, with 180 evaluations, under a variety of conditions.

Here's Why You Should Consider a Pro-Tech Practice Evaluation:

- Analysis can be used in buying, selling, merging or dissolving of a practice.
- Analysis can serve to confirm other appraisals.
- Analysis is processed quickly — usually within 10 days.

Get the hard facts.

Name ____________ Phone ____________
Address ____________
City ____________ State ______ Zip ______

Pro-Tech Information Systems
© 1986 All Rights Reserved
240 Cedar Knolls Rd., Suite 310, Cedar Knolls, NJ 07927-9990

Exclusive Marketing Agents: Sarantos & Co., Inc.

DOCTOR DRUG DISPENSING

"Catching On"

- Greater convenience & savings for patients
- More effective patient treatment
- A new profit center. . . no additional overhead
- Computerized labeling, inventory and ordering

For more information please call or write:

DOCTORS'

DOCTORS' Rx
145 Algonquin Parkway
Whippany, NJ 07981

(201) 428-1117
1 (800) 752-5588

Index to Advertisers

Ayerst Laboratories 19A, 20A, 21A, 22
St. Clare's Hospital 7
Classified Advertising 6A, 12A, 18
Family Practice Recertification Magazine 8
Knoll Pharmaceuticals 11A, 12
Eli Lilly and Company 10
Marion Laboratories 1A, 2
Medico Resocialization Progran 15
Peck Packaging Corp. 16
Post Graduate Medicine 4
Roche Laboratories . . . . 4A, 5A, 3rd & 4th Cov
Step-Saver Medical Systems 9
Upjohn Company . . . . 2nd Cov

To show you how many hypertensives stayed on

INDERAL® LA

(PROPRANOLOL HCl)

after a major nationwide trial...

...we had
to find
just the
right room.

60,073 patients (90%) who started on INDERAL LA stayed on INDERAL LA.[1*]

Surprising? Not really.

Because most patients on INDERAL LA (propranolol HCl) don't even know it's working.

A recent double-blind, placebo-controlled, crossover study in 138 hypertensive patients[2] revealed that INDERAL LA has a side effects profile unsurpassed by atenolol or metoprolol—which shows how well-tolerated once-daily INDERAL LA can be.

Sole therapy or concomitant therapy? Fifty-nine percent of the time, INDERAL LA stood on its own.

The patients in the nationwide compliance trial were no different from yours. Generally when the antihypertensive regimen is complicated, compliance may become a problem. So, the effectiveness of INDERAL LA as once-daily monotherapy is a big plus. Of the remaining hypertensives in the program, 36% were treated merely with the addition of a diuretic to INDERAL LA.

For the noncompliant patients in your practice, INDERAL LA may well be the answer.

Almost 20,000 of the patients in the nationwide compliance trial were identified as having been noncompliant with their previous antihypertensive therapy. Their physicians reported that 88% showed improved compliance when placed on once-daily INDERAL LA.

Control, comfort, and compliance

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl) LONG ACTING CAPSULES

Like conventional INDERAL Tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree, and bronchial asthma.

*After a 30-day trial with INDERAL LA, physicians reported that 90% of the patients would remain on INDERAL LA.

The one you know best keeps looking better

NOW AVAILABLE...

NEW LOW DOSE

INDERAL LA 60 mg

(PROPRANOLOL HCl) LONG ACTING CAPSULES

Please see next page for brief summary of prescribing information

The one you know best keeps looking better

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. INDERAL LA is formulated to provide a sustained release of propranolol hydrochloride. INDERAL LA is available as 60 mg, 80 mg, 120 mg, and 160 mg capsules.

CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor-blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor-stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately.

INDERAL LA Capsules (60, 80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate. Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours. When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL Tablets. The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially.

INDERAL LA should not be considered a simple mg-for-mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period.

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension; it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies.

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris.

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache. The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use.

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope. INDERAL LA also improves exercise performance. The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary.

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first-degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL.

WARNINGS. CARDIAC FAILURE: Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible).

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following *abrupt* discontinuance of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned, the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

MAJOR SURGERY: The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, eg, dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA: Beta blockers should be used with caution in diabetic patients if a beta-blocking agent is required. Beta blockers may mask tachycardia occurring with hypoglycemia, but other manifestations such as dizziness and sweating may not be significantly affected. Following insulin-induced hypoglycemia, propranolol may cause a delay in the recovery of blood glucose to normal levels.

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol may change thyroid function tests, increasing T_4 and reverse T_3, and decreasing T_3.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case, this resulted after an initial dose of 5 mg propranolol.

PRECAUTIONS. GENERAL: Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies.

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

CLINICAL LABORATORY TESTS: Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Caution should be exercised when patients receiving a beta blocker are administered a calcium-channel-blocking drug, especially intravenous verapamil, for both agents may depress myocardial contractility or atrioventricular conduction. On rare occasions, the concomitant intravenous use of a beta blocker and verapamil has resulted in serious adverse reactions, especially in patients with severe cardiomyopathy, congestive heart failure or recent myocardial infarction.

Aluminum hydroxide gel greatly reduces intestinal absorption of propranolol.

Ethanol slows the rate of absorption of propranolol.

Phenytoin, phenobarbitone, and *rifampin* accelerate propranolol clearance.

Chlorpromazine, when used concomitantly with propranolol, results in increased plasma levels of both drugs.

Antipyrine and *lidocaine* have reduced clearance when used concomitantly with propranolol.

Thyroxine may result in a lower than expected T_3 concentration when used concomitantly with propranolol.

Cimetidine decreases the hepatic metabolism of propranolol, delaying elimination and increasing blood levels.

Theophylline clearance is reduced when used concomitantly with propranolol.

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY: Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug.

PREGNANCY: Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose.

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

NURSING MOTHERS: INDERAL is excreted in human milk. Caution should be exercised when INDERAL (propranolol HCl) is administered to a nursing woman.

PEDIATRIC USE: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy.

Cardiovascular: Bradycardia, congestive heart failure, intensification of AV block, hypotension, paresthesia of hands, thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type.

Central Nervous System: Light-headedness, mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia, visual disturbances; hallucinations, vivid dreams, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics. For immediate formulations, fatigue, lethargy, and vivid dreams appear dose related.

Gastrointestinal: Nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

Allergic: Pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress.

Respiratory: Bronchospasm.

Hematologic: Agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura.

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported.

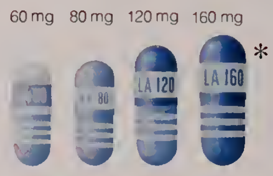
Miscellaneous: Alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl)

LONG ACTING CAPSULES



DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily. If patients are switched from INDERAL Tablets to INDERAL LA Capsules, care should be taken to assure that the desired therapeutic effect is maintained. INDERAL LA should not be considered a simple mg-for-mg substitute for INDERAL. INDERAL LA has different kinetics and produces lower blood levels. Retitration may be necessary, especially to maintain effectiveness at the end of the 24-hour dosing interval.

HYPERTENSION—*Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic. The dosage may be increased to 120 mg once daily or higher until adequate blood-pressure control is achieved. The usual maintenance dosage is 120 to 160 mg once daily. In some instances a dosage of 640 mg may be required. The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks.

ANGINA PECTORIS—*Dosage must be individualized.* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three- to seven-day intervals until optimal response is obtained. Although individual patients may respond at any dosage level, the average optimal dosage appears to be 160 mg once daily. In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established.

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS).

MIGRAINE—*Dosage must be individualized.* The initial oral dose is 80 mg INDERAL LA once daily. The usual effective dose range is 160-240 mg once daily. The dosage may be increased gradually to achieve optimal migraine prophylaxis. If a satisfactory response is not obtained within four to six weeks after reaching the maximal dose, INDERAL LA therapy should be discontinued. It may be advisable to withdraw the drug gradually over a period of several weeks.

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily.

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use.

*The appearance of these capsules is a registered trademark of Ayerst Laboratories.

REFERENCES:

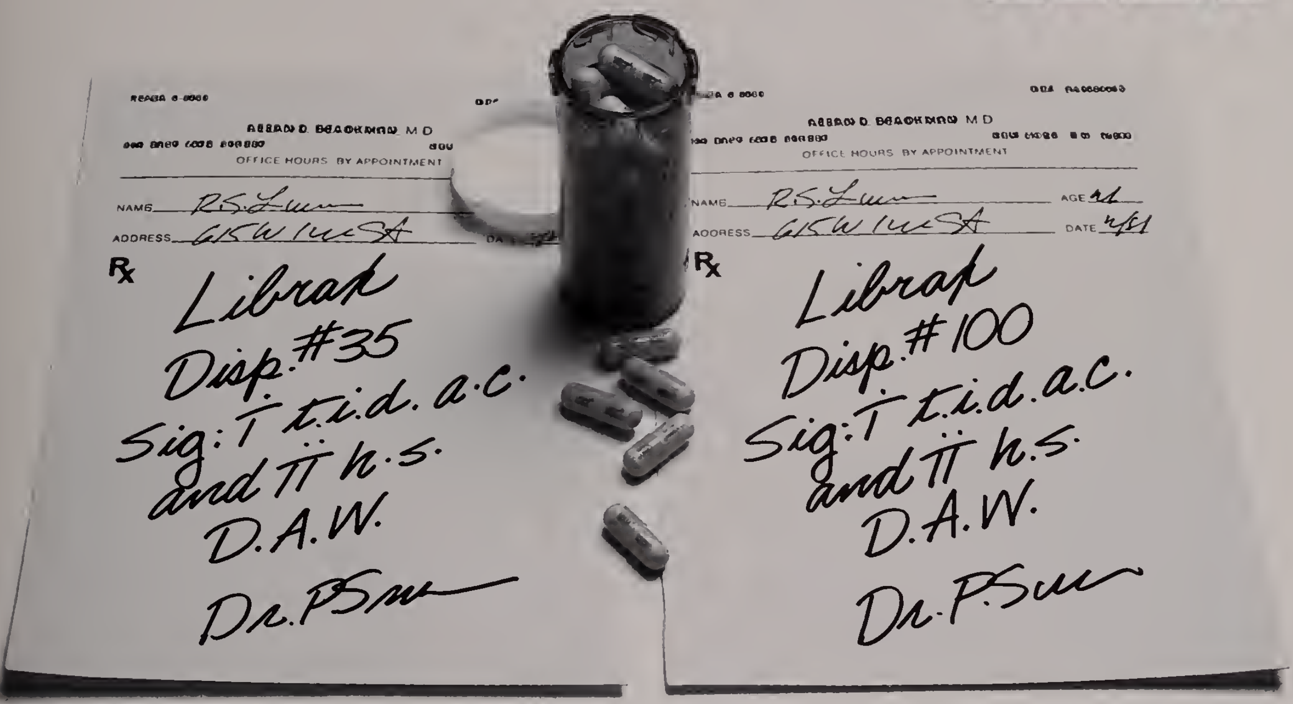
1. INDERAL LA National Compliance Evaluation Program. Data on file, Ayerst Laboratories.
2. Ravid M, Lang R, Jutrin I: The relative antihypertensive potency of propranolol, oxprenolol, atenolol, and metoprolol given once daily. *Arch Intern Med* 1985; 145:1321-1323.

R 7119/587

Ayerst®
AYERST LABORATORIES
New York, NY 10017

© 1987 Ayerst Laboratories

Specify Adjunctive LIBRAX®



NAME R.S. Lum
ADDRESS 615 W 1..St
Rx Librax
Disp. #35
Sig: $\dot{\text{T}}$ t.i.d. a.c.
and $\ddot{\text{TT}}$ h.s.
D.A.W.
Dr. P.S.

NAME R.S. Lum
AGE
ADDRESS 615 W 1..St
DATE
Rx Librax
Disp. #100
Sig: $\dot{\text{T}}$ t.i.d. a.c.
and $\ddot{\text{TT}}$ h.s.
D.A.W.
Dr. P.S.

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide.

Please consult complete prescribing information, a summary of which follows:

*

Indications: Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:
"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl/Roche) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

> ***Usage in Pregnancy:*** **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.
Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

PI. 0186

ROCHE®
Roche Products Inc.
Manati, Puerto Rico 00701

To insist
on the brand,
be sure to specify
"Dispense as Written."

When brain and bowel conflict...

It's time for the Peacemaker.

In irritable bowel syndrome* anxiety can aggravate intestinal symptoms, which may further intensify anxiety — a distressing cycle of brain/bowel conflict. Librax intervenes with two well-known compounds. The Librium® (chlordiazepoxide HCl/Roche) component safely relieves anxiety. And Quarzan® (clidinium bromide/Roche) provides antisecretory and antispasmodic action to relieve discomfort associated with intestinal hypermotility.

Dual action — for peace between brain and bowel. Because of possible CNS effects, caution patients about engaging in activities requiring complete mental alertness. Specify Adjunctive

LIBRAX®

Each capsule contains 5 mg chlordiazepoxide HCl
and 2.5 mg clidinium bromide

*Librax has been evaluated as possibly effective as adjunctive therapy in the treatment of peptic ulcer and the irritable bowel syndrome.

Copyright © 1987 by Roche Products Inc. All rights reserved. Please see summary of prescribing information on adjacent page.

STACKS

NEW YORK STATE JOURNAL OF MEDICINE

AUGUST 1987 • Volume 87, Number 8

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA
M.D.S.
AUG 12 1987

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

Contents

COMMENTARIES

Supervising interns and residents and limiting their working hours 425
PASCAL JAMES IMPERATO, MD

Humanism and medical ethics in a technological age 427
STEPHEN NORDLICHT, MD

Group B streptococcal infection in 1987 428
DENNIS L. STEVENS, MD, PHD

RESEARCH PAPERS

Low serum triiodothyronine in diabetic ketoacidosis 431
JAMES TURRO, MD, MILFORD FULOP, MD

Cigarette smoking among Ethiopian health professionals and students 433
ZEIN AHMED ZEIN, DR MED, MPH

HISTORY

Arthur Donaldson Smith, MD: Physician, explorer, naturalist, and diplomat—Part V 436
PASCAL JAMES IMPERATO, MD

CASE REPORTS

Group B streptococcal osteomyelitis in an adult 466
ALEXANDER A. MCMEEKING, MD; ROBERT HOLZMAN, MD; DIANE DESIDERIO, MS; BRUCE HANNA, PHD

Unusual thoracic myelopathy in two patients with Hodgkin disease 467
ROBERT L. RUFF, MD, PHD, ROBERT SCHELL, MD

Malignant angioendotheliomatosis manifesting as ascending spinal cord dysfunction 470
PATRICK CHEN, MD, PHD; ERIC GAETJENS, PHD; JOANNA SHER, MD; MAHENDRA SOMASUNDARAM, MD; TI-KAI LEE, MD; SHU-WEN HOW, MD; NAN-HWA CHO, MD

LETTERS TO THE EDITOR

Review of *Once a Doctor, Always a Doctor* 473
HEINZ HARTMANN, MD

Review of physicians' memoirs 473
CHARLES A. PERERA, MD

Dr Bluestone's essay review 473
FRANK A. DISNEY, MD

Malpractice by nonphysician healers 473
JEAN-MARC NORES, MD; JEAN-MICHEL REMY, MD; ANDRE D. NENNA, MD; PASCAL REYGAGNE, MD

Colostomy bag simulating bony metastases 474
KUSUM L. KUMAR, MD; DAVID L. SMITH, MD; LINDA M. LUCAS, MD

IMMUNIZATION GUIDELINES—1987 476

NEWS BRIEFS 479
BOOKS RECEIVED 481
OBITUARIES 483
GUIDELINES FOR AUTHORS 486
MEDICAL MEETINGS AND LECTURES 4A

Motrin® 800 TABLETS mg
ibuprofen

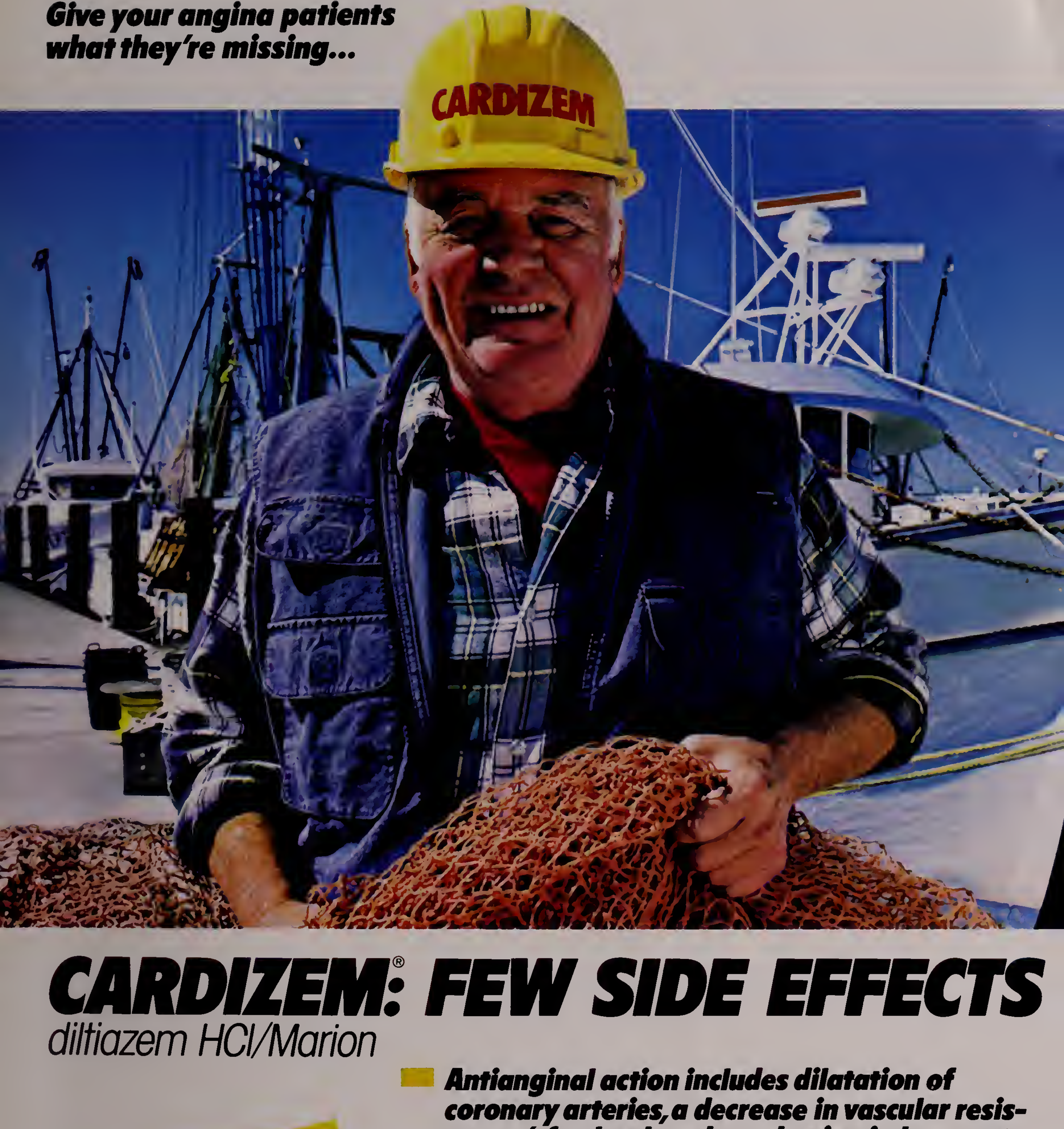
Extra strength
Convenience
Economy

Upjohn
A Century
of Caring
1886·1986

©1986 The Upjohn Company

J-6138 January 1986

Give your angina patients what they're missing...



CARDIZEM®: FEW SIDE EFFECTS

diltiazem HCl/Marion

90 mg GREATER DOSAGE FLEXIBILITY NEW 120 mg

- ***Antianginal action includes dilatation of coronary arteries, a decrease in vascular resistance/afterload, and a reduction in heart rate***
- ***Proven efficacy when used alone in angina[1]***
- ***Compatible with other antianginals[2,3]****
- ***A safe choice for angina patients with coexisting hypertension, asthma, COPD, or PVD[4,5]***

****See Warnings and Precautions.***

Please see brief summary of prescribing information on the next page.

CARDIZEM® diltiazem HCl/Marion

FEW SIDE EFFECTS IN ANTIANGINAL THERAPY

60 mg tid or qid

Brief Summary
Professional Use Information

CARDIZEM®
(diltiazem HCl) 30 mg and 60 mg Tablets

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1,243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt).
   Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, significant elevations in enzymes such as alkaline phosphatase, CPK, LDH, SGOT, SGPT, and other symptoms consistent with acute hepatic injury have been noted. These reactions have been reversible upon discontinuation of drug therapy. The relationship to CARDIZEM is uncertain in most cases, but probable in some. (See PRECAUTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. Diltiazem is excreted in human milk. One report suggests that concentrations in breast milk may approximate serum levels. If use of CARDIZEM is deemed essential, an alternative method of infant feeding should be instituted.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences as well as their frequency of presentation are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%). In addition, the following events were reported infrequently (less than 1%):

Cardiovascular:	Angina, arrhythmia, AV block (first degree), AV block (second or third degree — see conduction warning), bradycardia, congestive heart failure, flushing, hypotension, palpitations, syncope.
Nervous System:	Amnesia, gait abnormality, hallucinations, insomnia, nervousness, paresthesia, personality change, somnolence, tinnitus, tremor.
Gastrointestinal:	Anorexia, constipation, diarrhea, dysgeusia, dyspepsia, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH (see hepatic warnings), vomiting, weight increase.
Dermatologic:	Petechiae, pruritus, photosensitivity, urticaria.
Other:	Amblyopia, dyspnea, epistaxis, eye irritation, hyperglycemia, nasal congestion, nocturia, osteoarticular pain, polyuria, sexual difficulties.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: alopecia, gingival hyperplasia, erythema multiforme, and leukopenia. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

Issued 7/86

See complete Professional Use Information before prescribing.

References: 1. Pepine CJ, Feldman RL, Hill JA, et al: Clinical outcome after treatment of rest angina with calcium blockers: Comparative experience during the initial year of therapy with diltiazem, nifedipine, and verapamil. Am Heart J 1983; 106(6):1341-1347. **2.** Shapiro W: Calcium channel blockers: Actions on the heart and uses in ischemic heart disease. Consultant 1984,24(Dec):150-159. **3.** Johnston DL, Lesoway R, Humen DP, et al: Clinical and hemodynamic evaluation of propranolol in combination with verapamil, nifedipine and diltiazem in exertional angina pectoris: A placebo-controlled, double-blind, randomized, crossover study. Am J Cardiol 1985,55:680-687. **4.** Cohn PF, Braunwald E: Chronic ischemic heart disease, in Braunwald E (ed): Heart Disease: A Textbook of Cardiovascular Medicine, ed 2. Philadelphia, WB Saunders Co, 1984, chap 39. **5.** Schroeder JS: Calcium and beta blockers in ischemic heart disease: When to use which. Mod Med 1982,50(Sept):94-116.

Another patient benefit product from

PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC.
KANSAS CITY, MO 64137

0246M6

NEW YORK STATE
JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
ELIZA H. CALDWELL, MD
JOSEPH F. MURATORE, MD
JOHN T. PRIOR, MD
GITA S. SINGH*
STANFORD WESSLER, MD

*Medical student

Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Consulting Editor and Book Review Editor	RICHARD B. BIRRER, MD
Consulting Editor	NAOMI R. BLUESTONE, MD, MPH
Consulting Editor	CARL POCHEDLY, MD
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Managing Editor	CAROL L. MOORE
Advertising Director	ROBERT MILLER
Consulting Medical Writer	VICKI GLASER
Editorial Assistant	MILDRED J. AREMANN
Secretary	ELIZABETH J. SOMERS
Librarian	ELLA ABNEY
Assistant Librarian	ELEANOR BURNS

ASSOCIATE EDITORIAL BOARD 1987

MICHAEL E. BERLOW, MD
RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MYLES S. GOMBERT, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
JAMES M. MORRISSEY, ESQ
STEPHEN NORDLICHT, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. United Media Associates, Inc, 16 Bruce Park Ave, Greenwich, CT 06830. Telephone: (203) 661-9702.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

SAMUEL M. GELFAND, MD,	*President*
JOHN A. FINKBEINER, MD,	*Past-President*
CHARLES D. SHERMAN, JR, MD,	*President-Elect* *Vice-President*
JOHN H. CARTER, MD,	*Secretary*
GEORGE LIM, MD,	*Assistant Secretary*
MORTON KURTZ, MD,	*Treasurer*
ROBERT A. MAYERS, MD,	*Assistant Treasurer*
CHARLES N. ASWAD, MD,	*Speaker*
SEYMOUR R. STALL, MD,	*Vice-Speaker*

Councilors

Term Expires 1988

JAMES H. COSGRIFF, JR, MD,	Erie
RICHARD A. HUGHES, MD,	Warren
ANTONIO F. LASORTE, MD,	Broome
SIDNEY MISHKIN, MD,	Nassau

Term Expires 1989

ROBERT E. FEAR, MD,	Suffolk
STANLEY L. GROSSMAN, MD,	Orange
THOMAS D. PEMRICK, MD,	Rensselaer
RALPH E. SCHLOSSMAN, MD,	Queens

Term Expires 1990

DAVID M. BENFORD, MD,	New York
DUANE M. CADY, MD,	Onondaga
WILLIAM A. DOLAN, MD,	Monroe
ROBERT E. GORDON, MD,	Kings

Resident Councilor (representing the resident physician membership)

KATHLEEN E. SQUIRES, MD,	New York

Student Councilor (representing the medical student membership)

MICHAEL PACIOREK,	Onondaga

Trustees

RICHARD D. EBERLE, MD, (*Chairman*)	Onondaga
EDGAR P. BERRY, MD,	New York
JAMES M. FLANAGAN, MD,	Wayne
ALLISON B LANDOLT, MD,	Westchester
DANIEL F. O'KEEFFE, MD,	Warren
BERNARD J. PISANI, MD,	New York
VICTOR J. TOFANY, MD,	Monroe

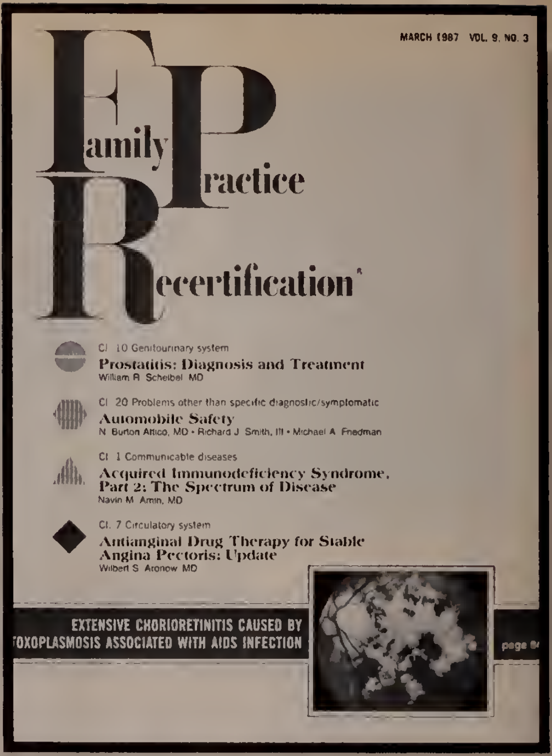
Executive Vice-President	DONALD F. FOY
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Vice-President Emeritus	GEORGE J. LAWRENCE, JR, MD
Director, Division of Policy Coordination	IRMA A ERICKSON
Director, Division of Scientific Publications	PASCAL JAMES IMPERATO, MD
Director, Division of Communications	EDWARD A HYNES
Director, Division of Computer Services	IVAN H. NAJMAN
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Governmental Affairs	GERARD L CONWAY, ESQ
General Counsel	DONALD R. MOY, JD
Director, Division of Management and Meeting Services	ROBERT MILLER
Director, Division of Membership Support Services	EUNICE M. SKELLY
Director, Division of Scientific and Educational Activities	PETER B EARNSWORTH, MD
Director, Division of Socio-Medical Economics	MARIO V MENGHINI
Medical Director, Committee for Physicians' Health	BARTON A. HARRIS, MD
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York. Copyright © 1987, Medical Society of the State of New York. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.



MARCH 1987 VOL. 9, NO. 3

Family Practice Recertification

Cl 10 Genitourinary system
Prostatitis: Diagnosis and Treatment
William R Scheibel MD

Cl 20 Problems other than specific diagnostic/symptomatic
Automobile Safety
N Burton Attico, MD • Richard J Smith, III • Michael A Friedman

Cl 1 Communicable diseases
Acquired Immunodeficiency Syndrome, Part 2: The Spectrum of Disease
Navin M Amin, MD

Cl. 7 Circulatory system
Antianginal Drug Therapy for Stable Angina Pectoris: Update
Wilbert S Aronow MD

EXTENSIVE CHORIORETINITIS CAUSED BY TOXOPLASMOSIS ASSOCIATED WITH AIDS INFECTION

The complete journal for family practice physicians

- Reaches 79,000 family physicians monthly
- Presents the most commonly seen patient problems in family practice
- Written by physicians for physicians
- The most current clinical updates in:
    - Cardiology
    - Diabetes
    - Pediatrics
    - Ob/Gyn
    - Psychiatry
    - Gastroenterology
- Provides 20 hours of CME Category 1 Credit

PRACTICAL ▪ CLINICAL ▪ EDUCATIONAL ▪ CURRENT

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

SEPTEMBER 1987

AROUND THE STATE

MANHATTAN

Sept. 14–16. **Lyme Disease and Related Disorders.** Marriott Marquis. Contact: Conference Dept, The New York Academy of Sciences, 2 East 63rd Street, New York, NY 10021. Tel: (212) 838-0230.

NASSAU

Sept. 30. **Rheumatology Update 1987.** 4½ Cat 1 Credits. Long Island Marriott Hotel, Uniondale. Contact: Sheldon P. Blau, MD, Department of Medicine/Division of Rheumatology, Nassau County Medical Center, 2201 Hempstead Tpke, East Meadow, NY 11510. Tel: (516) 542-4015.

SYRACUSE

Sept. 17. **Diabetes: Current Concepts and Challenges.** 6 Cat 1 Credits. Marriott, Syracuse. Contact: Laurie J. Kochanek, American Diabetes Association, PO Box 1037, Syracuse, NY 13201. Tel: (315) 472-9111.

AROUND THE NATION

CALIFORNIA

Sept. 22–26. **Doppler Ultrasound: Vascular and Abdominal Applications.** 30 Cat 1 Credits. San Francisco. Contact: Lisa Krehbiel, Institute for Medical Studies, 30131 Town Center Dr, Suite 215, Laguna Niguel, CA 92677. Tel: (714) 495-4499.

Sept. 28–Oct. 2. **The Physician in Management Seminar 1.** 31 Cat 1 Credits. The Doubletree at Fisherman's Wharf, Monterey. Contact: Sherry Mason, American Academy of Medical Directors, 4830 W Kennedy Blvd, Suite 648, Tampa, FL 33609. Tel: (813) 873-2000.

INDIANA

Sept. 16–18. **Research Design and Medical Biostatistics for the Clinician.** Hilton on the Circle, Indianapolis. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

LOUISIANA

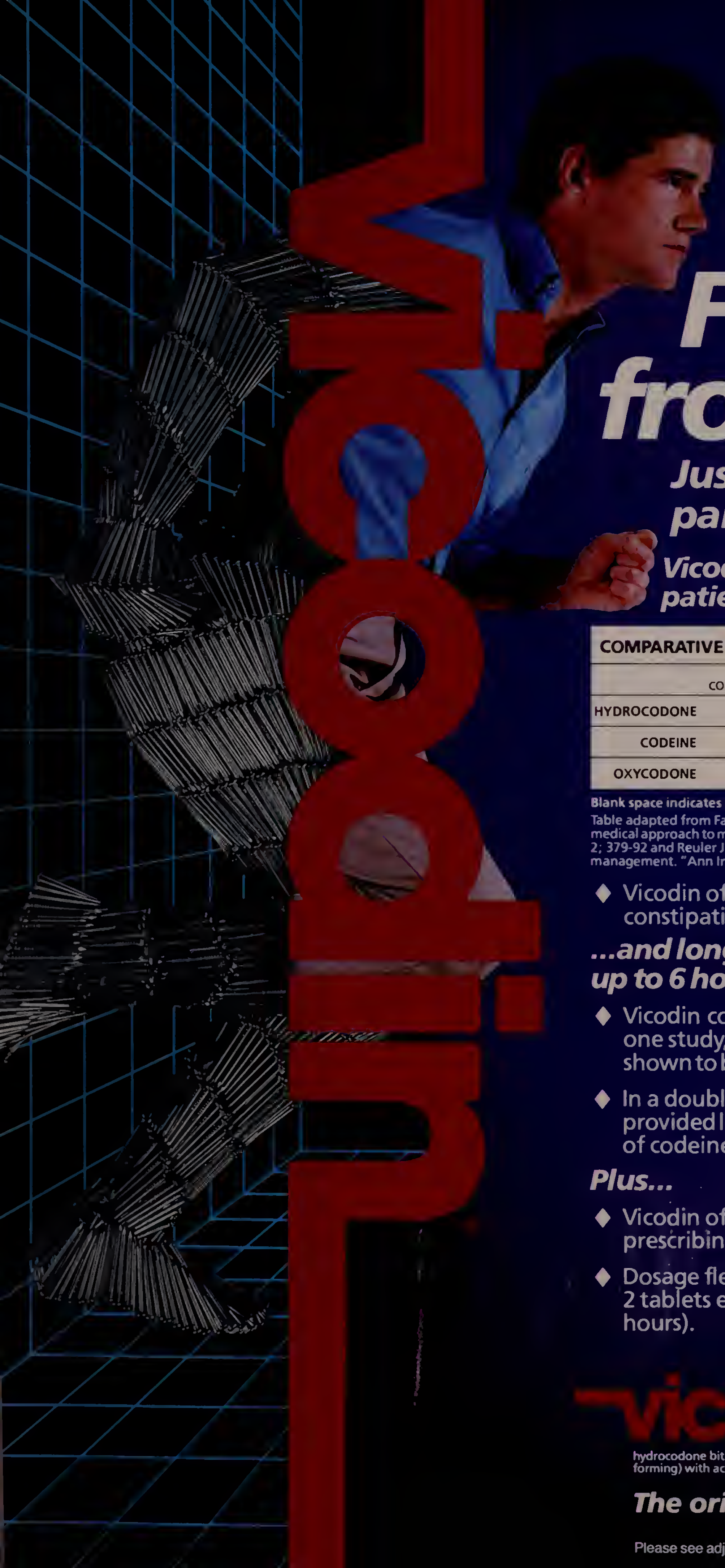
Sept. 13–19. **37th International Seminar on Leprosy.** National Hansen's Disease Center, Carville. Contact: Dr W. Felton Ross, American Leprosy Missions, One Broadway, Elmwood Park, NJ 07407. Tel: (201) 794-8650.

MASSACHUSETTS

Sept. 1–3. **Risk Analysis in Environmental and Occupational Health—With Emphasis on Carcinogenesis.** 18 Cat 1 Credits. Harvard University. Contact: Office of Continuing Education, Harvard School of Public Health, 677 Huntington Ave, Boston, MA 02115. Tel: (617) 732-1171.

Sept. 28–Oct. 2. **Managing Physicians—The Relationship Between Physicians and Health Delivery Organiza-**

(*continued on p 10A*)



Freedom from pain

Just one part of pain relief therapy.

Vicodin® provides greater patient acceptance

COMPARATIVE PHARMACOLOGY OF THREE ANALGESICS	CONSTIPATION	RESPIRATORY DEPRESSION	SEDATION	EMESIS	PHYSICAL DEPENDENCE
HYDROCODONE		X			X
CODEINE	X	X	X	X	X
OXYCODONE	XX	XX	XX	XX	XX

Blank space indicates that no such activity has been reported.

Table adapted from Facts and Comparisons (Nov.) 1984 and Catalano RB. The medical approach to management of pain caused by cancer. "Semin Oncol" 1975, 2; 379-92 and Reuler JB, et. al. The chronic pain syndrome: misconceptions and management. "Ann Intern Med" 1980; 93; 588-96.

- Vicodin offers: less nausea, less sedation, less constipation.

...and longer lasting pain relief– up to 6 hours.

- Vicodin contains hydrocodone not codeine. In one study, 10 mg. of hydrocodone alone was shown to be as effective as 60 mg. of codeine.[1]
- In a double-blind study, Vicodin (2 tablets), provided longer lasting pain relief than 60 mg. of codeine.[2]

Plus...

- Vicodin offers the convenience of CIII prescribing.
- Dosage flexibility–1 tablet every 6 hours or 2 tablets every 6 hours (up to 8 tablets in 24 hours).

1. Hopkinson JH III: *Curr Ther Res* 24: 503-516, 1978
2. Beaver, WT *Arch Intern Med,* 141:293-300, 1981.

vicodin®

hydrocodone bitartrate 5 mg. (Warning: May be habit forming) with acetaminophen 500 mg.

The original hydrocodone analgesi

Please see adjacent page for brief summary of prescribing information.

vicodin ℂIII

hydrocodone bitartrate 5mg (Warning May be habit forming). and acetaminophen 500mg

INDICATIONS AND USAGE: For the relief of moderate to moderately severe pain.
CONTRAINDICATIONS: Hypersensitivity to acetaminophen or hydrocodone
WARNINGS
Drug Abuse and Dependence: VICODIN is subject to the Federal Controlled Substances Act (Schedule III). Psychic dependence, physical dependence and tolerance may develop upon repeated administration of narcotics; therefore, VICODIN should be prescribed and administered with the same caution appropriate to the use of other oral-narcotic-containing medications.
Respiratory Depression: At high doses or in sensitive patients, hydrocodone may produce dose-related respiratory depression by acting directly on brain stem respiratory centers Hydrocodone also affects centers that control respiratory rhythm, and may produce irregular and periodic breathing
Head Injury and Increased Intracranial Pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a preexisting increase in intracranial pressure Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.
Acute Abdominal Conditions: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions.
PRECAUTIONS
Special Risk Patients: VICODIN should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture.
Information for Patients: VICODIN, like all narcotics, may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery; patients should be cautioned accordingly
Cough Reflex: Hydrocodone suppresses the cough reflex, caution should be exercised when VICODIN is used postoperatively and in patients with pulmonary disease
Drug Interactions: The CNS-depressant effects of VICODIN may be additive with that of other CNS depressants. When combined therapy is contemplated, the dose of one or both agents should be reduced. The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone. The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus
Usage in Pregnancy: Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose. There are no adequate and well-controlled studies in pregnant women. VICODIN should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.
Nonteratogenic Effects: Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent. The intensity of the syndrome does not always correlate with the duration of maternal opioid use or dose.
Labor and Delivery: Administration of VICODIN to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used
Nursing Mothers: It is not known whether this drug is excreted in human milk, therefore, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother
Pediatric Use: Safety and effectiveness in children have not been established
ADVERSE REACTIONS
Central Nervous System: Sedation, drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, dizziness, psychic dependence, mood changes.
Gastrointestinal System: Nausea and vomiting may occur; they are more frequent in ambulatory than in recumbent patients Prolonged administration of VICODIN may produce constipation.
Genitourinary System: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported
Respiratory Depression: (See WARNINGS.)
DOSAGE AND ADMINISTRATION: Dosage should be adjusted according to the severity of the pain and the response of the patient However, tolerance to hydrocodone can develop with continued use, and the incidence of untoward effects is dose related The usual dose is one tablet every six hours as needed for pain (If necessary, this dose may be repeated at four-hour intervals) In cases of more severe pain, two tablets every six hours (up to eight tablets in 24 hours) may be required

Revised January, 1987 MR 1987 5786

Knoll Pharmaceuticals
A Unit of BASF K&F Corporation
Whippany, New Jersey 07981

BASF Group

knoll®

Classified Advertising

PHYSICIANS WANTED

EMERGENCY MEDICINE POSITIONS—Full/part time emergency medicine physicians sought by multi state professional association for openings in metropolitan NY, PA, MD, DC, FL, New England and throughout U.S. Contact or send CV to Liberty Healthcare Corporation, 399 Market Street, Suite 400, Philadelphia, PA 19106, (215) 592-7400 or outside Pa (800) 331-7122.

ARIZONA-BASED PHYSICIAN RECRUITMENT firm has quality opportunities coast to coast. Available positions in most primary care and surgical specialties to include OB/GYN, Orthopedics, ER, and ENT. "Quality Physicians for Quality Clients since 1972." Call 602-990-8080; or send CV to: Mitchell & Associates, Inc., PO Box 1804, Scottsdale, AZ 85252.

PRIMARY CARE PRACTITIONERS (BC/BE) needed for unique practice situation, with ownership potential, in Long Island. Please reply with full particulars including telephone number to Dept. 443 c/o NYSJM.

BOARD CERTIFIED INTERNIST wanted for community of 10,000 (service area 45,000) in the lush Yakima Valley of central Washington. Financial assistance. Excellent income potential. Share coverage with other internists. First rate school system. Outdoor recreation second to none. Contact Prosearch, 305 NE 102nd Avenue, Portland, Oregon 97220. (503) 256-2070.

NEW YORK, WESTERN: Seeking primary care trained physicians for full time Emergency Department positions. Moderate volume. Attractive hourly compensation, plus malpractice insurance, Director position available. Contact: Emergency Consultants, Inc., 2240 S. Airport Road, Room 42, Traverse City, MI 49684: 1-800-253-1795 or in Michigan 1-800-632-3496.

NEW YORK, BUFFALO—Seeking full-time and part-time physicians residency trained in emergency medicine or primary specialty for 32,000 annual volume emergency department. Directorship available. Attractive compensation, malpractice insurance & benefit package. Contact: Emergency Consultants, Inc., 2240 S. Airport Rd., Room 42, Traverse City, MI 49684; 1-800-253-1795 or in Michigan 1-800-632-3496.

CHIEF/AMBULATORY CARE SERVICES—Barnert Memorial Hospital Center, a 290 bed community hospital in Paterson, New Jersey, is seeking to fill the newly-created position of Chief of Ambulatory Care Services to provide clinical leadership to the existing ambulatory care programs and to work with management to develop new ambulatory care programs. Applicants must be board-certified in Internal Medicine, Family Practice, or Preventive Medicine, and have experience in the delivery and management of ambulatory care. Send resumes to Alexander & Ebert, c/o Julie Wolman, 405 S. 24th Street, Philadelphia, Pa. 19146.

PHYSICIANS WANTED—CONT'D

FAMILY PRACTICE—Urban/rural community in south central New York seeks qualified physicians for hospital based, structured hours position or private practice association. 300+ bed progressive acute care facility with serving area of 150,000. Close proximity to Finger Lakes provides summer/winter recreation. Excellent family environment. Guarantee plus benefits. Reply Dept. #446 c/o NYSJM.

PRACTICES AVAILABLE

6½ YEAR INTERNAL MEDICINE PRACTICE FOR SALE in Dutchess County. Rapidly growing area, near two hospitals. Reasonable sale price. Reply Dept. 444 c/o NYSJM.

EQUIPMENT

50% OFF PREVIOUSLY OWNED MEDICAL—Laboratory, x-ray, ultrasound equipment. Buy, Sell, Broker, Repair. Appraisals by certified surgical consultants. Medical Equipment Resale & Repair, 24026 Haggerty Road, Farmington Hills, MI 48018. 1-800-247-5826. (313) 477-6880.

DISCOUNT HOLTZER SCANNING SERVICES starting at $35.00. Spacelabs holter recorders available from $1,275.00. Smallest and lightest holters update. Fast service (24–48 hours) turnover. Hook-up kits starting at $4.95. Special introductory offer of three free tests at the purchase or lease of the holter. Stress test electrodes available at 34 cents. Scanning paper available at $18.95. Cardiologist overread available at $15.00. If interested call (301) 870-3626.

EQUIPMENT FOR SALE: Ritter hydraulic chair, Sorensen De-Luxe treatment unit and cabinet, Bausch & Lomb Ferree-Rand perimeter, Marita cuspidor, Bausch & Lomb examiners stool, Castle sterilizer, two Bausch & Lomb equipment stands—all in excellent condition. Contact Jerome Lehner, M.D., 141 Hooker Avenue, Poughkeepsie, New York 12601.

FLUOROSCOPIC PICKER X-RAY MACHINE, 12 years old, with automatic colimation and phototiming and upright Bucky with mirror image intensifier but no television. In excellent working condition. Please call (313) 358-3410 9 A.M. to 5 P.M.

AUTO-OPHTHALMOSCOPE (proper.). Centrifuge, (60 cycle, 6 whole), quick-chem (Dow Bio Chem. Chem-Syst.) with two heating block and miscellaneous items. Call (607) 748-1255 or 754-7586, 12 to 3 P.M.

REAL ESTATE FOR SALE OR RENT

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

HIV Positive Confirmed. Then What?

AIDS is tough to treat. Ruthless to an increasing patient population, it challenges physicians and nurses as never before in their commitment to quality care.

The Spellman Center for HIV-Related Disease at St. Clare's Hospital and Health Center in New York City, accepts the challenge posed by AIDS and HIV infection. The first facility in the tri-state region to devote its resources solely to the care of AIDS patients, the Spellman Center designates its highest priority as compassionate patient care. A dedicated staff provides comprehensive medical, nursing, psychosocial, and dental treatment in an atmosphere that is warm and understanding.

The Spellman Center—a 60-bed Acute Care Unit, Emergency Service, Outpatient, Ambulatory Infusion and Dental Clinics—accepts referrals of patients with CDC-confirmed AIDS or ARC (AIDS-related complex), and those who are seropositive for HIV. Spellman also reaches out to offer testing, counseling and treatment to persons at risk for AIDS.

If you need consultation concerning your patients, or more information, please call Dr. Debra Spicehandler, the Medical Director, at (212) 307-1161.

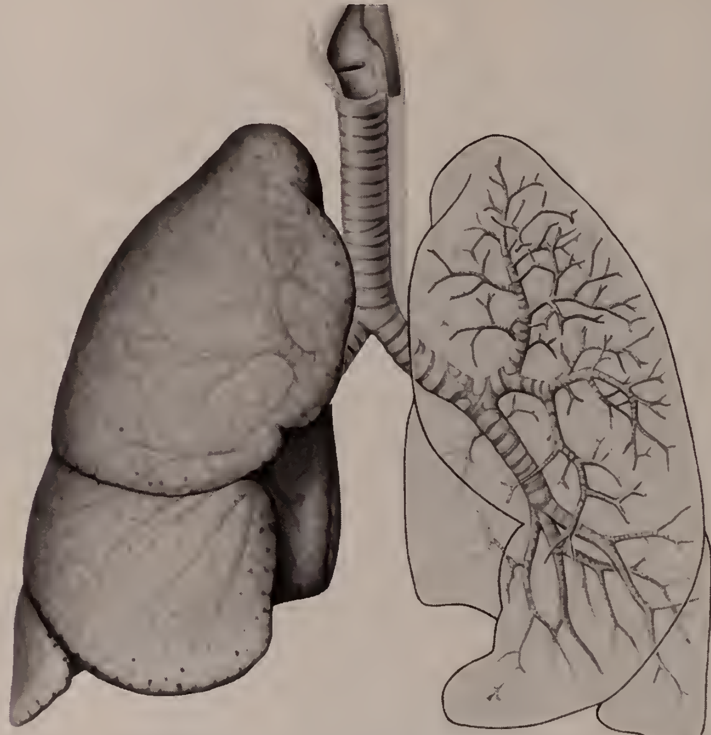
The Spellman Center also maintains an information hotline for patients with questions about AIDS: 1-800-433-AIDS.

The Spellman Center
for HIV-Related Disease
A Designated AIDS Treatment Facility
St. Clare's Hospital and Health Center

415 W 51st St
New York, NY 10019

An Affiliate of New York Medical College

Consider the causative organisms…



Ceclor® cefaclor

250-mg Pulvules® t.i.d.

offers effectiveness against the major causes of bacterial bronchitis

Haemophilus influenzae, Streptococcus pneumoniae
(ampicillin-susceptible and ampicillin-resistant)

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

Ceclor® (cefaclor)

Summary. Consult the package literature for prescribing information.

Indications: Lower respiratory infections, including pneumonia, caused by susceptible strains of *Streptococcus pneumoniae, Haemophilus influenzae*, and *Streptococcus pyogenes* (group A β-hemolytic streptococci).

Contraindication:
Known allergy to cephalosporins.

Warnings:
CECLOR SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS

Administer cautiously to allergic patients.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.

Precautions:

- Discontinue Ceclor in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of nonsusceptible organisms.
- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- Ceclor should be administered with caution in the presence of markedly impaired renal function. Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk. Exercise caution in prescribing for these patients.

Adverse Reactions: (percentage of patients)

Therapy-related adverse reactions are uncommon. Those reported include:

- Gastrointestinal (mostly diarrhea): 2.5%.
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
- Hypersensitivity reactions (including morbilliform eruptions, pruritus, urticaria, and serum-sickness-like reactions that have included erythema multiforme [rarely, Stevens-Johnson syndrome] or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever): 1.5%; usually subside within a few days after cessation of therapy. Serum-sickness-like reactions have been reported more frequently in children than in adults and have usually occurred during or following a second course of therapy with Ceclor. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.
- Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.
- As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely.
- Rarely, reversible hyperactivity, nervousness, insomnia, confusion, hypertonia, dizziness, and somnolence have been reported.
- Other: eosinophilia, 2%; genital pruritus or vaginitis, less than 1%; and, rarely, thrombocytopenia.

Abnormalities in laboratory results of uncertain etiology

- Slight elevations in hepatic enzymes.
- Transient fluctuations in leukocyte count (especially in infants and children).
- Abnormal urinalysis; elevations in BUN or serum creatinine.
- Positive direct Coombs' test.
- False-positive tests for urinary glucose with Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (glucose enzymatic test strip, Lilly).

[072886R]

PA 8794 AMP

©1987, ELI LILLY AND COMPANY

Lilly

Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285.

Eli Lilly Industries, Inc
Carolina, Puerto Rico 00630

700241

NEW YORK STATE
JOURNAL OF MEDICINE

August 1987 • Volume 87, Number 8

COMMENTARIES

Supervising interns and residents and limiting their working hours

The on-call labor and time demands placed on interns and residents have frequently varied between institutions and the services within them. The lot of interns and residents on surgery and obstetrics has often been a tougher one than that of future internists and pediatricians. Yet in some institutions, rotating through medicine and pediatrics can be as grueling. It is no secret that some fourth-year medical students give significant weight to the labor demands of various training programs when deciding where to apply. Being on call every other night and weekend is a far cry from being on every third or fourth night and weekend. Also, the labor demands placed on house staff while on duty are factors fourth-year students take into consideration. Academic programs tend to be more labor- and time-demanding, and those who opt for them do so because they seek their excellence. Intern and resident time and work loads are molded by a variety of interrelated factors including the number of resident positions, the number of beds on the service, characteristics of the patient population, bed occupancy rates, and others.

Excessive working hours and labor-intensive responsibilities have long been enshrined in American graduate medical education as the just and good lot of interns and residents. What better way to make good doctors of them than by subjecting them early on to the physical rigors which will become a permanent feature of their lives. This "plantation mentality," which has imposed high levels of physical and mental hardship on generations of house officers, also coincidentally well serves the interests of hospitals, which need house staff manpower to function, and private attending physicians, who without it would end up carrying additional burdens for the care of their patients. The quid pro quo for the house staff is learning and experience, the two noble end products most frequently cited by those who are called upon to defend the labor demands of graduate medical education. Yet the law of diminishing return should have long ago told us that beyond a certain point little or nothing is added to the knowledge and skills of house officers who are obtunded by lack of sleep. In a similar vein, the performance by house staff of well-mastered routine procedures can scarcely be viewed as enhancing their education. It is clear that there comes a point when interns and residents function more for the benefit of the institutions they serve than for that of either their patients or themselves.

A second and related issue is that of supervision of house staff. The extension of third-party coverage to large numbers of Americans in the mid-1960s, particularly through Medicaid and Medicare, posed a problem for teaching hospitals, which heavily relied for teaching on patients without medical insurance or the ability to pay, who were called "ward," "charity," or "service" patients. With the extension of medical insurance to these patients, they became entitled to the services of a private physician. Residency program directors and hospital administrators, often responding to the direction of residency review committees, established systems to de-privatize as many patients to the greatest extent possible with regard to their actual care, but not their insurance. In many hospitals this translated into inpatients being placed under the care of the house staff, while the private attending who admitted the patient in the first place was shifted to a peripheral position of noncontemporaneous supervision. Interns and residents, who enjoy the heady experience of being in charge and of practicing their newly acquired technical skills, rarely complain about this arrangement.[1] In some hospitals private attending physicians could not write orders on their own patients. Only interns and residents could. In other hospitals attending physicians could only write orders after consultation with interns and residents. This arrangement, the details of which have varied over time and between different hospitals, satisfied the requirements of residency review committees, the needs and wants of house staff, and was eventually seen by many attending physicians as not so bad after all. For while interns and residents provided the care, the attending physicians were spared much of the labor associated with it, yet were paid their fees. As these shifts in the division of labor and responsibility moved forward, so too did medical progress. Newer diagnostic technologies and therapeutic interventions progressively made the salvage and prolongation of life possible in situations where only a few years before patients would have died. These technologies and interventions have made a major impact on the working lives of interns and residents. They have greatly increased the time and labor demands on house staff, which translate into fewer hours of sleep and increased hours of intense and often grueling work with the mechanical intermediaries that sustain life. The few diehards who defend the rigors of internship and residency as a required *rite de passage* because they went through it themselves long ago are reading from a dated script. The internships and residencies they knew no longer exist.

There have always been concerns about the effects of inadequate supervision of house staff and physician fatigue on the quality of patient care. Common sense alone tells us that a fatigued individual's judgment, reflexes, and attention span are impaired to varying degrees. The same common sense tells us that interns' and residents' enthusiasm for being in charge is no substitute for supervised performance.

The severity and extent of the problems just outlined are difficult to quantify, and this in turn has led many to trivialize them. The problem of house staff fatigue, so long winked at, and the issue of house staff supervision, long viewed as a matter of perspective, were clearly seen as major factors in the death of a young woman, Libby Zion, in a New York hospital in 1984, by a grand jury which reported on the case in January 1987.[2] That grand jury made five reasonable recommendations based on the particulars of the case they were asked to examine. These recommendations dealt with required levels of training for physicians in emergency rooms, the contemporaneous supervision of house staff, limiting consecutive working hours for interns and residents, the issue of restraining patients, and the need for a computerized system to check for contraindicated drugs.[3] Three of the five recommendations were a charge to the New York State Department of Health to implement regulations governing required levels of training of emergency room physicians, the supervision of interns and junior residents, and the limitation of consecutive working hours.[3]

Dr David Axelrod, the New York State Commissioner of Health, appointed an ad hoc advisory committee of nine members, chaired by Dr Bertrand Bell, Director of the Ambulatory Care Service, Bronx Municipal Hospital Center, to consider the recommendations of the grand jury.[3,4] On June 2, 1987, the ad hoc committee sent its preliminary report to Dr Axelrod, making the following primary recommendations:

> There shall be at least one emergency service attending physician on duty 24 hours a day, seven days a week, in all hospitals which have more than 15,000 unscheduled visits annually. An attending physician shall be present and available in person to provide patient care in the emergency service.
>
> Emergency service attending physicians shall be licensed and board certified in Emergency Medicine, Surgery, Internal Medicine or Family Practice.
>
> House officers and attending physicians with direct patient care responsibilities shall not work for more than 12 consecutive hours per shift in the emergency service, separated by no less than eight hours off. House officers and attendings with direct patient care responsibilities in other areas should in general not work more than 16 hours per shift. Individuals who have worked the maximum in one facility should not work a consecutive shift in another hospital.[4]

The ad hoc committee also extensively considered the issue of in-person and timely supervision of residents and interns by attending or supervisory physicians. It endorsed this recommendation of the grand jury in principle but expressed the view that it would require further study and in-depth analysis.[3]

In June 1987 Dr Axelrod stated that he would embody the proposals in regulations to be presented to the State Hospital Review and Planning Council over the next several months. Meanwhile the ad hoc committee has continued to meet and to advise him.

Dr Axelrod has taken into account the reality that some of the proposed regulations may raise hospital operating costs.[2] Predicting the actual level of increased costs will be difficult because of the multiplicity of variables that must be considered. These costs will have to be met in part by higher Medicare and Medicaid reimbursement rates as well as higher rates from large private insurers such as Empire Blue Cross and Blue Shield.

It is reasonable to anticipate, however, that additional ways will also be found to deal with the cost concerns. There are a number of avenues open, including the assumption of more responsibility for the direct medical care of patients by attending and staff physicians. There is no reason why this cannot be done without compromising the quality of teaching programs.

Opponents can be expected to beat the drums of cost concerns, and many of the groups responsible for regulating graduate medical education programs will continue to characterize the problems as minor and the risks to patients as few. These positions will prove to be weak currency among the general public, whose common sense tells them otherwise.

It was the late Libby Zion's father, Sidney Zion, a well-known journalist, who first focused public attention on these problems. Transforming his personal grief over his daughter's tragic death into a crusade for reform, he started a process which continues to move forward and which will permanently affect graduate medical education for the better. That process which began with one man's tragedy has now been transformed into a commendable initiative of reform by Dr David Axelrod, New York State's Commissioner of Health. The reforms he has put forward are in the best interests of both doctors in training and the patients they serve. As such they deserve widespread support from hospitals, physicians, and the public at large.

PASCAL JAMES IMPERATO, MD
Editor

Professor and Chairman
Department of Preventive Medicine
and Community Health
SUNY Health Science Center at Brooklyn
Brooklyn, NY 11203

1. Imperato PJ: Doctors fashioned by the "me" generation [letter]. *New York*

Times, November 28, 1982, p14E.
2. Sullivan R: New York plans 12-hour emergency room shifts. *New York Times*, June 3, 1987, pp A1, B12.
3. Letter from Bertrand Bell, MD, Chairman, Ad Hoc Committee on Emergency Services, to Dr David Axelrod, Commissioner of Health, State of New York, New York, June 2, 1987.
4. Press release, State of New York Department of Health, Albany, NY, June 2, 1987.

Humanism and medical ethics in a technological age[*]

The present period in America's history brings to mind the familiar words of Dickens,[1] "It was the best of times, it was the worst of times." Today, as we witness the continued improvement in the quality of human life, we also sadly observe a regrettable erosion of the traditional and respected image of the physician and the medical profession. The very human and caring doctor now seems to be perceived as a highly trained technologist and an indifferent purveyor of cold, scientific facts. Ethical problems which heretofore did not exist are also being encountered, bringing with them further unanticipated criticisms. How shall we explain these paradoxes, when medicine is accomplishing more now than ever before? Perhaps with a better understanding of the profession's history and responsibilities we will more easily resolve the burgeoning problems that plague us today.

Medical care first came into existence when primitive man began using magic to alleviate the pain and suffering of the ill and injured. The sorcerer, the intermediary with the supernatural world, who provided this care was early man's first physician. However, as man continued to develop and his knowledge and skills increased, medical care was undertaken by others: priests, philosophers, and finally, the men of the embryonic science of medicine.[2]

Early medicine probably achieved its greatest development in Greece, during what is known to us as the Golden Age, a period in which ideas could develop freely. From a priestly healing cult there slowly evolved a system of medical care which achieved its greatest prominence on the island of Cos, where Hippocrates treated the ill, wrote, and taught.[3] Controversial as the Hippocratic Corpus, or Oath, may be, it presents us with an awareness of a changing moral responsibility for the physician, and although it is no longer quite as meaningful, it has continued to guide physicians to the present day.

Medicine and philosophy in that age were interrelated, and within this intellectual framework a much more scientific approach to medicine was created. The Greek physician was guided by and encouraged to adhere to two basic principles: the love of the art of healing, philotechnia; and the love of man, philanthropia.[4] Yet, despite this love for their fellow men, the medical care given by early physicians varied considerably. The slave did not receive the care of the free man, nor did the poor receive the attention given to the rich. The true meaning of medical philia was still to be realized.

Probably what should be recognized is that much more is demanded of the physician in this present period. The criticisms frequently directed at contemporary medicine, however, may also be recognized as an echo from the past. For the Romans as for the Greeks philia was also held in the highest esteem, and was an attribute sought for in the physician. These feelings were succinctly expressed by Seneca[5] as he wrote in his *de Beneficiis*, "If, therefore, a physician does nothing more than feel my pulse . . . instructing me what to do and what to avoid without any feeling, I owe him nothing more than his fee, because he does not see me as his friend, but as a client . . . ." These problems of another age, two thousand years ago, regrettably are still troubling us today.

The physician, in caring for the ill, should possess not only medical competency, but also the ability to provide care with genuine compassion. The absence of the latter justifies the criticism that physicians are becoming mere technicians. The acquisition of compassion may be more difficult than is generally recognized. Compassion must be understood as an affective characteristic not to be confused with a cognitive appreciation of the humanities. The latter can be learned in the classroom, but not so the former. It is in this context that one can appreciate the greatness and the humanism of Sir William Osler,[6] who recognized this and chose for his teaching the most meaningful setting available—the bedsides of his patients. We also learn the depth of Osler's[7] dedication to medicine from this address which he gave to his students at St Mary's Hospital in London: "You are in this profession as a calling, not as a business, as a calling which exacts from you at every turn self-sacrifice, devotion, love and tenderness to your fellow men." These moving words, exemplifying the true meaning of medical philia, must never be forgotten, and should remain for us a guiding light in our professional lives.

As technology has advanced, it has also become increasingly evident that the medical ethics of the past are no longer sufficient. With today's expanded armamentarium, the physician is capable of undertaking procedures that can control the creation or termination of human life. Who will now make these decisions, and on what basis, and whose values will be considered, are the questions being raised. Today the patient demands the right to participate in the making of decisions, in contrast to the past, when this was done by the physician alone. We are beginning to deal with the patient's personal values, and are becoming aware that these values may be as vital and important for the patient's life as the physical considerations. The patient's values must be respected, but they can only be accepted if they do not create injustices for others. The principle of autonomy, we learn, cannot be absolute.

---

[*] Presented at the Annual CME Assembly of the Medical Society of the State of New York, March 13, 1987.

The importance of an orientation to value questions continues to increase as we observe the advances of modern biomedical science. Examples such as genetic engineering, organ transplantation, and surrogate motherhood are only the introductions to further developments which lie before us, and which will demand an even greater understanding. Physicians will be faced with these new ethical problems, and it will be their responsibility to help resolve them, for they involve the physical and emotional health of their patients. Physicians who are interested will have to undertake further training in philosophy, and specifically ethics, which concerns itself with the rightness or wrongness of human conduct. Meanwhile, one can be very grateful to the philosophers and theologians who have so generously given of themselves and their time to assist physicians, as well as to teach them.

As physicians continue to meet these new and difficult challenges, they are also engaged in creating a very different world. One may recall, and now more fully understand, what Dobzhansky[8] perceived when he wrote, "By changing what he knows about the world, man changes the world he knows; and by changing the world in which he lives, man changes himself." Yet in terms of the physician's relationship to his patient, this would not be valid. Concern, caring, and dedication are existent feelings for the physician and remain unchanged although the larger world changes.

Health and illness, life and death are now being viewed in a more societal and less individualistic framework. Although physicians often give lip service to the concept of the sacredness of human life, the emphasis is now focused more on the quality of life. These changes challenge earlier metaphysical suppositions in relation to what we value, what we think, and why we exist. Physicians also face questions as to how long life should be maintained in terminally ill patients, fears about continuing in vitro fertilization, and whether they have the wisdom to deal with further technological advances. Society has the right to ask all these questions and to scrutinize what physicians are doing, but physicians have not been indifferent to these problems and have been raising these as well as other questions.

We have already entered a growing world of technology. Our research must continue, however, and we should not retreat from furthering medical knowledge. If physicians remember that their goal is the enrichment of human life, they will have no difficulty meeting the challenges of this technological age. My confidence and hope for our profession are similar to Carl Sandburg's[9] hope for man, expressed when he wrote,

> I am credulous about the
> destiny of man,
> And I believe more than I
> can ever prove
> Of the future of the
> human race.

STEPHEN NORDLICHT, MD
Consulting Psychiatrist
Memorial Sloan-Kettering
Cancer Center

St Luke's Roosevelt Hospital
Center
New York, NY 10021

1. Dickens C: *A Tale of Two Cities.* New York, Collier and Son, 1922.
2. Sigerist HE: *A History of Medicine.* New York, Oxford University Press, 1951.
3. Castiglioni A: *A History of Medicine.* New York, Alfred A. Knopf, 1958.
4. Lain-Entralgo P: *Doctor and Patient.* New York, McGraw-Hill Book Co, 1969.
5. Seneca LA: *De Beneficiis.* Cambridge, Harvard University Press, 1967.
6. Cushing H: *The Life of Sir William Osler.* New York, Oxford University Press, 1967.
7. Osler W: The reserves of life. *St Mary's Hospital Gazette* 1907; 13:95–98.
8. Dobzhansky T: *Mankind Evolving: The Evolution of the Human Species.* New Haven, Conn, Yale University Press, 1962.
9. Sandburg C: *The People, Yes.* New York, Harcourt Brace Jovanovich Inc, 1936.

Group B streptococcal infection in 1987

The case of group B streptococcal osteomyelitis reported by McMeeking et al[1] in this issue of the *Journal* illustrates the broad spectrum of disease caused by this important pathogen. *Streptococcus agalactiae* causes serious infection in men and women of all age groups. It was originally discovered as a pathogen in women of childbearing age, causing puerperal fever.

Puerperal fever was described as early as 1800 during epidemics in the United States, England, Canada, Europe, and Australia.[2] Epidemiologic studies of these epidemics provide some of the earliest data regarding nosocomial infection. It was in 1843 that Ignace Semmelweiss made the notable observation that the doctors and medical students transported "cadaveric particles" from the autopsy table to women in labor via their unwashed hands; the women became infected and subsequently died.[2] In the same year, Oliver Wendell Holmes also published an opinion that puerperal fever was inserted into the vaginal vault by the attending physician during labor and delivery.[3] That statement was inflammatory to many contemporary physicians and generated heated debate in the American medical literature for nearly 40 years.[2,4] In spite of the adoption of aseptic techniques during child delivery, epidemics of puerperal fever continued, albeit with less frequency. One such epidemic occurred at the Sloane Hospital for Women in New York City in 1927 and was described in 1928.[4] The authors demonstrated that the sources of hemolytic streptococci were the oral and nasopharyngeal cavities of the delivery room personnel, including both doctors and nurses.[5] Thus, this epidemic of puerperal fever was also nosocomially caused, though the source of group B streptococcus was different.[5]

More recently, group B streptococcus has been recognized as an important cause of neonatal sepsis, meningitis, and pneumonia,[6] and surveys among women of childbearing age indicate that the major human reservoirs are the genitourinary and gastrointestinal tracts.[7] Thus, colonization of these tracts during the third trimester of pregnancy constitutes a risk factor to the unborn child, particularly if the neonate is of low birth weight or if there is premature rupture of the membranes.[8] Thus, the source of bacteria may be the birth canal of the mother and not hospital personnel.[8]

In contrast to the epidemic occurrence of puerperal sepsis in the 1800–1930 era, group B streptococcal infection today occurs as a sporadic infection among neonates, compromised hosts, diabetic patients, and hospitalized patients.[9] For example, Verghese et al[9] described 23 sporadic episodes of bacteremia occurring at the Veterans Administration Medical Center in Boston over a seven-year period and demonstrated that 70% of these infections were acquired in the hospital. The most common sources of bacteremia were the respiratory tract, urinary tract, and intravenous access sites.[9] The patient with mild diabetes mellitus and a urinary source of infection described by McMeeking et al[1] is similar to those described by Verghese et al.[9]

Recognition of group B streptococcal infection has only been possible through improved means of identifying this bacterium and, more importantly, of distinguishing it from other hemolytic streptococci such as *Streptococcus pyogenes* (group A). The streptococcal classification system currently in use was developed through the diligent work of Rebecca Lancefield at the Rockefeller Institute over a four-decade period beginning in the 1930s.[10] In addition, the CAMP test used by clinical laboratories as a presumptive test for group B streptococcus has also been of great importance for screening and epidemiologic purposes.[11] As illustrated in the case report in this issue,[1] however, the use of latex agglutination techniques, such as the Phadebact Streptococcus Test Kit (Pharmacia Diagnostics, Piscataway, NJ), has allowed both large and small clinical microbiology laboratories to distinguish rapidly and accurately among clinically important streptococcal pathogens. Thus, we will likely encounter greater numbers of infections attributable to group B streptococcus.

Within the group B classification, according to Lancefield, there are five capsular serotypes designated I_a, I_b, I_c, II, and III. Antibody against the group B polysaccharide of Lancefield does not afford protection, whereas antibody against the capsular antigenic determinant does.[12] The capsular polysaccharide does not activate complement in the absence of specific antibody.[13] Thus, type-specific antibody is necessary for protection[12,14,15] and effects this by enhancing granulocyte phagocytosis of homologous serotypes of group B streptococcus.[12,13] It would have been of interest to determine if the patient described in this case report[1] had specific antibody against the isolated strain of group B streptococcus.

In general, host factors that predispose to infection in nonpregnant adults have not been studied. Clinical series and case reports implicate diabetes mellitus, peripheral vascular disease, chronic renal failure, and cirrhosis as predisposing conditions.[9,16,17] Studies of neonatal infection have been more extensive and demonstrate that specific antibody against the capsular polysaccharide affords type-specific protection from that group B streptococcus.[14] Thus, women who are colonized with a particular serotype of group B streptococcus during the third trimester are protected from postpartum sepsis if they develop antibody against that particular serotype.[6–8,14,15] Similarly, neonates who acquire maternal IgG against that serotype of group B streptococcus will be protected.[14,15] Conversely, neonates devoid of type-specific maternal IgG who are delivered through a birth canal colonized with group B streptococci are at risk of developing pneumonia, sepsis, or meningitis.[14,15] It is therefore of more than academic interest to detect group B streptococcal colonization of the vagina among women during the third trimester of pregnancy in order to identify high-risk patients.[14,15]

At the outset, it would appear that there are few if any problems in the treatment of serious group B streptococcal infection. Clearly, the vast majority of clinical group B streptococcus isolates are sensitive to penicillin, ampicillin, cefamandole, cefuroxime, cephalothin, erythromycin, chloramphenicol, clindamycin, and vancomycin.[18] Yet, clinical failures have been described in neonates with group B streptococcal meningitis who were treated with penicillin, prompting some physicians to recommend antibiotic treatment with penicillin and gentamicin combined.[19,20] To support this view, Baker et al[18] demonstrated that penicillin and gentamicin were synergistic against group B streptococcus in vitro, but only if penicillin was given first.

A major thrust of research pertaining to group B streptococcus infection has been to establish a means of preventing neonatal infection. The first step has been to identify high-risk situations—ie, vaginal colonization of the mother during the third trimester of pregnancy or, more specifically, the 34th–36th week of gestation. This approach has been possible only since the advent of rapid techniques of identifying the organism. Treatment of colonized mothers with intravenous ampicillin at the time of delivery has been shown to be efficacious in preventing neonatal group B streptococcal colonization.[21,22] Another approach has been to administer penicillin to all newborns immediately after parturition.[23] The latter regimen has reduced the number of early onset group B streptococcus infections at Mt Sinai Hospital in New York City.[23] However, the risks of adverse effects of penicillin given routinely in all live births must be weighed against the incidence of infection (2–3/1,000 live births). Another strategy recently utilized chlorhexadine vaginal washes prior to delivery for mothers colonized with group B streptococcus.[24]

Immunologic means of preventing or treating actual infection in humans have been a focus of considerable study, largely as a result of the observation of Shigeoka et al,[25] who reported 100% survival of infants with group B streptococcal infection who received transfusions of whole blood containing type-specific antibody to the infecting group B streptococci. In contrast, only 50% survival occurred among infected infants who received transfusions of blood that did not contain type-specific antibody.[25] Although high titer type-specific antibody has been demonstrated in some human gamma globulin preparations,[26] the efficacy of passive immunization by administration of these products to either the mother or the neonate has not been verified. Ideally, prevention of group B streptococcal infection will involve detection of antibody-negative women of childbearing age, followed by active or passive immunization before or during pregnancy in order to provide transplacental protection (IgG) to the newborn infant. The next decade will undoubtedly bring major advances in the understanding, prevention, and treatment of group B streptococcal infection. Monoclonal antibodies will play a major role in the development of highly specific and sensitive diagnostic reagents and may also provide an additional therapeutic modality.

DENNIS L. STEVENS, MD, PHD
Associate Professor of Medicine
University of Washington
School of Medicine

Chief, Infectious Diseases
Veterans Administration Medical Center
Boise, ID 83702

1. McMeeking AA, Holzman R, Desiderio D, et al: Group B streptococcal osteomyelitis in an adult. *NY State J Med* 1987; 87:466-467.
2. Lansing DI, Penman WR, Davis DJ: Puerperal fever and the group B-beta hemolytic streptococcus. *Bull Hist Med* 1983; 57:70-80.
3. Holmes OW: On the contagiousness of puerperal fever, in *Medical Essays.* Boston, Houghton-Mifflin Publishers, 1891, pp 103-172.
4. Watson BP: Outbreak of puerperal sepsis of New York City. *Am J Obstet Gyn* 1928; 16:157-179.
5. Meleney FL, Zau ZD, Zaytzeff H, et al: Epidemiologic and bacteriologic investigation of Sloane Hospital epidemic of hemolytic streptococcus puerperal fever in 1927. *Am J Obstet Gyn* 1928; 16:180-194.
6. Baker CJ: Summary of the workshop on perinatal infections due to group B streptococcus. *J Infect Dis* 1977; 136:137-152.
7. Anthony BF, Eisenstadt R, Carter J, et al: Genital and intestinal carriage of group B streptococci during pregnancy. *J Infect Dis* 1981; 143:761-766.
8. Anthony BF, Okada DM, Hobel CJ: Epidemiology of group B streptococci. *Am J Obstet Gynecol* 1976; 124:630-634.
9. Verghese A, Mireault K, Arbeit RD: Group B streptococcal bacteremia in men. *Rev Infect Dis* 1986; 8:912-917.
10. Lancefield RC, Freimer EH: Type-specific polysaccharide antigens of group B streptococci: *J Hyg* (*Camb*) 1966; 64:191-203.
11. Christie R, Atkins NE, Munch-Petersen E: Note on lytic phenomenon shown by group B streptococci. *Aust J Exp Biol Med Sci* 1944; 22:197-200.
12. Hemming VG, Hall RT, Rhodes PG, et al: Assessment of group B streptococcal opsonins in human and rabbit serum by neutrophil chemiluminescence. *J Clin Invest* 1976; 58:1379-1387.
13. Edwards MS, Nicholson-Weller A, Baker CJ, et al: The role of specific antibody in alternative complement pathway-mediated opsonophagocytosis of type III, group B streptococcus. *J Exp Med* 1980; 151:1275-1287.
14. Baker CJ, Kasper DL: Correlation of a maternal antibody deficiency with susceptibility to neonatal group B streptococcal infection. *N Engl J Med* 1976; 294:753-756.
15. Cleat P, Ross J, Needham J: Antibody levels in mothers colonized with group B streptococci during pregnancy and in their newborn infants, as measured by an enzyme linked immunosorbent assay. *Med Microbiol Immunol* 1980; 168:49-53.
16. Stevens DL, Haburchak DR, McNitt TR, et al: Group B streptococcal osteomyelitis in adults. *South Med J* 1978; 71:1450-1451.
17. Bayer AS, Chow AW, Anthony BF, et al: Serious infections in adults due to group B streptococci. Clinical and serotypic characterization. *Am J Med* 1976; 61:498-503.
18. Baker CN, Thornsberry C, Facklam RR: Synergism, killing kinetics, and antimicrobial susceptibility of group A and B streptococci. *Antimicrob Agents Chemother* 1981; 19:716-725.
19. Boughton DD, Mitchell WG, Grossman W, et al: Recurrence of group B streptococcal infection. *J Pediatr* 1976; 89:183-185.
20. Walker SH, Arturo AQ, Qunitero BA: Recurrence of group B III streptococcal meningitis. *J Pediatr* 1976 89:187-188.
21. Yow MD, Mason EO, Leeds LJ, et al: Ampicillin prevents intrapartum transmission of group B streptococcus. *JAMA* 1979; 241:1245-1247.
22. Morales WJ, Lim DV, Walsh AF: Prevention of neonatal group B streptococcus sepsis by the use of a rapid screening test and selective intrapartum chemoprophylaxis. *Am J Obstet Gynecol* 1986; 155:979-983.
23. Hodes HL: Penicillin prophylaxis and neonatal streptococcal disease. *Hosp Pract* 1980; 15:115-125.
24. Christensen KK, Dykes AK, Christensen P: Reduced colonization of newborns with group B streptococci following washing of the birth canal with chlorhexidine. *J Perinat Med* 1985; 13:239-243.
25. Shigeoka AO, Hall RT, Hill HR: Blood transfusions in group B streptococcal sepsis. *Lancet* 1978; 1:636-638.
26. Fischer GW, Weisman LB, Hemming VG, et al: Intravenous immunoglobulin in neonatal group B streptococcal disease. Pharmacokinetic and safety studies in monkeys and humans. *Am J Med* 1984; 76:117-123.

Low serum triiodothyronine in diabetic ketoacidosis

JAMES TURRO, MD, MILFORD FULOP, MD

ABSTRACT. Thirteen patients with diabetic ketoacidosis were identified on admission, and serum triiodothyronine and thyroxine concentrations were measured. These measurements were repeated in 11 of the 13 patients after metabolic control was achieved, 3–21 days later. The initial triiodothyronine values were subnormal (< 90 ng/dL) in 11 patients, with a mean of 77.5 ± 22.4 (standard deviation) ng/dL for all 13 patients. Follow-up triiodothyronine values increased significantly to a mean of 110.0 ± 22.1 ng/dL ($p < 0.005$). The initial thyroxine values were normal in ten of the 13 patients, but these values increased on average by 1.3 ± 2.3 μg/dL. Serum triiodothyronine values are initially decreased in patients with diabetic ketoacidosis, suggesting that thyroid hormone measurements should be delayed in these patients until metabolic control is achieved.

(NY State J Med 1987; 87:431–433)

Thyroid function tests are frequently performed in patients with acutely uncontrolled diabetes mellitus. Marble et al[1] state that "the presence of hyperthyroidism should be suspected in diabetic patients with or without thyroid enlargement in the presence of unexplained increase in insulin requirement, ketoacidosis or instability of the diabetes." Documenting an abnormality in thyroid hormone levels in diabetic patients with ketoacidosis is not straightforward. In the last decade, several studies have described low serum triiodothyronine levels in patients with severely uncontrolled diabetes,[2-5] including some patients with ketosis or ketoacidosis.[6,7] Most of these studies have involved children or adolescents. Although the occurrence of a low serum triiodothyronine level in diabetic ketoacidosis (DKA) is known to endocrinologists, this abnormality is often not appreciated by many other physicians. To further assess this phenomenon in adults, we measured serum triiodothyronine (T_3) and thyroxine (T_4) levels in 13 patients admitted with a diagnosis of diabetic ketoacidosis. The level of serum triiodothyronine was usually low during the acute episode.

PATIENTS AND METHODS

Patients with diabetic ketoacidosis were identified on admission to Bronx Municipal Hospital Center between July 1985 and December 1985. All the patients had blood pH < 7.36, a positive serum and/or urine nitroprusside test for ketones, and an elevated anion gap. No patient was taking any medication known to affect serum triiodothyronine or thyroxine measurements, and all were clinically euthyroid. All the patients were insulin-dependent diabetics, and in two patients, this episode of ketoacidosis was the symptomatic onset of the disease. Blood specimens were obtained on admission, either prior to any treatment or shortly after the initiation of therapy. Follow-up measurements were obtained after metabolic control was achieved, after 3–21 days (average, 8.5 ± 5.7 days), usually just before discharge.

Serum triiodothyronine levels were measured using a double antibody radioimmunoassay; the normal range is 90–180 ng/dL (Diagnostic Products Corporation, Los Angeles, Calif). Serum thyroxine levels were measured by radioimmunoassay; the normal range is 5–12 μg/dL (Corning Medical, Medfield, Mass). The normal range for T_3 sponge uptake values (Corning Medical, Medfield, Mass) is 25–35%, and for the derived free T_4 index is 4.5–14. Mean values are reported ± 1 standard deviation, and the changes in serum T_3 and T_4 values were compared using a two-tailed t test for paired values. One initial serum T_3 value reported as < 50 ng/dL was counted as 50 ng/dL to calculate the mean. This value was obtained in a patient who did not have a follow-up measurement.

RESULTS

All patients were clinically euthyroid, and all but two of those retested had normal follow-up levels of serum triiodothyronine and thyroxine. Seven patients were men, six were women, and their average age was 31.7 ± 16.3 years (range, 17–64 years). The mean blood pH was 7.12 ± 0.17, and the serum glucose level was 754 ± 406 mg/dL.

The mean initial serum T_3 level was 77.5 ± 22.4 ng/dL. The follow-up values were normal in all but one patient and averaged 110.0 ± 22.1 ng/dL. Figure 1 shows the individual initial and follow-up serum T_3 values. The mean difference of 29.5 ± 22.7 ng/dL in the 11 patients with follow-up samples was highly significant ($p < 0.005$). Initial T_4 values were normal in ten of the 13 patients, averaging 7.5 ± 2.4 μg/dL. Two of the other patients had slightly low values initially, both 4.8 μg/dL, and one had a slightly elevated T_4 value (13.1 μg/dL). Follow-up T_4 values rose in seven of the ten patients with paired specimens, with a follow-up mean value of 8.4 ± 2.6 μg/dL. These values are also shown in Figure 1. The differences between the initial and follow-up thyroxine values averaged 1.3 ± 2.3 μg/dL ($p = 0.1$). The patient with a low follow-up T_4 value subsequently had a thyroid-stimulating hormone (TSH) determination of 2.8 μU/mL (normal). The patient with a slightly elevated initial T_4 value (13.1 μg/dL) had a normal free T_4 index (10.3). The patient with a slightly elevated follow-up T_4 value (13.6 μg/dL) had a normal follow-up measurement (8.9 μg/dL) taken later.

The T_3 sponge uptake was measured initially and later in ten patients, and was, on average, normal. The initial mean was 30.0% ±

From the Department of Medicine, Albert Einstein College of Medicine, and the Bronx Municipal Hospital Center, Bronx, NY.

Address correspondence to Dr Fulop, Department of Medicine, Jacobi Hospital, Pelham Parkway South & Eastchester Rd, Bronx, NY 10461.

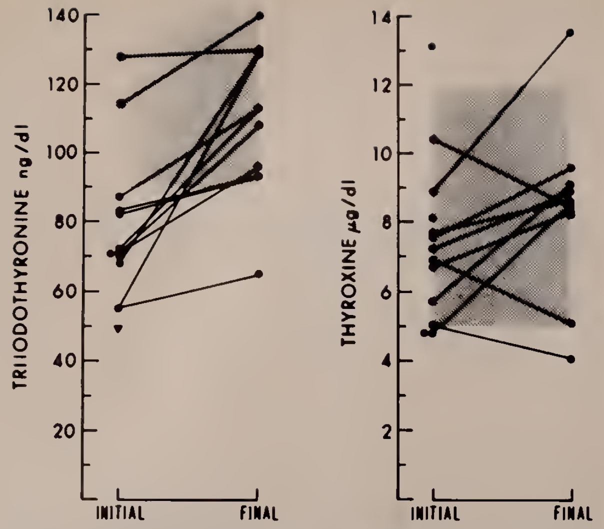


FIGURE 1. Initial and follow-up serum triiodothyronine and thyroxine concentrations in diabetic ketoacidosis. **Left,** initial T_3 values in 13 patients and follow-up values in 11 (triangle, initial value < 50 ng/dL). **Right,** initial T_4 values in the 13 patients and follow-up values in ten. The shaded areas indicate the normal range for T_4 (5–12 µg/dL) and the lower part of the normal range for T_3 (90–180 ng/dL).

3.3, the follow-up 29.3% ± 2.9, and the mean difference was −0.7% ± 3.4 (not significant). The initial and follow-up mean free T_4 indexes were also normal in those ten patients. The initial mean was 7.0 ± 1.6, and the follow-up mean was 8.2 ± 2.5. The difference between the initial and follow-up mean free thyroxine index values averaged 1.1 ± 2.0 ($p = 0.1$).

DISCUSSION

This survey was prompted by our finding a low-normal serum triiodothyronine level (96 ng/dL) in a patient with diabetic ketoacidosis and iatrogenic hyperthyroidism. This patient had been taking diet pills, including 200 µg of levothyroxine, intermittently for 12 years. Repeat thyroid function tests performed two weeks after admission showed a serum T_3 level of 266 ng/dL, a T_4 of 21.7 µg/dL, and free T_4 index of 20.6. Similar low serum T_3 values in patients with DKA and hyperthyroidism have been reported by Naeije et al,[8] Mayfield et al,[9] and Ahmad and Cohen.[10]

We studied 13 euthyroid patients with DKA and found that 11 had subnormal serum T_3 values on admission. Euthyroid patients with various illnesses may have abnormal serum levels of thyroid hormones, including triiodothyronine.[11] Also, starvation [11] and, specifically, carbohydrate deprivation[12] can result in decreased serum T_3 concentrations. In the past decade, severely uncontrolled diabetes,[2-5] including episodes associated with ketosis and ketoacidosis,[6,7] has been recognized as another cause of low serum triiodothyronine values. Serum T_3 decreases rapidly after loss of diabetic control. Madsbad et al[13] withdrew insulin from 15 young type I diabetics and found a gradual decrease in serum T_3 during the ensuing 12 hours and an increase of "reverse triiodothyronine" within four to six hours. Balsam and Ingbar[14] found that liver slices from diabetic rats converted less T_4 to T_3 than did slices from normal rats, presumably because of decreased thyroxine′-5′-monodeiodinase activity in liver homogenates that increased after several days of insulin treatment. The hepatic 5′-monodeiodinase is responsible for deiodinating T_4 to T_3, and perhaps also for deiodinating reverse T_3. Most workers in this field believe that the low serum T_3 and elevated reverse T_3 levels found in uncontrolled diabetes result mainly from preferential deiodination of T_4 to reverse T_3,[14,15] and, perhaps, from deceased deiodination of reverse T_3. Recently, however, in studies of perfused rat livers, Jennings et al[16] found that the decreased hepatic production of T_3 during starvation seemed to result mainly from decreased hepatic uptake of T_4, rather than from decreased 5′-monodeiodinase activity. Whether this may also be a factor in uncontrolled diabetes, with its attendant impairment of glucose uptake and effective "cellular starvation," remains to be seen. There is believed to be an increased incidence of idiopathic hypothyroidism in diabetes,[17] but that was not at issue here, with only one of the 13 patients having a persistently low serum T_3 level (on early follow-up).

In about 25% of cases of DKA no easily identifiable precipitating cause can be found.[18] Thyroid function tests are often performed in these patients. Our findings indicate that serum triiodothyronine values obtained in patients with DKA should not be relied on. These values are often subnormal and increase significantly after a week or so of improved glycemic control. The same is apparently true of patients with severely uncontrolled diabetes without ketoacidosis. The search for possible hyperthyroidism as a precipitant for an episode of DKA can be safely delayed until glycemic control is achieved. Of course, essential treatment should be started immediately for patients with diabetic ketoacidosis and florid hyperthyroidism, without waiting for the results of thyroid function tests.

REFERENCES

1. Marble A, Krall L, Bradley RF, et al: *Joslin's Diabetes Mellitus.* Philadelphia, Lea and Febiger, 1985, p 794.
2. Pittman CS, Suda AK, Chambers JB Jr, et al: Abnormalities of thyroid hormone turnover in patients with diabetes mellitus before and after insulin therapy. *J Clin Endocrinol Metab* 1979; 48:854–860.
3. Gilani BB, MacGillivray MH, Voorhess ML, et al: Thyroid hormone abnormalities at diagnosis of insulin-dependent diabetes mellitus in children. *J Pediatr* 1984; 105:218–222.
4. MacFarlane IA, Sheppard MC, Black EG, et al: The hypothalamic-pituitary-thyroid axis in type I diabetes: Influence of diabetic metabolic control. *Acta Endocrinol* 1984, 106:92–96.
5. Schlienger JL, Anceau A, Chabrier G, et al: Effect of diabetic control on the level of circulating thyroid hormones. *Diabetologia* 1982; 22:486–488.
6. Alexander CM, Kaptein EM, Lum SMC, et al: Pattern of recovery of thyroid hormone indices associated with treatment of diabetes mellitus. *J Clin Endocrinol Metab* 1982; 54:362–366.
7. Postellon DC, Becker DJ, Foley TP: Alterations in triiodothyronine (T_3) and reverse triiodothyronine (rT_3) concentrations in newly diagnosed patients with juvenile diabetes mellitus. *Diabetes* 1978; 27(suppl 2):498.
8. Naeije R, Golstein J, Clumeck N, et al: A low T_3 syndrome in diabetic ketoacidosis. *Clin Endocrinol* 1978; 8:467–472.
9. Mayfield RK, Sagel J, Colwell JA: Thyrotoxicosis without elevated serum triiodothyronine levels during diabetic ketoacidosis. *Arch Intern Med* 1980; 140:408–410.
10. Ahmad N, Cohen MP: Thyroid storm with normal serum triiodothyronine level during diabetic ketoacidosis. *JAMA* 1981; 245:2516–2517.
11. Schimmel M, Utiger RD: Thyroidal and peripheral production of thyroid hormones. Review of recent findings and their clinical implications. *Ann Intern Med* 1977; 87:760–768.
12. Danforth E Jr, Horton ES, O'Connell M, et al: Dietary-induced alterations in thyroid hormone metabolism during overnutrition. *J Clin Invest* 1979; 64:1336–1347.
13. Madsbad S, Laurberg P, Weeke J, et al: Very early changes in circulating T_3 and rT_3 during development of metabolic derangement in diabetic patients. *Acta Med Scand* 1981; 209:385–387.
14. Balsam A, Ingbar SH: The influence of fasting, diabetes, and several pharmacological agents on the pathways of thyroxine metabolism in rat liver. *J Clin*

Invest 1978; 62:415-424.
15. Gavin LA, McMahon FA, Moeller M: The mechanism of impaired T_3 production from T_4 in diabetes. *Diabetes* 1981; 30:694-699.
16. Jennings AS, Ferguson DC, Utiger RD: Regulation of the conversion of thyroxine to triiodothyronine in the perfused rat liver. *J Clin Invest* 1979; 64:1614-1623.
17. Feely J, Isles TE: Screening for thyroid dysfunction in diabetes. *Br Med J* 1979; 1:1678.
18. Davidson M: *Diabetes Mellitus: Diagnosis and Treatment*. New York, John Wiley and Sons, 1981, p 196.

Cigarette smoking among Ethiopian health professionals and students

ZEIN AHMED ZEIN, DR MED, MPH

ABSTRACT. The smoking habits of 418 medical and paramedical students and 260 health professionals in Northern Ethiopia were determined using questionnaires developed by the World Health Organization. The rate of smoking was 18.4% among students and 8.9% among health professionals, with a preponderance of male smokers in both groups. There were relatively more physicians and paramedical personnel who smoked than there were nurses who smoked. The rate of smoking among these health care workers is lower than that reported among Ethiopian students and teachers, and physicians and nurses in other African countries. However, the association between cigarette smoking, alcohol consumption, and the use of tranquilizers and Khat (*Catha edulis Forsk*) is alarming. The establishment of a national antismoking campaign is recommended to address the problems of drug abuse in general, and cigarette smoking in particular.

(NY State J Med 1987; 87:433-435)

During the past 30 years, smoking has become a major cause of death in many developed countries. In Africa, by contrast, the rapid rise in cigarette consumption and diseases related to smoking has only recently attracted attention.[1-3] Information on the prevalence of smoking in Africa is inadequate and sporadic, and in general is confined to students[4-7] and the general population.[7-9] This paucity of information is also applicable to Ethiopia, where, in spite of the doubling of cigarette consumption between 1965 and 1976,[1] only three surveys by this investigator remain as a source of epidemiologic data.[10-12] This study is a continuation of these surveys and addresses the prevalence of smoking among health professionals.

SUBJECTS AND METHODS

The survey was conducted in the Gondar Region, in northwestern Ethiopia, which has a population of 2.7 million, 7.0% of whom live in urban areas. Health facilities in the region include a teaching hospital and two rural hospitals with a total bed capacity of 438, as well as 13 health centers and 116 health stations. At the time of the study (August 1985), there were 102 physicians, 129 nurses, and 1,213 paramedical personnel. On the basis of a 20% rate of daily smoking among 70 health personnel in a pilot survey at the Gondar College of Medical Sciences (GCMS), a sample size of 260 health personnel was determined to be optimal for this study.

A closed-ended, self-administered questionnaire, prepared in the local vernacular, was anonymously administered in each health care facility, using medical students in their final year of study as supervisors. All questions were derived from the questionnaire for health professionals prepared by the World Health Organization.[13] Initially, the response rate was 79.4%—some health workers were absent on the day that the questionnaires were administered—but a follow-up survey on the second day reached most health workers. There was no difference in the "absent group" with regard to age or smoking behavior compared with responders ($p > 0.05$).

At the same time, all medical and paramedical students enrolled at GCMS were given the drug-use questionnaire developed by the World Health Organization,[14] using the same procedures used in the survey of health personnel. The definitions of daily smokers, occasional smokers, and nonsmokers were those proposed by the World Health Organization.[13]

RESULTS

The characteristics of the study population are shown in Table I. There were more men than women. The average ages of the students and health professionals were 21 and 30 years, respectively, suggesting a young cohort. The rates of smoking are presented in Table II. There were more smokers among the students (18.4%) than among health professionals (8.8%). More men were smokers in both groups. The percentage of occasional smokers was also greater among students (10.1%) than among health professionals, again with a preponderance of male (occasional) smokers in both groups.

The rates of smoking among physicians, nurses, and paramedical professionals were 25.5%, 3.0%, and 7.0% respectively. The attitudes of health professionals were surveyed with regard to perception of smoking as a health hazard. Of the 260 health professionals, 94.6% strongly agreed about the health hazards of cigarette smoking, while 0.4% strongly disagreed. Indeed, 77.7% were very much concerned about its harmful effects on health.

The smoking habit was found to be associated with other habits involving polydrug use (Table III). Smokers abused Khat (*Catha edulis Forsk*), an amphetamine-like plant material, tranquilizers (mainly diazepines, and chlordiazepoxide), and alcohol. The fre-

---

Address correspondence to Dr Zein, Associate Professor and Associate Dean, Gondar College of Medical Sciences, PO Box 109, Gondar, Ethiopia.

Based on a presentation at the EURO Conference on Smoking and Health Professionals, May 30-31, 1986, Venice, Italy.

TABLE I. Age and Sex Distribution of Study Population

	No. Surveyed	Percent	Mean Age (yr)	Age Range (yr)
Health professionals				
Men	164	63.0	—	—
Women	96	37.0	—	—
Total	260	100.0	30.1 ± 3.8*	18-56
Students				
Men	354	84.7	—	—
Women	64	15.3	—	—
Total	418	100.0	21.0 ± 1.2*	17-40

* Standard deviation.

quency of polydrug abuse was significantly greater among smokers than among nonsmokers (chi-square = 118.4; degrees of freedom = 1; $p < 0.001$).

DISCUSSION

This study of the smoking habits of Ethiopian health professionals indicates that the rate of cigarette smoking was greater than that observed in the general population (8.9%) for the same region (Zein, unpublished data, 1986), but less than that observed among school teachers (25.0–34.1%) in the same region.[11]

The highest rate of cigarette smoking was observed among physicians and paramedical personnel, with the lowest rate occurring among nurses. The present rate of cigarette smoking (18.4%) among students is lower than the rate observed in the same group in 1983 (24.8%).[12] The decline in student smoking was not explicable, but was certainly not due to any antismoking drive among student smokers.

The preponderance of male smokers was similar to the sex distribution of smokers in developing countries.[15] The ratio of male to female smokers among health professionals was 10:1; among students, however, it was only 1.8:1 and had narrowed in comparison with the 1983 level (6.6:1) in the same population.

The findings of this study are difficult to compare with those from developing countries in general and Africa in particular, as surveys among health professionals and students are few and have been conducted using different survey methodologies. Nevertheless, the rate of smoking in Ethiopia is lower than rates of 19.8%–40.3% reported among Ugandan and Nigerian students.[4,5]

Ethiopian health professionals, particularly physicians and nurses, smoke less than their Sudanese (37.0%) and South African (22.8%–37.6%) counterparts.[16,17] The present study site is situated in a comparatively less urban setting than previous studies. Similar surveys in the major urban centers of Ethiopia would probably reveal higher rates of smoking among health professionals.

The perception of cigarette-related health hazards by the participants in this study is encouraging, but the association of smoking with polydrug abuse among students is alarming and begs not only for an antismoking campaign, but also for comprehensive educational and legislative measures directed against a range of habits. The availability of inexpensive cigarettes, the easy access of health workers to mind-altering agents, the absence of smoking restrictions in hospitals, and, above all, the involvement of youth labor in the production of cigarettes are serious obstacles to an effective antismoking campaign. The need for a national antismoking and antidrug effort has become urgent in Ethiopia.

REFERENCES

1. Taha A, Ball K: Smoking and Africa. The coming epidemic. *Br Med J* 1980; 280:991–993.
2. World Health Organization: Report of the WHO Expert Committee on Smoking Control. *Controlling the Smoking Epidemic.* Technical Report Series 639. Geneva, World Health Organization, 1979.
3. Willard N: Tobacco: Third world warning. *World Health Organization Chron* 1983; 37:86.
4. Elegbeleye OO, Femi-Pearse D: Incidence and variables contributing to onset of cigarette smoking among secondary school children and medical students in Lagos, Nigeria. *Br J Prev Soc Med* 1976; 30:66-70.
5. Araya OP, Bennet FJ: Smoking among university students in Uganda, an analysis of prevalence and attitudes. *E Afr Med J* 1970: 47:18.
6. Kaur H: Drug taking among university students. *Niger Med J* 1978; 8:112-113.

TABLE II. Prevalence Rates of the Smoking Habit

	Smokers		Occasional Smokers		Nonsmokers	
	No.	%	No.	%	No.	%
Professionals						
Men (N = 164)	21	12.8	16	9.8	127	77.4
Women (N = 96)	2	2.1	3	3.1	91	94.8
M+W (N = 260)	23	8.8	19	7.3	218	83.9
Students						
Men (N = 354)	70	19.8	36	10.2	248	70.0
Women (N = 64)	7	10.9	6	9.4	51	79.7
M+W (N = 418)	77	18.4	42	10.1	299	71.5

TABLE III. Percent of Smokers* and Nonsmokers Reporting Multiple Drug Use

Drug Combinations†	Smokers‡ (N = 149)	Nonsmokers‡ (N = 269)
Cigarettes only	5.4	—
Khat only	16.1	0.0
Tranquilizer only	9.4	0.0
Alcohol only	41.6	0.0
Khat, tranquilizer	4.0	5.2
Khat, alcohol	13.4	15.2
Tranquilizer, alcohol	6.7	14.6
Khat, alcohol, tranquilizer	3.4	4.8
No drugs	—	60.2

* Includes occasional smokers.
† For smokers, drug combinations include cigarettes as well.
‡ $p < 0.001$.

7. Femi-Pearse D, Adeniyi-Jones A, Oke AB: Respiratory symptoms and their relationship to cigarette-smoking, dusty occupations and domestic air pollution: Studies in random sample of an urban African population. *West Afr Med J* 1973; 22:57-63.
8. Baker MD, Johnston JR, Turner DM: Smoking habits of blacks in industry. *S Afr Med J* 1978; 54:67-69.
9. Bewley BR, Johnson MR, Danks MH: Teachers' smoking. *Brit J Prev Soc Med* 1979; 33:219-222.
10. Zein A, Masresha A: The prevalence of cigarette smoking among secondary school children in Gondar City, Ethiopia. *Ethiop Med J* 1979; 17:41.
11. Zein AZ: Cigarette smoking among Ethiopian school teachers. *E Afr Med J* 1986; 63:244.
12. Zein AZ, Admasu M, Tadesse M, et al: Patterns of cigarette smoking among Ethiopian medical and paramedical students. *Ethiop Med J* 1984; 22:165-171.
13. World Health Organization: Guidelines for the conduct of tobacco-smoking surveys among health professionals. WHO/SMO, 1984.
14. Smart RG, et al: Methodology of student drug surveys. World Health Organization offset publication No. 50. WHO, Geneva.
15. World Health Organization: Smoking control strategies in developing countries. Tech Rep Ser No. 695. Geneva, WHO, 1983.
16. Ballal SG: Cigarette smoking and respiratory symptoms among Sudanese doctors. *East Afr Med J* 1984: 61:95-103.
17. Jacka E, et al: Smoking habit of nurses. *Curation* 1984; 7:37.

FROM THE LIBRARY

MEDICAL RESEARCH

. . . Research in its broadest terms is the search for truth. The truth in Science can be found only by trial or experimentation; but before the trial is made there must be a reason for its execution. This is to be found in an idea. The idea is the most valuable thing in Research. Ideas never come to a man who accepts everything he hears without mental reservation. They never come to the careless nor to those who never ask why. They never come to the man who is satisfied with Science as it is today. Ideas come only to the man who asks why and who tries to answer his own questions. They come to the man who thinks about the facts that he reads and observes. It is not the man who has knowledge alone who becomes the Research man, but rather the man who is able to utilize the knowledge which he has. Knowledge alone is not power. Thinking is power. Ideas cannot be forced or called forth at will. There must be some influence which stimulates thought. Human disease is a constant challenge to the Research man in Medicine. There is inherent in the Research man the desire to overcome obstacles, desire for achievement and the desire for the betterment of Science. Competition must not be underrated as a stimulus to thinking. Some of the world's greatest thoughts have been the outcome of criticism from an enemy.

My personal experience has been that ideas come in the quiet hours of night when one is free from outside disturbance. It is true that there are moments free from work during the busy day, but there is no possibility for continuity of thought when one does not know when an interruption may occur. Ideas come when one is in familiar surroundings, free from the distraction of the newness of things. One does not have ideas if one is physically or mentally tired. On the other hand, quietude, restfulness and peacefulness of the occasion will not produce thoughts without the stimulation for the desire of personal accomplishment. Even if the surroundings are conducive to thought there is no guarantee that quiet thinking will produce a constructive idea. I do not think it is possible to make a habit of thinking but very often one idea leads to another. New facts give further food for thought. . . .

FREDERICK G. BANTING, MD
(*NY State J Med* 1924; 24:311-315)

Arthur Donaldson Smith, MD

Physician, explorer, naturalist, and diplomat—Part V*

Pascal James Imperato, MD

During the four-year period 1895–1899 that spanned the interval between Arthur Donaldson Smith's two journeys to Lake Rudolf, six other expeditions reached the lake. Arthur H. Neumann, the ivory hunter, had come up from the south in 1895, and Cavendish and Andrew, who were primarily out for sport, arrived in 1897. In late December 1897 the Ethiopian Emperor Menelik sent a military column down to the lake under Ras Wolda Giorgis, the governor of the southern province of Kafa, in order to assert Ethiopian sovereignty over the region. This military expedition was accompanied by a Russian advisor, Captain Alexander X. Boulatovich (also spelled Boulatovitch and Bulatovich), who left good records of it. They moved down the Omo valley and arrived at the northern end of the lake on March 29, 1898, where they planted the Ethiopian flag. This Ethiopian military force was primarily sent out to foil any attempts by the British military expedition in Uganda under Macdonald to lay claim to this region. Macdonald's subordinate, Austin, did arrive, however, at the northern end of the lake on September 12, 1898, five and a half months after Wolda Giorgis and Boulatovich got there (see part IV). He came up from the south along the western shore of the lake. The next expedition to reach the lake was essentially Ethiopian controlled and under the titular leadership of an Englishman, Captain Montagu Sinclair Wellby (Fig 1). He set out from Addis Ababa with an Ethiopian escort on December 19, 1898, and reached the northern end of the lake in March 1899. Wellby was followed by an Ethiopian military expedition under the leadership of a Russian military officer-adventurer, Nicholas Stepanovic Leontiev (also spelled Leontieff and Leontief). He and his army left Addis Ababa on June 1, 1899, and arrived at the northern end of the lake on August 21, 1899. Some of his subordinates travelled down the western side of the lake almost to its southern end. Leontiev had come down to the lake along a more easterly route than had Boulatovich and Wolda Giorgis. However, both expeditions had primarily one purpose, namely the establishment of Ethiopian sovereignty over the region.

Arthur Donaldson Smith was well aware of the steady Ethiopian push towards the south and anticipated that any expedition going into the Lake Rudolf area would probably encounter the Ethiopians, necessitating a well-armed escort. Yet he does not seem to have known about the specifics of the expeditions involving Boulatovich and Leontiev. He knew of Wellby's plans to get to the lake, having learned of them in late 1898 before he left with Percy Cox for Somaliland.[1] Smith wrote to Cox from London describing his experience at the Foreign Office where he had gone to request permission to go to Lake Rudolf and the Nile. In his letter he said:

> . . . Sanderson whom I saw at the Foreign Office did not give me much encouragement. The last thing he said was "you know if

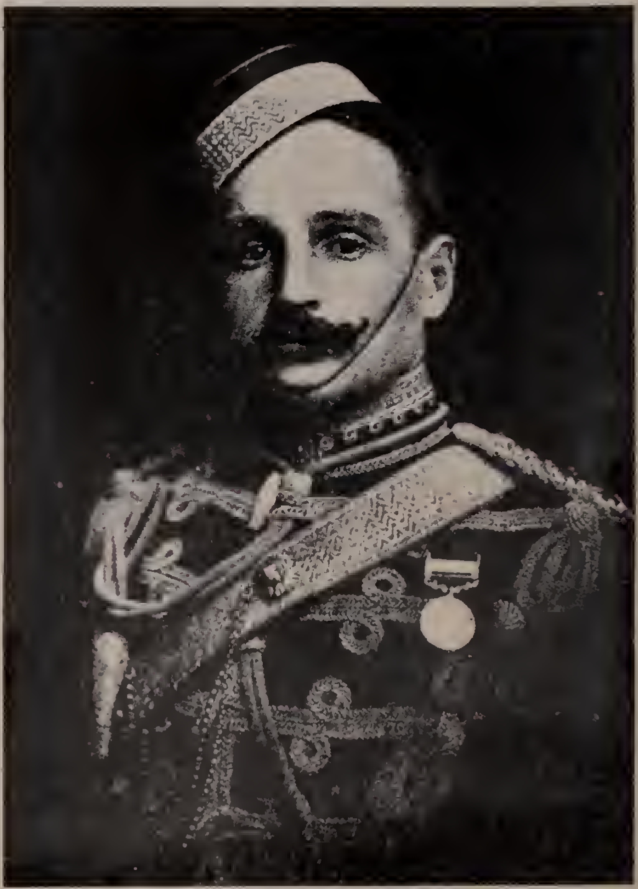


FIGURE 1. Captain Montagu Sinclair Wellby (reproduced from *'Twixt Sirdar and Menelik*, London and New York, Harper & Brothers, 1901).

---

* Parts I, II, III, and IV were published in the February, March, April, and June 1987 issues, respectively.

Address correspondence to Dr Imperato, Editor, *New York State Journal of Medicine*, 420 Lakeville Rd, Lake Success, NY 11042.

> Lord Salisbury gave you permission he would bring down a hornets nest around his ears." This means that he has refused permission to many and all applicants. Wellby was refused among others so he hit upon the plan of going with Harrington to Menelik's capital with the idea of crossing to the Nile north of Fashoda. I do not think he will succeed. Harrington told me that Menelik would not give permission to anyone to go through southern districts.[1]

Wellby was as determined as Smith and was to benefit from Menelik's Bismarckian diplomacy, which facilitated his getting to Lake Rudolf.

WELLBY'S EXPEDITION TO LAKE RUDOLF AND THE SOBAT RIVER

Montagu Sinclair Wellby and Arthur Donaldson Smith had much in common. Both were born in 1866 into well-to-do families, Wellby being educated at Rugby and later at Sandhurst.[2(pxv)] Gazetted a lieutenant in the 18th Hussars, he was promoted to captain in 1894, the year he made his first journey to Somaliland where he mapped much unexplored country. This coincided with Smith's first trip to Somaliland in 1893–1894, during which he hunted with William Lord Smith, MD, of Massachusetts. Wellby was back in Somaliland exploring the Dalbahanta country in 1895, the year that Smith first reached Lake Rudolf from Somaliland. In 1896 Wellby along with Lieutenant Malcom conducted an expedition to Central Asia, travelling from Kashmir to Northern Tibet, Mongolia and China, finally arriving in Peking, from which Smith set out in 1897 enroute to Mongolia (see part IV). In 1897 Wellby took part in the Tirah campaign as a transport officer in the Tochi Valley.[2(pxv–xvi)] Smith also participated in the Tirah campaign in 1897 as both a physician and a foreign news correspondent (see part IV).

Wellby had toyed with the idea of leading a military expedition from the south to join up with Kitchener coming down from the north (see part IV). However, as Wellby says, "...with the fall of Khartoum, my castles in the air were...considerably shattered." [2(p27)] He goes on to say:

> ...it was evident to me that there were large stretches of country, very imperfectly known, lying east of the Nile and north of our Uganda protectorate...the sooner we learnt the value of these regions...the more advantageous it would be for us and for them...It also appeared to me as clear as daylight that when the Dervish host should have been destroyed and Khartoum have fallen, King Menelik would be forced by mere circumstance to lend a readier ear to our wishes, and would also see the wisdom of winning our perpetual friendship.[2(p3)]

This was putting it rather mildly. Ethiopia was now virtually encircled on the map by British red. Menelik's apprehensions before the fall of Khartoum about British designs in Ethiopia were now greatly heightened. He was to deal with the British through tact, diplomacy, and a demonstration of his military capabilities.

Wellby received a telegram in August 1898 at Simla in India from the Intelligence Division of the War Office "informing me that Captain Harrington, H.B.M.'s agent in Abyssinia, was about to return to that country, and was willing to take me with him if I cared to go." [2(p26)] The British government was not giving Wellby permission to go to Lake Rudolf, only to go to Ethiopia with Harrington. While Harrington states in his introduction to Wellby's book that Wellby was his assistant, it is clear from the latter's later actions that he was somewhat of a free agent.[2(pxv)] He saw the opportunity of travelling with Harrington as "...the only way of getting into Abyssinia...." [2(p6)] Wellby had to hurry out of India in order to meet Harrington, who was scheduled to be in Zeila on the Somali coast on September 7, 1898. Reaching Zeila via Aden, Wellby found Harrington who had arrived the previous day.[2(p9)] Confirming what Smith had been told in London, Wellby said, "Harrington himself could give us very little hope of our being able to travel beyond King Menelik's capital, whilst the other two officers ridiculed the proposition." [2(p9)] This was reinforced by a Mr Seru, the first Ethiopian Wellby met, who "...gave us but little encouragement. Indeed he threw more cold water on our schemes, by scoffing at the idea of King Menelik allowing us to travel beyond his capital." [2(p11)]

Leaving Harrington outside of Zeila, Wellby travelled to Berbera where he arrived on September 11, 1898. His plan was to cross to Somaliland to Lugh, strike for the south of Lake Rudolf, and then from there go north up to Khartoum. However, as he found out, "this journey...interesting as it would have been, transgressed boundary treaties, and speedily vanished into dreamland." [2(p13)] Wellby found that in Berbera "...every stone of it is strictly under the official thumb, entailing endless rules, regulations, taxes, and certificates from every shooting party." [2(p13)] Wellby was effectively blocked from setting off for Lake Rudolf via Somaliland, as Smith and Cox would be several months later, and decided to rejoin Harrington and go on to Addis Ababa, the Ethiopian capital. It was there that he would pursue his scheme to get to Lake Rudolf and the Nile Valley while Smith and Cox were hatching a scheme to do much the same. Harrington and Wellby reached Addis Ababa on October 25, 1898. Harrington brought along with him the famous phonograph recording made by Queen Victoria for Emperor Menelik.[2(p16)] They were well received by Menelik, who then took Wellby with him as he led an army of some 50,000 troops into Tigre against Ras Mengasha, the governor, who had defied him. This experience provided the British with some updated information on Menelik's armed forces, which were formidable. Menelik was not averse to this intelligence getting back to London via Wellby and Harrington.

Menelik eventually gave Wellby permission to journey south to Lake Rudolf with an expedition primarily composed of Ethiopians. Wellby's reaction to this news was ecstatic:

> Harrington...told me to my astonishment and delight that his Majesty had not only granted me permission to travel wherever I liked in his country, but had also promised to forward letters to the chief officials of all the districts through which I intended to travel, informing them of the fact. Such kindness surpassed my expectation, and as I shook hands and said good-bye to the king next day, I earnestly wished him every success in the campaign he was then commencing. I also asked him if I could not make return for all his kindness. "No," he replied, "only let me have copies of the maps you make." [2(p88)]

In effect Menelik was letting Wellby tour his domain under Ethiopian escort. This reaffirmed Ethiopian territorial claims all the way to Lake Rudolf and the beginning of the Sobat River and neutralized Wellby from acting as an agent for staking out British claims in the same regions. Wellby's final party consisted of 30 Ethiopians, nine Somali, and five Sudanese. The latter were remnants of Bottego's expedition who, being penniless, could not leave the country. They were acting as artillery instructors to the Ethiopians.[2(p103),3] Wellby also took with him Shahzad Mir, a surveyor of the 11th Bengal Lancers, who had travelled with him in Tibet, and his fox terrier, Lady, who had been with him in the Tirah campaign.[2(pp6,102–104),3] The expedition left Addis Ababa on December 19, 1898, a few days before Smith and Cox headed inland from Berbera on their expedition for the Gaekwar of Baroda.

Wellby travelled in a southwesterly direction along the chain of Ethiopian Rift Valley lakes, finally reaching the northeast shore by Lake Rudolf in March 1899. There he decided to go north to throw some light on the issue of the Omo River. He "...held the testimony afforded by an experienced and scientific traveller like Dr Donaldson Smith to be sufficiently convincing...."[2(p197)]

The Omo question had been satisfactorily resolved by Vittorio Bottego, the Italian explorer, during his 1896–1897 expedition. Thus Wellby's claim of being convinced of the correctness of Smith's earlier supposition that the Omo flowed into either the Juba or the Ganana is rather surprising. Even Smith accepted the veracity of Bottego's findings and viewed the Omo question as resolved. However, Wellby had a more practical reason for wanting to go north. He had gathered from Smith's writing "that there were certain natives dwelling on the banks of the river who cultivated dhura, and I was anxious, if possible, to buy grain from them....[2(p198)] The "natives," as Wellby referred to them, fled at the approach of the Ethiopians.

Heading south, Wellby marched along the eastern shore of Lake Rudolf, and was probably the first explorer to travel to the west of the Longondoti Hills which fringe the lake shore.[2(pp215–216)] On April 8 he reached the southern end of the lake.[2(p218)] It was while he was at Lake Rudolf that Wellby drove a three-sided pointed instrument through the first finger of his left hand while opening a tin.[2(p213)] He eventually lost half of his finger. On reaching the southern end of the lake he then moved northwest through the land of the Turkana people, turning north at 5° north latitude. When at 7° north latitude he went northwest again, arriving in the Sudan-Ethiopia border country on June 1, 1899. He then moved on to the source of the Sobat River, finally reaching Nasser Fort. From there he went on to Fashoda and then down the Nile to Omdurman on the steamer *Fateh*.[2(p393)] On arriving in Cairo he met Kichener and was able "...to furnish him with the maps that Shahzad Mir had spent so much pains over—maps too, that covered hundreds of miles of unknown land."[2(p401)] Kichener, who less than nine months before had conquered the Mahdist state and more recently dislodged the French from Fashoda (see part IV), visited the expedition members and presented the Ethiopians and Somali with rugs, suits of clothes, cotton, and cloth.[2(p400),3] From Cairo, Wellby's men returned home, the Ethiopians to Zeila and Harrar, the Somali to Berbera. It was late August, a year after he had left Bombay to join Harrington at Zeila. Leaving his dog Lady and her pup with Lady Rennell Rodd in Cairo, Wellby travelled back to England from Alexandria.

Wellby had made a remarkable journey, traversing some previously unexplored country, particularly that lying west of Lake Rudolf. He had moved north through the very region that Smith so anxiously wanted to explore from east to west. Unfortunately, Wellby did not provide very good descriptions of either the peoples or the regions he visited, gave few dates in either his posthumously published book or article, and used Amharic (Ethiopian) names for people and areas, including Lake Gallop for Lake Rudolf. Turton makes the cogent observation that the highland Ethiopians with whom Wellby travelled had a "natural bent for shooting and stealing," by Wellby's own admission, which did little to foster peaceful contact with local people.[4] Turton accurately observes that one learns virtually nothing about local people from Wellby's account.[4] Von Hohnel noted that Wellby's retinue was primarily made up of Abyssinians, whom local peoples had every reason to avoid.[5] Some of them had had sad experiences in 1897–1898 when Ras Wolda Giorgis and his army went into the southern borderlands around the Omo delta and Lake Rudolf to establish Ethiopian sovereignty. The Russian army officer, Alexander X. Boulatovich, accompanied this military expedition. Turton notes that Wellby took a rather benign view of Ethiopians raiding and shooting.[4] While Wellby describes a number of skirmishes with local peoples, it is safe to assume that they were more numerous and more severe in terms of injury and death than he owns up to. For by his own admission, the Ethiopians' "...chief topic of thought and conversation was killing and raiding."[2(p215)] The Sudanese were not much better according to Wellby because on previous journeys they had been accustomed "to shoot natives and carry off property in the most regrettable manner...."[2(p226)] Yet Wellby rationalized some of the raiding and stealing on grounds of necessity. He related that his Indian surveyor, Shahzad Mir, had become unknowingly influenced by the Ethiopians, being new to the country.[2(p226)] He says of Mir, "...he was perfectly convinced in his own mind that we should never be able to come to a friendly understanding with any tribes at all on our journey and that we should have to take what we required by force...."[2(p226)] Rationalizing one of Mir's thefts of local stock, Wellby says, "...had not Shahzad Mir then helped himself to donkeys and sheep, we should probably be still struggling along the shore of the lake."[2(p227)] Wellby, while admitting to the evil of Ethiopian raids, rises to the defense of the Ethiopians in saying:

> In previous pages I have made repeated references to the raids carried out by the Abyssinians, and have, like everyone else, condemned these raids as being altogether unworthy of that nation. Yet to straight away throw blame on the Abyssinians without entering into the causes of these raids...would be unfair.[2(p330)]

Harrington thought highly of Wellby and praised him: "...[His] unfailing tact, his cheerfulness, resource in dangers and hardships, and the winning qualities which endeared him to all who ever knew him, won him the confidence and affection of the natives with whom he came in contact."[2(xvii)] Wellby was courageous as well, for as mentioned above, he cut the forefinger of his left hand opening a tin of preserves and, even though an infection developed leading to gangrene, "...he marched miles in intense agony, holding a mortifying finger upright during the whole of the long and miserable march, concealing his pain, so that his followers would not lose heart." 2(pxviii) From all of these accounts it is clear that Wellby was a courageous soldier, a skilled diplomat, and endowed with superior endurance and a unique ability to get on well with alien peoples. However, his published accounts of this African expedition show that he was not a naturalist, geographer, or ethnographer. Had he been, his expedition would be viewed in a different light than it is today because it would have contributed so much more to our knowledge about the regions he visited. In reading his book and article today one cannot help but feel that to a certain extent here was a missed opportunity, that had someone else with complementary talents been along on the journey or had the Ethiopians not been there so much more would have been learned. Wellby's journey strengthened Menelik's diplomatic offensive with regard to the British because he willingly came away with high praise for the Ethiopians and confirmatory testimony that Menelik's domains did in fact extend to where he said they did. He also observed the presence of a buffer zone where neither British nor Ethiopian rule had penetrated and "...wondered what would eventually be the fate of the country and tribes situated 'twixt Sirdar [the Egyptian title for Commander in Chief given to General Sir Herbert Horatio Kichener who then ruled for Britain in the Sudan] and Menelik." 2(p380)

Shortly after Wellby's return to England in 1899 he rejoined his regiment in South Africa where the Boer War was in progress. On July 30, 1899, he was seriously wounded at an engagement at Mertzicht and died from his wounds on August 5 at Paardekop.[6] His paper describing his expedition was read before the Royal Geographical Society by his father on June 18, 1900, and later published in the *Geographical Journal*.[3] His book, posthumously published in 1901, was dedicated to Menelik II of Ethiopia.[2] It was favorably reviewed in the *Geographical Journal*, the reviewers noting that "...Captain Wellby was an enthusiastic admirer of Menelik and his verdict regarding the Abyssinians as a whole is decidedly favorable.[7]

Wellby's major contribution to the exploration of this part of Africa was his discovery that the lands between the Ethiopian highlands and the Nile were not mountainous, as had been previously thought, but rather flat and easy to traverse. Those who attended the verbal presentation of his paper before the Royal Geographical Society underscored this point with their comments.[3] No European had ever seen these lands before, Wellby being the first. Although he had cut a south-to-north swath through the regions Smith wanted to explore to the west of Lake Rudolf, there still remained the expanse from the northern end of the lake to Wellby's line of march and from there to Macdonald's south-to-north route and from there to the Nile. Smith's anxiety about beating rivals into these unexplored regions was well founded. For following close upon Wellby's heels was a Russian officer and an Ethiopian military party whose destination was Lake Rudolf. In addition, a major British expedition under the leadership of James J. Harrison and W. F. Whitehouse planned to strike west from Lake Rudolf for the Nile, traversing the very regions Smith had longed to be the first to explore.[8]

THE RUSSIANS IN ETHIOPIA

Although it is well known today that the Soviets play an important role in the Marxist Ethiopia of the 1980s, few realize that the Russians were prominent players in this part of Africa in the last two decades of the 19th century.[9,10] The Russian agenda in Ethiopia was a complex one consisting of colonial territorial ambitions, a desire for influence and access to riches and trade, dreams of solidarity between the Russian Orthodox Church and the Coptic Church of Ethiopia, which had much in common, and the opportunity to use yet another stage on which to maneuver in the interests of big power European politics. The Franco-Russian alliance in Europe drew the Russians to the side of the French in Ethiopia and elsewhere in this part of Africa, placing them in opposition to British interests. The various elements of Russia's agenda in Ethiopia did not always function simultaneously or necessarily with the official sanction of the czar's government. Religious, Slavophile, and powerful commercial interests in Russia promoted unofficial expeditions and missions to Ethiopia and in so doing eventually drew the government into sending a semiofficial diplomatic mission to Menelik in 1895.2(pp 84-88),10(v2, pp 237-274) This mission was under the leadership of Captain Alexander Eleiseiv and Captain Nicholas S. Leontiev. The Russians disembarked at the French port of Djibouti where they were received by Léonce Lagarde, the governor (see part IV), and assisted in every way by the French, who greatly facilitated their travel to Addis Ababa.

The real purpose of the Eleiseiv-Leontiev mission to Menelik was to induce the emperor to send a diplomatic mission to St Petersburg and to persuade him to unify the Coptic Church with the Russian Church. The Ethiopians, realizing that the czar claimed to be the protector and leader of all Orthodox Christians, including themselves, were benign and vague in their response to religious unification.9(p86) Although this mission fell short of its political objectives, it was able to convince the Ethiopians to send an official mission to Russia, which in itself was a major accomplishment, given the Ethiopians' reluctance to travel outside of their own country. Eleiseiv died of sunstroke, leaving Leontiev at the head of the Russian mission. Leontiev then accompanied the Ethiopian delegation back to Russia where they were received with great pomp by the head of the Holy Synod, their official sponsor, and on July 13, 1895, by Czar Nicholas II at Peterhof.10(v2,p149) The visit of Ethiopians to the Russian court stirred dreams among the Slavophiles and imperialists that Russia would eventually obtain a political protectorate over Ethiopia via the route of French concessions of ports on the Gulf of Aden and Menelik's embracing of Orthodoxy. It was understood by all that "...lucrative concessions for various Russian enterprises would follow in Ethiopia." 9(p88)

As Jesman notes "...various Russian expeditions were now arriving in Ethiopia thick and fast." 9(p88) However, the first permanent diplomatic mission, headed by P. M. Vlassov, was not set up in Addis Ababa until October 1897.9(p91) The Russians lived in Addis Ababa on a grand scale and this along with the presence of a number of dashing Cossacks greatly impressed the Ethiopians.[9(p91)]

Russian military officers, of whom there were several in Ethiopia at this time, found ready employment in Menelik's army. Jesman makes the cogent observation that after the Battle of Adowa where the Ethiopians defeated the invading Italians, Menelik came to the realization that he had to keep a standing army instead of relying on calling one up as the need arose. Unable to take on his rival, Ras Mengasha of Tigre, in the north because of political and international reasons, he decided to deploy his 200,000 ill-disciplined troops elsewhere to keep them busy, and to place Russian officers among them.[10(p93)] Jesman maintains that Menelik sent his generals south and west to extend the frontiers of his dominions in an attempt to turn what had been a gradual cultural expansion into outright conquest.[9(p93)] It is doubtful that much cultural expansion had been taking place as Jesman states, given the historical record which clearly shows that the highland Ethiopians had no stomach for the lowlands that Menelik now wanted to subjugate. But that is an issue tangential to this discussion. Although conquest may have been one of Menelik's means for keeping his armies busy, his principal motivation for extending the limits of his kingdom was the steady encroachment of the British, whom he greatly feared. With regard to Lake Rudolf and the Omo River delta to its north, two Russian military officers were to play major roles with Ethiopian conquest armies sent into these regions. The first was Lieutenant Alexander X. Boulatovich, a young nobleman and an officer of the Grondo Hussars,[9(p94),10(v2,p262)] and the second was a uniquely colorful figure, Captain Nicholas S. Leontiev, a Russian nobleman who was destined for an unusual career as a buccaneer and international intriguer during the unbridled days before political boundaries were etched across the map of this part of Africa.

ALEXANDER X. BOULATOVICH—THE FIRST RUSSIAN AT LAKE RUDOLF

Menelik, always suspicious of British intentions towards Ethiopia, became particularly alarmed in mid-1897 by Macdonald's activities to the south. Sanderson makes the important point that while Menelik saw Kichener to the north as his greatest threat, he also overestimated the threat posed by Macdonald in the south.[11] He was fully prepared to confront Macdonald because he was confident that he would win in any military encounter. Lord Salisbury was anxious to avoid any such confrontation so as to not jeopardize operations in the Sudan and along the course of the Nile. Menelik of course would never have believed this for one instant. He acted on the basis of his own analysis of the situation and decided in late 1897 to send a military force down to Lake Rudolf in order to assert Ethiopian sovereignty over the region.

Menelik entrusted this military operation to Wolda Giorgis, who was then the governor of the Kafa province in the southern part of the country. Lieutenant Alexander X. Boulatovich was sent out from Addis Ababa to meet Wolda Giorgis in Kafa and to travel with him down to the shores of Lake Rudolf.[12] Boulatovich was an officer in the Grondo Hussars of the Imperial Guard.[10(v2,p249)] Jesman notes that Boulatovich wore a gold earring in one ear as was his right as an officer of the Grondo Hussars.[9(p94)] This he says made him popular with the Ethiopians because in their country such an earring was a badge of honor worn by those who were mighty hunters. Jesman gives Prince Henri d'Orleans as the source of this story.[13(p104)] Prince Henri (Fig 2), who was in Ethiopia at the time, says the following of Boulatovich's earring: "... we camp near a lieutenant of the Russian guard, Boulatovich, who returned; he had just killed several elephants, and they put into his ear a small button of gold filigree." [13(p104)] Boulatovich, like Leontiev, was extremely popular with the Ethiopians because of his openness and willingness to closely associate with them. Boulatovich left for Kafa on December 17, 1897, with a caravan of 32 men, among whom was another Russian soldier, Zelepukin, also a Hussar of the Imperial Guard. He took along with him 18 mules and three horses.[12] He met Wolda Giorgis who had under his command an army of from 16,000 to 20,000 men, and on the 24th of January 1898 they began their march towards the southwest in the direction of Lake Rudolf. Enroute they passed near Mount Smith, the mountain range that Smith had named after himself during his 1894–1895 expedition (see part III). Boulatovich says in the Italian language version of his memoirs of this trip that "...una di esse aveva forma piramidale col vertice acuminato; era il monte Dime, al quale Smith diede il proprio nome...La latitudine del monte Dime mi risultò di alcuni minuti più meridionale di quella determinata dalla Smith." [12] In saying this Boulatovich was succumbing to taking a swipe at Smith for naming the mountain after himself and for allegedly miscalculating its exact geographical position. Yet Boulatovich did not hesitate to name another chain of mountains in Kafa after Czar Nicholas II.[12]

Descending the Omo Valley, the expedition reached the northern shore of Lake Rudolf on March 29, 1898, where Wolda Giorgis raised the Ethiopian flag. They did not remain long in the area for as Boulatovich says, "...e il giorno dopo prendemmo la via del ritorno." [12] Two weeks later Boulatovich was back in Kafa and by May 5 in Addis Ababa.[12] During the journey Boulatovich, who was also a military engineer, constructed a series of Ethiopian forts that were intended to impede the presumed advance

FIGURE 2. Prince Henri of Orleans after shooting two Colobus monkeys (*Colobus guereza*), 1897 (reproduced from *Une Visite à l'Empereur Ménélick*, Paris, Librarie Dentu, 1898).

of the British, specifically Macdonald, from the south.[9(p94),10(v2,p274),12] Wolda Giorgis and Boulatovich had achieved Menelik's objective of establishing Ethiopian claims as far south as the Omo River delta and the northern shores of Lake Rudolf.

Boulatovich returned to Russia later in the year and on January 13, 1899, presented the results of his trip before a conference of the Imperial Russian Geographical Society.[9(p95)] By then he had been promoted to captain.[12] This presentation was published in French in St Petersburg[14] and later in Italian in Rome, along with a 1:2.8 million map similar to Bottego's for the Omo delta and the northern end of Lake Rudolf.[12] The Italian translation of his presentation was unnecessarily and, as Butzer notes, "abusively footnoted by G. Roncagli." [15(p112)] Roncagli's acidic comments, which for the most part are inappropriately critical of Boulatovich's observations, contributed to the view held for many years that his journey was of little scientific interest. His verbal presentation before the Russian Geographical Society had to be brief and he clearly made no attempts to provide the geographical and anthropological details scholars treasure. Yet Boulatovich also wrote a detailed account of his travels through Kafa in Russian, containing numerous illustrations and a map which won the praise of F. J. Bieber, who published one of the earliest classical studies of Kafa in 1920 and 1923.[16] He said the following of Boulatovich: "... the first European who penetrated into Kafa after the Ethiopian conquest and who had the opportunity of freely traveling the country was a Russian, a captain of the Imperial Guard, A. X. Boulatovich. He published a book about his voyage full of numerous illustrations and a map; unfortunately not accessible but to the Russians." [16(v1,pp19-20)] Bieber praises Boulatovich for his detailed account of the history of the region and of the Ethiopian conquest, and his detailed descriptions of peoples and customs, geography, commerce and trade, ceremonials, and even the status of women.[16(v1,pp19-20)] Because this work was not translated from the Russian it remained inaccessible to most western scholars, and thus Boulatovich's worth as a geographer and ethnographer was not appreciated until more recently.

Boulatovich made the important observation in his brief account before the Imperial Russian Geographical Society that Smith had mistaken a branch of the Omo for the river itself during his attempt to resolve the issue of the source of Lake Rudolf.[12] This is correct as Smith, by his own later admission, probably followed the course of the Mago, a major affluent of the river. Boulatovich's observation in this regard is all the more significant because he travelled through the region before he had access to the findings of Bottego's expedition which resolved the issue of the Omo.[12]

Boulatovich returned to Ethiopia in July 1899 and travelled on behalf of the Ethiopians in the northwest in the region of several affluents of the Blue Nile.[10(v2,p276)] This expedition lasted four months, he returning through Shoa via a different route than he had followed on entering.[10(v2,p277)] He returned to Ethiopia again in 1906 but there are no known records of his activities during the year that he was there.[10(v2,p277)] He participated in the Russo-Japanese War, fought against the Germans in World War I, and was taken prisoner. He and a group of other Russian soldiers escaped from their German prisoner of war camp and returned to Russia. He was killed on the eve of the October Russian Revolution by a band of pillagers at a country home he had built himself.[10(v2,p275)] The Italian General Gherardo, who was taken prisoner at the Battle of Adowa, got to know him well and describes him as a "... young man from a noble family, strange, full of energy and vitality, quasi-mystical and of delicate sentiments." [17(p126)] In more recent years Boulatovich's contributions to our knowledge of that part of Africa in those times have been increasingly recognized and appreciated.[18] Most writers give his middle initial as K. However, Zargi, who is a leading authority on the 19th century Russian experience in Ethiopia, states that his middle name was Xavierovic.[10(p323)] It is this form that has been followed in this presentation.

NICHOLAS S. LEONTIEV—A RUSSIAN BUCCANEER

Wellby's expedition to Lake Rudolf left Addis Ababa some seven months after Boulatovich returned. The next Ethiopian-sponsored expedition to the lake area was far more elaborate than the previous two and was led by a Russian nobleman, Captain Nicholas Stephanovic Leontiev (Fig 3). Leontiev had first arrived in Ethiopia in 1895 with Captain Alexander Eleiseiv at the head of a semiofficial Russian mission to the court of Emperor Menelik. Eleiseiv's death from sunstroke placed Leontiev at the head of the mission and in effect launched him on a colorful career as a buccaneer in Ethiopia that was to last for several years. Leontiev's star rose in St Petersburg when he returned there with an Ethiopian mission to the czar in 1895. Back in Ethiopia he was in Addis Ababa during the days before the war between Italy and Ethiopia broke out in 1896. Although he did not participate in the Battle of Adowa, he arrived on the scene two days later and attempted to play a role in the peace negotiations between Italy and Menelik.[9(p114),10(v2,pp179-235)] The Russian press and officials in St Petersburg saw him as the central ingredient in Russia's attempts to forge a colonial role in Ethiopia.[9(p114)] Leontiev was greatly liked by both the Ethiopians and the Europeans who got to know him, and his

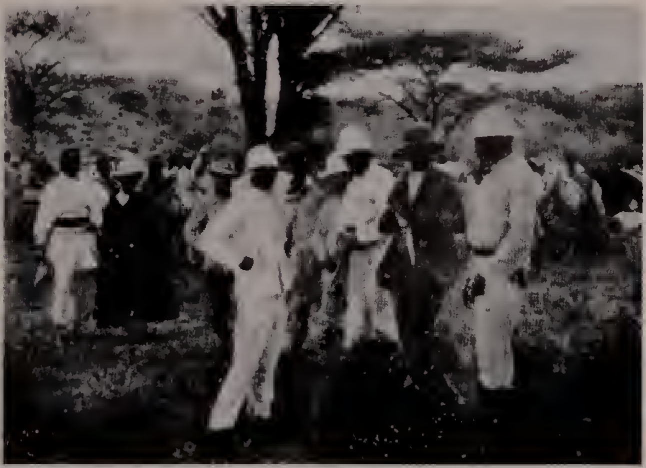


FIGURE 3. Count Nicholas S. Leontiev (first on right) standing next to Léonce Lagarde, Ethiopia, April 1897 (reproduced from *Une Visite à l'Empereur Ménélick*, Paris, Librarie Dentu, 1898).

"breezy friendliness"[9(p112)] cast a wide net of contacts. Jesman makes the point that unlike many other Europeans of the day, he had a "total absence of race prejudice"[9(p112)] which was an enormous asset in addition to his political skills.

Leontiev rapidly rose in Menelik's favor which eventually stirred up resentment against him among jealous Ethiopian nobles. However, in 1897, when James Rennell Rodd's mission to Ethiopia arrived in Addis Ababa, his star was still rising. Rodd said of Leontiev:

> The Russian Leontieff, on whom I called with Wingate, was a handsome tall fair-bearded adventurer of plausible address. He was described as Count Leontieff, having apparently received that title, hitherto unknown in Ethiopia, from Menelik, who had also, we were amused to hear, exalted M. Lagarde to the rank of the Duke of Entotto![19(v2,pp157-158)]

Shortly after Rodd met Leontiev, the latter was off to Europe in order to obtain financial backers for a proconsulate Menelik had promised him in the southern part of the country bordering on Lake Rudolf.[9(p115),10(v2,pp294-308)] Menelik had in effect promised to make Leontiev governor (Dejazmach) of Equatoria, a newly created province in the south. Leontiev found no paucity of bankers, merchants, aristocrats, and others who were willing to invest in what they were told was one of the potentially richest corners of Africa. Leontiev saw this new province as a sort of personal fiefdom, the Russians as a future colony or protectorate, and the French as a foothold for themselves in Ethiopia. They quickly fell in with Leontiev's scheme, led by Prince Henri of Orleans who planned to travel with Leontiev into the new province.[9(p115),13] Prince Henri had arrived in Ethiopia in early 1897, hoping to join a joint French-Ethiopian expedition to the Nile.[13(pp1-2)] However, he had a violent argument with Bonvalot (see part IV) and as a result did not take part in the expedition.[19(p152)] Prince Henri, who was an outspoken advocate for France's plans for a place on the Nile, made no attempts to hide his anti-British sentiments in the many interviews he regularly gave to the press.[19(p152)]

Leontiev's chartered steamship loaded with supplies and weapons sailed from Holland for Djibouti on December 3, 1897. The ship stopped off in London where it was impounded on the grounds that its dangerous cargo of ammunition and weapons violated regulations, which required advance notification. This Leontiev was of course not foolish enough to give.[9(p114)] The British naturally had other reasons for impounding the ship. Leontiev and the ship's captain were heavily fined and the matter was to go to court for a public hearing, which Leontiev could not allow. So he ran off to Paris and in effect started all over again. The action of the Port of London was seen in Paris and St Petersburg as a calculated attempt to stall Leontiev, whose mission could not bode well for British interests in that corner of Africa. It had the net effect of rousing strong patriotic feelings in Russia and anger in Paris, which resulted in the original investment being rapidly replaced. In addition, the French government now entered the picture in an official manner by promising Leontiev 300 Senegalese riflemen who were to join him in Djibouti.[9(pp115-116)] Leontiev and Prince Henri arrived at Djibouti on March 16, 1898, with all of their supplies, weapons, and ammunition. Along with them came a number of French nationals whom they had recruited. The Europeans in the party were told by Leontiev that they would all eventually become shareholders in the new dominion, Equatoria, that he was about to establish in southern Ethiopia.[20(p119)]

Leontiev was officially invested as Dejazmach in Addis Ababa by Menelik and given almost absolute authority over all of the lands to the northeast of Lake Rudolf.[9(p117),10(v2,p305)] No Europeans except the Swiss Alfred Ilg, who was Menelik's chief advisor, had risen so high at the emperor's court. Not surprisingly, Leontiev soon became the object of both European and Ethiopian envy. Leontiev's plans went off course when on June 30, 1898, he was accidentally wounded in Harrar while demonstrating a machine gun to the governor of the city. His wounds were serious enough to require that he be repatriated to Europe. The Russian ambassador P. M. Vlassov refused to send Russian doctors who were in Addis Ababa to help Leontiev, seeing in his injuries the hope of ridding himself of an interloper who had caused him so much trouble.[9(p117)] This did not go over well with Menelik and the Ethiopians, who saw in Vlassov's actions the abandonment of a fellow countryman in distress. Menelik sent his own physician to treat Leontiev and, although Vlassov continued to be a powerful diplomatic and political presence in Addis Ababa, his handling of this incident caused him to fall from the emperor's favor. Fearing that he might be assassinated by Leontiev supporters, he eventually fled from Addis Ababa.[9(p118)] The Russians, standing on their own pride, retaliated by not sending an ambassador to replace Vlassov for almost four years.[9(p118)]

Leontiev rapidly recovered in Europe and was back in Ethiopia within a few months. However, Prince Henri had to return to France for family reasons and was not able to participate in Leontiev's expedition into his new domain near Lake Rudolf.[9(p122)]

LEONTIEV'S EXPEDITION TO LAKE RUDOLF

Leontiev's expedition to the Lake Rudolf region was by far one of the largest and best equipped to ever enter the area up to that time. His second in command was Baron Chedeuvre, who was an officer of the Russian regiment of Dragoon Guards who had been given the Ethiopian title of *Fitaurari* (colonel) by Menelik for the purposes of the expedition. Under him were Ensign Babitchev and a French lieutenant, Sebillou. In addition there were several cossacks of the Imperial Personal Bodyguard Squadron, and a physician, Dr Kahn, who was the expedition's medical officer. The French component of the expedition consisted of 130 Senegalese riflemen who had been brought in from Dakar in West Africa, Marius Bouchier, a French colonial administrator who acted as the quarter master for the expedition, and Captain Leymarie, who commanded the Senegalese troops. In addition Leontiev had recruited a troop of Arab scouts in Harrar who were mounted on dromedaries. The Ethiopians sent 2,000 cavalry and infantry with Leontiev under the command of Dejazmach Tessama, who the year before had travelled with the French in their attempt to reach the Nile from the east (see part IV). Leontiev had at his disposal 1,200 modern repeater rifles and two Maxim guns. Several Russian officers joined the expedition later on.[9(pp118-119),10(v2,p306),18]

Leontiev left Addis Ababa on June 1, 1899, heading south for Lake Rudolf, following a route similar to that taken by Wellby a short time before. After passing through Walamo he headed west toward the Omo River, eventually arriving at the town of Bako, where he established an Ethiopian military and administrative presence.[21,22] Moving further south towards the lake he encountered "...forte resistance"[21] from the local populations. This resulted in serious armed conflicts between Leontiev's entourage and the local people, which he describes in only a very general manner.[21] He provided idyllic descriptions of the country he traversed in his published accounts mainly for the benefit of his investors and future investors who might have been interested in everything from copper and ivory to cattle breeding.[21,23] However, as Jesman notes, "...news from other sources about Leontiev's activities in this faraway corner of Ethiopia were less rosy." [9(p121)] There was a drought in progress at the time of his visit which forced him to "resort to harsh methods in order to feed his men." [9(p121)] The expedition reached the northern end of Lake Rudolf on August 21, 1899.[18] There he found the Union Jack put up by Austin who had come up from the south the year before in 1898. Leontiev says of this that:

> Nous parvenons à un campement délaissé, en 1898, par un officier d'une puissance étrangère venu de sud...il arbora son pavillon sur territoire déjà officiellement redvendiqué par l'Empereur d'Ethiopie. Je retablis les droits de l'Empereur en mettant à sa place le drapeau éthiopien.[18]

Thus, on finding Austin's Union Jack, Leontiev removed it, replacing it with the Ethiopian colors on the grounds that the territory had already been claimed by Menelik. It is of interest that he avoids mentioning both Great Britain and Austin by name. His stay at the northern end of Lake Rudolf was scarcely peaceful. A four-hour combat erupted between the local inhabitants and his men, resulting in 216 of the latter being killed and several injured. Among the injured was Baron Chedeuvre and a cossack named Gogassoff. Given the fact that the local people had only poisoned arrows and spears, one can gauge the numbers of them that must have been mustered in an attempt to drive Leontiev and the Ethiopians out. Leontiev expresses regret in a matter-of-fact manner that the nature of the country they were in prevented them from using machine guns on the local Africans.

Sebillou hoisted several Ethiopian flags near the Omo delta and supervised the construction of a fort on a rise overlooking both the lake and the Omo estuary.[21] The fort was built on the left bank of the Omo while Leontiev returned to Addis Ababa in order to confer with the emperor.[21] He had reasons for returning to Addis Ababa. However, before he did, he instructed Baron Chedeuvre and Dr Kahn to take a column down the western shores of the lake and to explore the lands between it and the Nile.[21] This was an ambitious order that was only partially carried out. Chedeuvre and his men only travelled down the western shore of the lake to just below the Turkwell River, where on October 1, 1899, they set up Fort Menelik II.[21] They then returned north again.

Leontiev's expedition did contribute something to an understanding of the geography of the Lake Rudolf region. As Butzer notes, his 1:1.5 million map, constructed by Doctor Kahn and the French in the party, accurately depicts many of the meanders of the Omo River delta.[15(p114)] Butzer says of Leontiev's map: "...Leontiev's rendition of the Omo channel configuration remained unique until the 1930's...." [15(p114)] However, the Royal Geographical Society in summarizing the geographical contributions of the expedition at the time noted that:

> Count Leontieff claims as a new discovery, that of the peninsula cutting off the bay at the north-west end of the lake; but this is an error, as it was already shown in Mr. Cavendish's map. Little seems to have been added to the information already collected by that traveler, Captain Bottego, and Major Austin, with whose accounts Dr. Kahn's statements agree...[19]

Butzer, a modern geographer, notes that Leontiev's contributions to knowledge of the Omo delta were overlooked by a number of people who subsequently wrote about the region.[15(p114)]

LEONTIEV'S EXPEDITION IN PERSPECTIVE

Leontiev carried Menelik's standard into the heart of what is now northern Kenya, so far south in fact as to make Menelik uncomfortable. Leontiev returned to Addis Ababa because the emperor recalled him, concerned about the larger international implications of his activities. As Jesman cogently notes, Menelik was worried that an unchecked Leontiev would eventually entangle Ethiopia in an unwanted conflict with Great Britain, which at the time was the leading European power in the region. Relations between Ethiopia and Britain were strained at the time and Leontiev's removal of Union Jacks was highly provocative.[9(p123)] It was widely known that Leontiev was fiercely anti-British and Menelik feared that these sentiments would get out of hand and drag him into a disastrous confrontation. Menelik's view of the British had also changed since the time Boulatovich went down to Lake Rudolf. He was aware of how they had put down the Ashanti in West Africa and he was convinced that although they were suffering from some initial reverses in the Boer War in South Africa, they had the capability to effectively fight a local campaign against him and win.[9(p123)] He had moved from a position of willingness to confront to one of diplomatic compromise, and seems to have realized that the northern end of Lake Rudolf was about as far south as he could push his frontier.

Of lesser concern to Menelik, but yet troubling to him, were the widely circulated reports of how Leontiev had treated the local populations. Powell-Cotton, who was in Ethiopia in 1899, planning to travel with the Harrison-Whitehouse expedition to Lake Rudolf, says of Leontiev:

> ...M. Gattiker, the brother-in-law of M. Ilg rode up, and after mutual salutations, gave us some very interesting information about Count Leontieff's expedition on the Omo, in the direction of Lake Rudolf. This Russian officer, had, it appeared, found a great drought, as a result of which the cattle were dying and the crops burnt up. M. Gattiker added, that Leontieff and his men boasted of having shot natives down at sight for the sake of the ornaments they wore, that they raided every village they came to for ivory, and that if the natives did not fly at their approach they poured volleys into them until they did. All this augured ill for the success of our expedition." [24(p72)]

Powell-Cotton received his information from the brother-in-law of Alfred Ilg, a Swiss national, who for many years served Menelik as an advisor and later as counsellor of state. Thus Menelik was surely aware of these accounts and of their impact on the international image he was trying to build. However, this was not all that had transpired during the voyage. Some of the Senegalese riflemen and Dr Kahn deserted the expedition.[25(v1,p129)]

John Lane Harrington, the British agent in Addis Ababa, was assured by the emperor that the Union Jacks would be replaced.[25(p203)] In order to separate Leontiev from the British sphere to the south, Menelik deprived him of the frontier regions near Lake Rudolf in January 1900, sending there instead Dėjazmach Loma,[9] who was instructed to avoid antagonizing the British.[9(p123)] Leontiev's star was beginning to set, although his dismissal was still two years off. The precipitating cause of his final disgrace was Menelik's anger on discovering that he had set up several companies in Great Britain and in Belgium for the purpose of mining gold in his Equatoria province. Since gold mining was the exclusive monopoly of the Ethiopian crown, Leontiev had seriously infringed on a major imperial prerogative.[10(v2,pp308-309)] Menelik learned of the foreign investors in Leontiev's gold mining scheme from Belgian investors who complained to him after they realized that they had been swindled.[9(p123)] Menelik, according to Jesman, was also acutely embarrassed and felt that his international position was lowered by Leontiev's scheme involving nationals of a neutral country such as Belgium.[9(p123)]

In a sense Leontiev's gold mining scheme was the last straw. He was ordered out of the country and on May 8, 1902, Menelik sent a circular letter to all of the European ambassadors in Addis Ababa informing them that Leontiev had made a number of false statements in Europe concerning the alleged gold-mining concessions he had obtained. The emperor stated that Leontiev had inaccurately translated the text of an Imperial Letters patent that had been addressed to him on June 10, 1897, and that all of this had resulted in heavy financial losses for a Belgian company. The emperor asked the diplomats to inform their governments of Leontiev's misdeeds so that in the future he would not be able to create disputes between various parties. Expressing his disappointment in the man who had to some degree served him well, he said: "Je ne pensais pas que le dédjas Leontieff sortirait ainsi de ma volonté et de ma service." [10(v2,p309)] Jesman makes the important observation that Leontiev had become too flamboyant and politically too inconvenient for Menelik and simply had to go.[9(p124)] He spent the winter of 1902–1903 in Djibouti and by April 1903 had liquidated all of his interests in Ethiopia.[25(v2,p130)]

Leontiev briefly fought in the Russo-Japanese War in Manchuria and returned to Djibouti in 1906, from which he made an unsuccessful attempt to return to Ethiopia.[9(pp124-125)] He set up a questionable agricultural plantation in Djibouti and an arms importing firm with the help of the Oriental secretary of the Russian legation in Addis Ababa, Ludwik I. Skarjinskij, but these came to nothing.[9(p125)] Jesman states that Leontiev probably died in Paris in very reduced circumstances in 1911, and in any case before 1914.[9(p125)] Zaghi, who consulted Ilg's papers in Zurich, gives his year of death as 1910. Ilg wrote to Leontiev from Zurich in November 1909, giving his thoughts on the political events then unfolding in Ethiopia.[10(v2,p312)] On July 22, 1910, Ilg wrote to Countess Leontiev, offering his condolences on her husband's death. In this letter he said: "May the earth be light on our poor friend." [10(v2,p312)]

Thus came to an end the life of one of the most unusual and colorful freelancers who raced across the African scene at the close of the colonial scramble for Africa and who figured in the exploration history of Lake Rudolf. In summing up his life and career Jesman says:

> He failed in Ethiopia . . . through lack of understanding of the wider implications of his designs. He had never been able to make up his mind what he ultimately wanted. Sometimes he was a shrewd empire-builder like Cecil Rhodes or Peters; sometimes he imagined himself a great feudal lord . . .; sometimes he was first and foremost a big-game hunter. At all times he was an adventurer in quest of big money . . ." [9(p125)]

SMITH'S JOURNEY TO LAKE RUDOLF AND THE NILE

Arthur Donaldson Smith and Percy Cox left Somaliland for India on April 9, 1899, travelling via Aden (Fig 4). It was Smith's plan to return to East Africa and travel into the partially unexplored regions lying between Lake Rudolf and the Nile. In describing his preparations for this expedition he says:

> The whole of the winter of 1898–99 I was in the jungle of Somaliland, hoping for an opportunity to cross the border, but owing to political reasons and the gratuitous interference of the Consul-General [James Hayes Sadler], I was unable to put my project

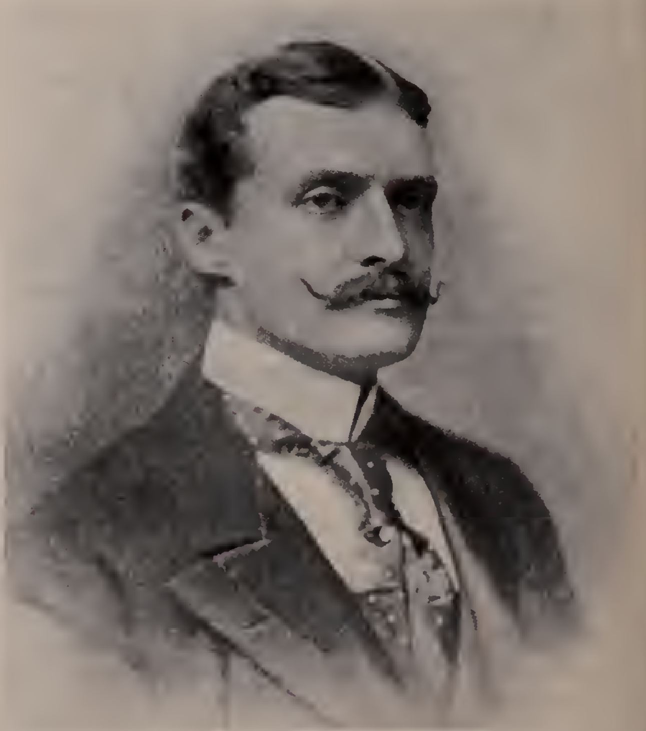


FIGURE 4. Arthur Donaldson Smith, MD, in 1900 at the time of his expedition to Lake Rudolf and the Nile (reproduced from H. Johnston, *The Nile Quest. A Record of the Exploration of the Nile and Its Basin*, New York, Frederick A. Stokes & Co, 1903).

> into execution until mid-summer. During May and June, while negotiations with the Foreign Office were underway, I employed my time in recruiting twenty-eight Sikhs and Gurkhas in the Punjab. I thought it advisable to enter *via* Kismayu, but the Mombasa local authorities stopped me. To be brief, I fitted out three distinct expeditions before I managed to get finally out of the reach of incomprehensible officials. I started from Berbera on August 1, 1899. . . ." [27]

Cox assisted Smith in the recruitment of the men and even escorted them to Aden, as Graves describes:

> Cox might have taken home-leave immediately but felt he must first fulfill his obligations to Donaldson-Smith and he consequently took charge of the transport of the Indians to Aden on his way home. He travelled in a small vessel, leaving his wife to follow him to Aden in a liner. About eight miles east of Aden his ship encountered a storm, so violent that she could make little headway. . . the ship was clearly in danger and her Master informed Cox that he would draw in towards the Arabian coast to get what protection it offered, and send a boat along the shore to get assistance from Aden.[28(pp62-63)]

Cox volunteered to go with the small boat to Aden in order to get assistance for the ship, whose coal supply was running low.

> After twenty-four hours drenching and buffeting, Cox reached Aden where the Authorities immediately sent help to the ship. He found his wife who had arrived before him, waiting anxiously for news on the dreary Quarantine Island. A few days later they sailed for England.[28(p63)]

Smith attempted to go into the interior through as much unexplored territory as possible. This accounts for his trying to enter via Mombasa and Kismayu. Bottego and Ruspoli, the two Italian explorers, had reached the Lake Rudolf region by entering near Kismayu, and Chanler had entered not far from Mombasa. Smith could have explored much unknown territory by following parallel routes, but this was denied him by the British authorities as he states. His candid, acid comments about Sadler, made as they were in an address before the Royal Geographical Society on November 12, 1900, when he presented the results of his expedition, indicate the level of his anger.[27] Sadler had acted as a spoiler from the beginning for reasons which are only partially clear (see part IV). He and other British colonial officials of the day were not keen about a non-British national exploring uncharted territory claimed by Britain.

Once Smith got off, his troubles were far from over. In fact they had just begun. After the first march, ". . . eleven of my Sikh ex-Sepoys deserted, having become aware that they must sweat occasionally, and that they could not be supplied with the manifold luxuries they were accustomed to in the Indian army." [27] In a letter written to J. Scott Keltie at the Royal Geographical Society from Fort Berkeley on the Nile seven months later on March 21, 1900, Smith says more of this desertion and of Sadler's interference:

> . . . Col. Sadler was most gratuitous in his interference and the ball having been sent rolling I met with opposition in Mombasa . . . eleven of my Sikhs deserted one march from Berbera and were promptly enlisted by Sadler in Gov't service. They had been previously told by Sadler's agent that they were wanted by Gov't. Exceedingly rich diplomat is Col. Sadler . . ." [29]

Smith had caught Sadler with something more than a smoking gun. Chanler had done as much with General Lloyd Mathews, who had probably sabotaged his expedition by encouraging the desertion of his men (see part II). Whereas Mathews succeeded in causing the collapse of Chanler's expedition, Sadler failed in using the same strategy against Smith. Sadler's sabotage was not only confined to encouraging desertion. Smith states:

> Knowing that I should never succeed if I took one of the officially recognized headmen with me, I had great difficulty in getting Somali camel-men, since all the Berbera headmen formed a ring against me. Several camel-men having deserted me at Hargeisa and fearing further official interference, I started across the Haud with only seventeen Somalis, and as many Gurkhas and Sikhs, besides my assistant Mr. Frazer [Smith's taxidermist], and my excellent Goanese cook [Gaetano] kindly lent me by Captain P. Z. Cox." [27]

Although Smith does not elaborate on the reason why the official headmen in Berbera formed a ring against him, it is obvious that they were encouraged to do so by Sadler, the British resident, who had sweeping authority over them. Despite all of Sadler's attempts to destroy the expedition, Smith made it to the Webi Shebeli River by September 8, 1899, a little more than a month after leaving Berbera.[27] He had covered 400 miles and mapped many new areas. His party consisted of only 36 men by then.

> All the way to the Juba River we had work to do filling in what had been to all intents and purposes a blank upon the maps, except where I came upon my old line of march in 1895 at Turr.[27]

Smith's route crisscrossed his 1895 one and he tried to move through as much parallel unexplored country as he could, surveying as he went, using instruments borrowed from the Indian Survey Department. By October he was in the country of the Boran, who this time he found ". . . most friendly." [27] He also had some good words to say about the Ethiopians: "In 1895 I saw the worst side of their treatment; but on my recent journey, I found that their treatment of the tribes, once thoroughly brought to submission, was commendable." [27] When he arrived at El Dere, Smith had reached the midpoint of his journey, 750 miles from Berbera and an equal distance from the Nile.[27] Thus far Smith had had a number of problems with both the Somali and the Indians in his party and had some rather harsh words to say about them.

> My Somalis gave me infinite trouble and annoyance. They were never satisfied unless they had over ten pounds of camel meat or mutton a day per man, and when food was scarce I had to be constantly on the alert to prevent them from poisoning my transport animals, so that they could eat the meat, or from stealing sheep, but they were not, however, as miserable petty thieves as most of my Indians. All but four of them mutinied at a place called Gof on November 4, and remained out over night, putting me in a decidedly disagreeable situation. If I call my Somalis howling humbugs, I describe some bad points of their character, but it would be unjust in me not to mention their superb physical condition, swift-footedness and endurance, and their intelligence, which had them compare favorably with the Indians.[27]

Of some of the Gurkhas and Sikhs he said: ". . . [they] continually strove to commit suicide by hiding if the march were long, and gave me infinite trouble in hunting them up."[27]

Smith even captured a robber near Milmil and enlisted him in his party, so in need was he of men.

> One of the robbers were captured by my boys and brought before me. He was a lad of eighteen, with wiry limbs and an intelligent expression, and struck me as a likely recruit. So I contented myself by making him march handcuffed for a day, and then enlisted the homeless fellow as a camel-man. He became known as Ali Hatbury, or "Ali of the handcuffs," and proved one of my best boys.[27]

In early November Smith passed Egder, which he had visited on his first expedition. In 1895 it had taken him nine months to reach Egder from Berbera. He had now reached it in just three months.[27] Smith found Arab and Somali traders from Kismayu in the region who were trading in ivory and cattle. He and his men moved on toward Mega and then headed for the southeast end of Lake Stefanie. Before reaching the lake, Smith obtained a ". . . specimen of the tiny gazelle, or dig-dig, of this plain, which I presented to the British Museum, [which] has been proved to be new to science by Mr. Oldfield Thomas (*Madoqua guntheri smithii*). . . ."[27] While in this region Smith ascended a mountain called Janissa from which he was able to pick out the points of his previous journey, the Tertala Range, Lake Rudolf, Lake Stefanie, and Mount Koroli. He was able to ". . . check my work, which I was most pleased to find correct."[27] Thus Smith was not only surveying new territory but also checking the accuracy of the mapping he had done during his previous trip. While on Janissa he shot two specimens of Chanler's reed buck, *Cervicarpa chanleri*, ". . . a beautiful antelope not known to exist near Lake Stefanie."[27] Smith and his men moved on toward Lake Stefanie, looking forward to plenty of fresh fish and plenty of water for washing.[27]

Smith reached Lake Rudolf on September 10, 1899, some four months after leaving Berbera. During his 1894–1895 trip it had taken him a year to reach the lake from Berbera. He found that the Murle of the Omo delta had been reduced to poverty and said that "The secret of the natives' distress was to be found in Abyssinian raids."[27] However, this was only part of the story. The Omo was ". . . half-dried up like all the streams we had crossed"[27] (Fig 5). It appears possible that a combination of drought and Ethiopian raids was responsible for the sorry state of the inhabitants of the northern end of the lake. Concerning the source of Lake Rudolf, Smith says the following:

> On my first journey I followed up what I supposed to be the Nianam for a long distance to Mela; but since I was there that illustrious explorer, the late Captain Bottego, discovered another river, the Omo, coming in from the west. It is clear to me now that my river, which I will call the Mela [Mago] and the Omo together in equal volume join at Gumba to form the Nianam, the name given by Count Teleki to the large stream flowing into the lake.[27]

In saying this Smith admitted that he had in fact travelled up the Mago on his first trip and not the Omo. He put forth the opinion that the Mago and the Omo contributed equal volumes of water to a third and final stream, the Nianamm, which then flowed into the lake. He could not have accurately assessed the two rivers from a hydrological point of view, and was thus making something of an educated guess at best. The Omo River is in fact the principal source of Lake Rudolf and the Mago, which Smith explored, a major tributary.

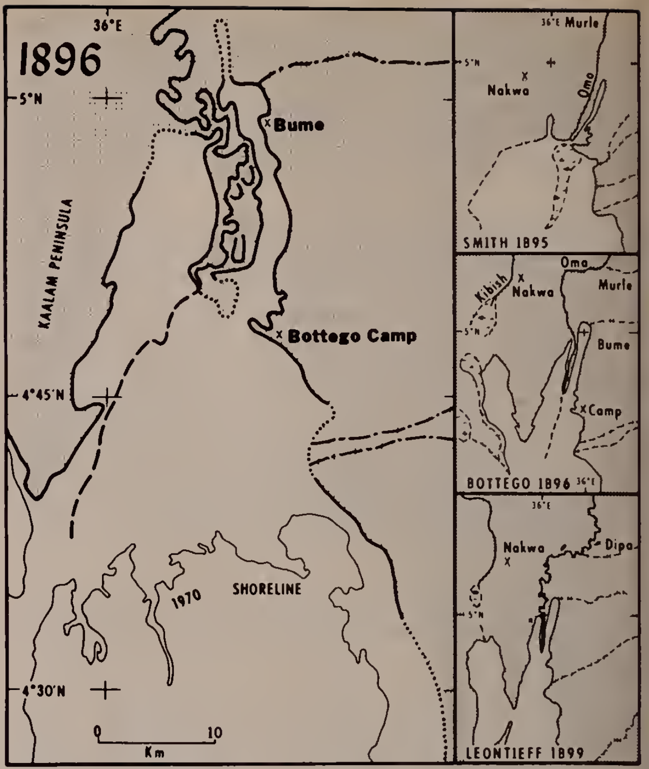


FIGURE 5. The changing shape of the Omo delta and the northern end of Lake Rudolf (reproduced from *Recent History of an Ethiopian Delta*, Chicago, University of Chicago Department of Geography, 1971 (courtesy of Professor Karl W. Butzer).

Smith crossed the Omo and headed north for a short distance and then west toward the Nile. On his way north into the country of the Mursu (Mursi) he obtained a specimen of a Grant's gazelle that ". . . Mr. Thomas has proved to be new after examining a specimen which I have given your museum (*Gazella grantii brightii*)." Thomas did indeed think that this specimen represented a new subspecies and named it *Gazella Granti smithi*.[30]

Smith found that the Mursu had escaped from the raids of the Ethiopians and that they were in ". . . a most flourishing condition."[27] He shot a couple of hippos for them and they brought him food consisting of sorghum, lentils, beans, maize, and dried tamarinds. He then said that:

> I bought a small tusk or two from them at first to start trade; but when I discovered a long line of ebony-like forms bearing about a ton of ivory upon their shoulders to my camp, I had to cry a halt, as it was impossible for me to transport more ivory than I then had with me.[27]

On January 3, 1900, Smith left the Omo and started west for the Nile, which was slightly more than 300 miles away. The rains began then and as Smith says, ". . . followed us all the way to our camp of January 13. They were very scanty, but it was curious that they would progress so from east to west."[27] On January 21, 1900, he came to a clear flowing brook shaded by cedar trees, where he took time out for a swim.[27] One of the photographs published in his article in the *Geographical Journal* describing the journey shows a Gurkha "... following my example in having a delicious cool plunge." [27] Smith retained a lifelong interest in swimming and while in general practice in Roulette, Pennsylvania, many years later frequently swam in the local streams and ponds. Shortly afterwards, he "... crossed at right angles the line of march of the late Captain Wellby." [27] He entered the country of the Margois people, who he discovered were "cow worshippers." [27] It was in this region that he came across Ruppel's reedbuck and obtained a complete specimen for the British Museum which up to that time only possessed a skull." [27] Shortly thereafter he shot a male and female spotted bushbuck, *Tragelaphus bor*, Heuglin, which were then the only complete specimens ever to be obtained, even though the antelope had been described previously.[27]

In late February 1900 Smith entered the country around Tarangole, the northernmost point reached by Macdonald, who had headed the British Juba Expedition in November 1898 (see part IV). He says of this area: "... I was near Tarangole, the most northerly point of Colonel Macdonald's expedition, where friendly arrangements had been entered into between the British Government and the native chief...." [27]

Smith met with this chief, named Amara.

> I received him with all the honours that I could command with my insignificant though trained escort ... His curiousity as to how I had come was too great, however, for him to wait until I called on him. It took him a long time to grasp the fact that we had not wandered either from Uganda or Egypt; ... In return for the many presents which I made him, he gave me much durrha flour, honey, and ground nuts, but it was not until I had bidden him accept a large leopardine blanket that covered my bed that his heart really warmed to me. I am sure that he has never owned anything which pleased him more than this rug, which resembles the skin of some marvellous animal species of the cat family.[27]

Amara wanted to make Smith a gift of a large elephant tusk. The man he sent for it returned by mistake with two small cow elephant tusks.

> Amara was so enraged that, picking up one of the latter [tusk], he began beating the wretched messenger in a terrible manner. The smiling chief had suddenly turned into a ferocious brute that when I endeavored to prevent his killing his subject he at first turned upon me a pair of eyes so full of passion that I feared he might deal his next blow at me ... the unconscious body of his victim was almost hurled out of camp, and others sent to bring the proper tusk, which did not arrive until two o'clock in the morning.[27]

Smith learned from Amara that "... there was an Englishman stationed on the east bank of the Nile considerably south of Lado." [27] Apparently he was unaware that the British had penetrated so far south along the Nile. He says: "... so I abandoned my intention of going to the Belgians." [27]

Smith reached Fort Berkeley, a British post on the Nile, on March 14, 1900, seven and a half months after he had left Berbera in Somaliland. He had filled in a number of gaps on the map of Africa and had achieved his goal of being the first to cross west from Lake Rudolf to the Nile. There he met Captain William K. Dugmore of the Uganda Rifles, with whom he remained for seven weeks.[27] Deep in the interior of Africa, his next problem was getting his men, equipment, and specimens out through the best route possible.

Smith learned that the British had begun cutting a navigable route through the Sudd, the vast area of floating vegetable matter that covers a large area of the Nile.

> I kept my men for nearly a month at the post, hoping that a steamer would come up the river and take us out *via* Omdurman. But on April 13, I found it necessary to send Mr. Frazer with all my men to Mombasa, except my cook and my bright Gurka orderly, Hasap Singh. Famine was reigning throughout the upper Nile districts, and had it not been for the great kindness of the Belgians in giving me grain, I could not have kept my men at all. I was obliged to remain longer myself, since I could get no transport for my collections and kit; and I had just made arrangements with the Belgians to go down the Congo, a journey of four months in the rains, to the west coast, when to my joy, Major Peake turned up in a gunboat, after having cut through the "sudd," and most kindly took me away with him on May 5, on his homeward journey of 1100 miles to Omdurman.[27]

The Belgians had established themselves along the Nile through a treaty with great Britain and the latter now faced the challenge of removing them from there, which they eventually did. Frazer and the bulk of the expedition travelled through Uganda and what is now Kenya, eventually reaching Mombasa. Smith reached Cairo in early June, from which he travelled to London. He had mapped over 500 miles of previously unexplored territory and had brought back several hundred different species of birds, mammalia, plants, reptilia, batrachia, fishes, and butterflies.[27]

SMITH COMMUNICATES HIS FINDINGS TO THE ROYAL GEOGRAPHICAL SOCIETY

A week after arriving at Fort Berkeley on the Nile, Smith wrote to J. Scott Keltie at the Royal Geographical Society, not only outlining the accomplishments of his expedition, but also detailing the problems he had had with Colonel Sadler in Somaliland.[29] He said:

> Just a line to let you know that I have at last reached the Nile after following along 5°30′ north, for the most part between the Omo (I suppose the river north of Rudolf is the Omo now?) and this place ... I met with much obstruction at the start which caused me to fit out really three separate expeditions ... I had no headman—only a pack of vagabonds with me (I knew they were such before we started) but here I am with maps and very good collections all safe so far. Of course this is a long way from the coast but there are posts at intervals and my boxes are fairly safe. My transport animals have broken down so that I cannot take out my boxes at present. So I propose to send Fraser [Smith spells the name as both Fraser and Frazer in his various publications and correspondence] with my men empty handed to Mombasa while I go west with my stuff via Congo under Belgian auspices ....[29]

This statement about travelling under Belgian auspices was sure to irritate the British, who in 1900 were moving toward a showdown with King Leopold II of the Belgians over the latter's claims to territory in the Bahr el Ghazel area that borders on the upper Nile.[31(pp14-15)] Smith's subsequent comments were also intended to give the Royal Geographical Society and the British their comeuppance.

> I am sorry that President Sir Clements Markham [of the Royal Geographical Society] expressed the wish that it should be done by an Englishman. I do not know whether the President will forgive me or not but I shall probably find this out before I leave Antwerp for America."[29]

This was a loaded statement that contained a not so veiled threat. Smith was now the conquering hero, scornful of whatever Markham and the British had to say. Moreover, he wasn't planning to visit England, candidly saying he would sail from Antwerp to America. He intended this as more than a snub for the Royal Geographical Society. There were serious political and military dimensions as well. He was now in the possession of unique maps of British-claimed territories and detailed information about them that could be placed in the wrong hands as the British saw it. And Smith gives a not so subtle hint that he was considering doing just that.

> . . . I am undecided to whom I shall give my maps. The Germans so far have treated me with the most honor in doing justice to my maps. They like just what I can hand over to them, viz everything triangulated and checked and re-checked. You must acknowledge that the Germans have treated my maps far better than the R.G.S. and also that subsequent to an ephemeral puff after reading a paper, I found no one to stand up for me when I was snubbed on the occasion of Cavendish's paper [see part II]. If the latter were only the affair of a night so must the former have been. . .[29]

Smith had apparently been deeply hurt by Cavendish's allegations that he had badly treated the Boran (see part II). Moreover, he was angry that no one at the Society had stood up for him. This is not surprising given that they only had his side of the story and Cavendish's. In addition, the latter was the first British explorer to reach the lake and his fellow countrymen were not about to publicly denounce him and side with Smith, who was an American.

In his letter of March 21, 1900, from Fort Berkeley, Smith asked Keltie for a quid pro quo. In essence, if the Society wanted his maps and the other fruits of his expedition they were going to have to pay for them.

> I will make a business proposition to the Council of the R.G.S. If they deposit £2,000 I will promise them that the money shall only be used in future exploration by me and that I will spend an equal sum of my own and that I will start on such expeditions within a certain fixed period etc. . . the R.G.S. can spare the money from Antarctic regions. . . I want something more than ephemeral kudos from the R.G.S. Patriotism and gratitude combined call me to America and gratitude to Germany and to Italy where they have made me an honorary member of their society [Geographical Society]. The latter part of this letter is private but you will understand my feelings and as you have acted as my friend before I hope for some friendly advice from you."[29]

Given the contents of the letter it is clear that Smith was not averse to Keltie's sharing all of it with the leadership of the Society. He had after all achieved his goal of exploring the lands between Lake Rudolf and the Nile in the highly professional manner respected by all of the European geographical societies. He had done this despite incredible obstacles placed in his path by the British government and local British officials such as Sadler in Somaliland. He had what the British now wanted and was clearly in a position to dictate terms. They had no desire to see his maps fall into either German, Belgian, French, or Italian hands for a number of reasons, among which was the obvious embarrassment that would have ensued if British-claimed territory were laid out in map form for the first time in the publication of a rival European colonial power.

SMITH RETURNS TO LONDON

The Royal Geographical Society promptly published a brief account of Smith's expedition in the *Geographical Journal* soon after receiving it from Smith:

> Dr. Donaldson Smith sends a short account of his recent journey, of which the following is an outline. . . The survey work, carried out in part by Mr. C. Fraser, gave results coinciding satisfactorily with those obtained on the former journey. . . The results of the expedition include, in addition to the surveys, extensive zoological, botanical, and ethnological collections. The fauna changed almost completely after the Omo had been passed, and the species obtained, especially of birds, probably include many new to science. . .[32]

What is significant in this brief account is the statement that part of the mapping was done by Smith's taxidermist-assistant, Fraser, who was a British subject. True, Fraser's work was for hire and the survey results were Smith's private property in a sense. However, Fraser's participation now provided the Royal Geographical Society with some degree of face saving even if Smith opted to publish his maps in another country.

The brief notice in the *Geographical Journal* also notes that Fraser and most of Smith's men exited through British territory and that Smith himself travelled down the Nile on Major Peake's steamer.[32] Peake's serendipitous arrival at Fort Berkeley probably altered what otherwise might have been a different course of events. Smith arrived in London from Cairo but by June he was still nurturing ill feelings towards the Royal Geographical Society. In a letter written on June 24, 1900, to J. Scott Keltie he says the following:

> The very cordial tone of your letter gives me much pleasure. It relieves my feelings to learn that the President has expressed an interest in my work and a friendly feeling towards myself. I am trying my best to persuade myself that Sir Clements Markham will undertake to see that my work will be given full justice. As far as the events of the past years are concerned, I could drop them from my mind were it not that I am unable to explain the recent treatment of my telegram and the way Wellby's journey was handled; I mean the raison d'etre of its title. It is hard for me not to think that it was very unkind not to publish the telegram immediately and broadcast. I took the opportunity to let Mrs. Fraser know that her husband was well by inserting "Fraser well." I thought she would see this in the papers and that likewise my friends would learn of my arrival.
>
> I also said that I had explored "along about 5° 30′ north" which showed that I had taken the course which would most completely cover new ground between Lake Rudolf and the Nile. Nearly a month after this I am astonished that you have given Wellby the honor of having explored the country "between Lake Rudolf and the Nile." Until Wellby got well north he must have been continually in touch with the work of the Macdonald Expedition. So that the unknown part which I explored along its long axis had only a line run across its shortest width and that not near the centre either by Wellby. . . *Other things being equal* I shall not resent the die being cast by the President in favor of an Englishman—it would be only natural. . . I am exceedingly sorry that I cannot accept your kind invitation to dine with you tonight

> at the Geographical Club. I have had so much fever that I am afraid to go out at night just yet. . .[33]

Smith was obviously upset over the title of Wellby's article, which implied that he had explored all of the country between Lake Rudolf and the Nile Valley.[3] Wellby had in fact traversed this area but in a south-to-north direction. His survey work and maps did not compare to those Smith had made on his east-to-west march over the same tract of territory.

On November 12, 1900, Smith read his paper before the Royal Geographical Society, which was published soon thereafter in the *Geographical Journal*.[27] This same article was later reprinted in the *Alumni Register* of the University of Pennsylvania, and in the *Bulletin of the Geographical Society of Philadelphia*.[34,35] Smith's presentation was well received and after it comments in praise of his accomplishments were made by Major R. G. T. Bright, who had accompanied Austin to Lake Rudolf. He paid Smith a great compliment when he said: ". . . I am inclined to agree with Dr. Donaldson Smith in his surmise that at one time the Nile, the Sobat River, and Lake Rudolf were one vast inland sea." [27] This surmise would later be proven accurate by subsequent geographers.

Dr Bowdler Sharpe was generous in his praise for Smith's natural history collections and later published a full description of the birds collected.[36] This was accompanied by a full-page color plate of two species of birds, drawn by J. G. Keulmans. One of the species of birds depicted in the color plate was new to science when Smith collected it on his 1894–1895 trip and was named *Plocerpasser donaldsoni* after him. A total of 135 species of birds were collected by Smith and described by Sharpe.[36] While most of the species were known to science, Smith helped in mapping their distribution in the areas he travelled through. Sharpe said:

> . . .on reaching the Omo River he found a great change in the aspect of the fauna. This is confirmed by the species of birds which he obtained on the marches between the Mursu country and the Nile. A West African element. . . was then traceable farther eastward to the Omo River. . . A certain Abyssinian element is also present among the birds collected between the Omo and the Nile. . .a number of Somali-land and East African forms extend beyond the Omo River district, with a slight mixture of Uganda species. . .[36]

Oldfield Thomas carefully examined the 23 specimens of mammals that Smith brought back and which were presented to the British Museum.[30] He noted that ". . .the specimens were prepared by Mr. W. Carlile Fraser who accompanied Dr. Smith as a collector and taxidermist. . . ." [30] Thomas named a subspecies of dik-dik which Smith brought back after him, *Madoqua guentheri smithii*. A subspecies of Grant's gazelle was also named after Smith, *Gazella granti smithi*.[30]

Sir Clements Markham, the president of the Royal Geographical Society, presided over the session at which Smith made his presentation. He had many laudatory comments to make about Smith's journey:

> . . . I was particularly struck by the remarks . . . he made respecting the meteorology of the country. . .we get very little idea of the important geographical work he has done from simply listening to his paper, even when taken in conjunction with the paper he read to us a few years ago. He has not only explored a new region, but he has done so with great care; he has made a most valuable series of surveys and triangulations. He produced on his first journey a number of extremely valuable maps, and will, I have no doubt, do the same with regard to his latest journey. He has also made, as Dr. Bowdler Sharpe told us, a most important collection of specimens of mammals and birds, as well of batrachians and other lower forms. All this, I think, places Dr. Donaldson Smith in a very high position as an explorer of unknown countries, and I think that he deserves the highest praise that can be bestowed on him by geographers. I now propose a very cordial vote of thanks to Dr. Donaldson Smith, which I feel sure will be carried unanimously.[27]

Smith could not have hoped for any greater praise than this which would have erased to some degree the ill feeling he nurtured toward Sir Clements Markham and the Society. The Society, however, never gave Smith the £2,000 he asked for. Many years later, when he was in private practice in Roulette, Pennsylvania, he often spoke of their not having done so, despite his having given them his maps.[37] His paper was promptly published by the Society and Keltie provided every possible assistance in facilitating this. In an undated letter written by Smith to Keltie from the Craven Hotel he says of his preparation of the manuscripts:

> . . . I had a typewriter to a late hour last night, running it over. This morning I must see Mr. Ravenstein at his house, and Monday morning I have an engagement at Kew Gardens with Dr. Gunther about fishes.[38]

In another undated letter to Keltie he wrote:

> I have looked through the proof very hastily and approve it, but I beg to see the proof of the maps as I wish to supervise them closely. . . I beg you to excuse this very hasty letter as it is after my last London dinner and London dinners are very serious matters. I hope to see you in America before I leave for wild parts. My address will be the Rittenhouse Club, 1811 Walnut Street, Philadelphia. . . .

On January 3, 1901, Smith presented the results of his expedition before the Geographical Society of Philadelphia and on that occasion Amos Bonsall, president of the Society, presented him with the newly created Elisha Kent Kane Medal,[34] named after the famous American physician-explorer known for his expeditions to the Arctic.[39] Smith later published a popular account of his journey in the *Independent* magazine.[40] The only other popular magazine account of him appeared earlier in *Critic* in 1897.[41]

In the spring of 1901 Smith was back in London where on May 20, Sir Clements Markham of the Royal Geographical Society presented him with one of the Society's medals. Another medal was presented at the same session to His Royal Highness, the Duke of Abruzzi, for his exploratory work in the Arctic. The Italian ambassador accepted the medal on behalf of the duke. In presenting the medal to Smith, Sir Clements Markham said:

> The Council of the Royal Geographical Society had no hesitation in awarding you one of the Royal Medals, on account of your now memorable journeys on two occasions across unknown parts of Africa. Your first journey was undertaken, as every one knows, under very great difficulties indeed; then you found your way to

> the northern side of Lake Rudolf, and explored the lower course of the Omo, and returned by the east side of the Lake and by a previously unknown route. That was a very important journey indeed. But, in my opinion, it is almost eclipsed by your second journey, although perhaps not more difficult, still far more striking and important, when you made your way from the coast to the upper Nile over an entirely unknown country. In my first address to this Society I mentioned three great blocks on the continent of Africa which were entirely unknown, and that which you have traversed and discovered is one of them, therefore, personally, I must offer you my very warmest congratulations. But the great utility of your journey is this: you have not, like an ordinary explorer, made a common route survey, but you have made a scientific survey, a triangulation frequently checked by astronomical observations with theodolite and chronometer, and it is that admirable work which you have done under considerable difficulties which has influenced the Council in deciding that you have well deserved the medal which is now awarded to you. Mr. Choate, his Excellency the American Ambassador, had promised to receive your medal, but the Council has been most gratified that you should have considered it yourself of such importance that you should have made a journey from the United States on purpose to receive it. I have much pleasure in placing the medal in your hands, and I may mention that this will be the last time the effigy of our late sovereign Queen Victoria will appear on the Patron's Medal.[42]

After receiving the medal, Smith said a few words:

> Sir Clements Markham and Fellows of the Royal Geographical Society, I wish to thank you exceedingly for this, and to express my appreciation of the very high honour the Geographical Society has conferred upon me. After labours and hardships, it is a great pleasure and satisfaction that such a very great authority as the Royal Geographical Society should confer this distinction upon me. As Sir Clements Markham has said, it will appeal to me very much, as I know it will to many people in the United States, to know that this will be the last time the face of Queen Victoria will appear on the medal. It is a special gratification to me that I have received this from the hands of so distinguished an explorer and geographer as Sir Clements Markham.[42]

The Patron's Medal was a coveted prize and in receiving it Smith entered the ranks of the great and revered in 19th century exploration. None who were present at the meeting could have surmised that Smith's career as an explorer had in effect come to an end. The Society's medalists had frequently gone on to even greater achievements of exploration. However, Smith would never return to the regions of Africa he knew so well nor lead expeditions into other unknown areas of the world. Many would follow in his footsteps to Lake Rudolf and the Omo delta, enlarging on the discoveries he had made; his life, though, would follow another path. Smith arrived back in the United States on June 8, 1901, according to the 1901 *Social Register* (p 298).

THE HARRISON-WHITEHOUSE EXPEDITION TO LAKE RUDOLF

In November 1899, three months after Smith had left Berbera on his journey to the lake and the Nile, a British expedition under James J. Harrison arrived in Aden. Its purpose was to ". . . carry a complete survey through the Hawash Valley to Lake Rudolf, and if possible across to the Nile, at the same time combining a sporting trip in which attention was to be directed to the beasts and birds of the countries visited." [43] The party consisted of Archibald E. Butter; P. G. Powell-Cotton; W. F. Whitehouse; a surveyor, Donald Clarke; and a taxidermist, R. Perks.[43] Powell-Cotton went with the group as far as Addis Ababa and then left the party for a sporting trip to other areas of Ethiopia.[24] Harrison's party left Addis Ababa on January 18, 1900, heading for Lake Rudolf and the Nile. Smith had left the Omo two weeks before, on January 3, 1900, and was marching through unexplored territory toward the Nile. Harrison and his party arrived at the northern end of Lake Rudolf in March 1900, where ". . . we hoisted the English flag; doing so at the request of Colonel Harrington and with the acquiescence of the emperor, to denote the Abyssinian and English boundary lines . . . Needless to say as the flag went up the whole party sang 'God save the Queen,' a bottle of champagne having been brought along in which to drink her Majesty's health." [43]

Harrison was replacing the flag taken down by Leontiev the previous August when he led an expedition to the area.[21] He abandoned the idea of going over to the Nile "owing to want of water and the uninhabited nature of the country." [44] At the time he made this decision, in March 1900, Smith was moving close to the Nile a few hundred miles away. Harrison probably miscalculated the true nature of the country to the west of the lake, thinking it an uninhabited wasteland devoid of water. Wasteland or not, Smith had, under the same drought conditions, forged ahead for the Nile, finding to his surprise that the area was fairly well watered and inhabited. Harrison and his party moved down the eastern side of the lake, passing through Nyemps and reaching the railhead on May 13, 1900, a date which found Smith making his way down the Nile.[27]

Harrison was a wealthy sportsman whose journey enabled the British to reassert the general northern limits of their territorial claims in the Lake Rudolf region. Harrison was also a soldier, having served in the Prince of Wales Yorkshire Hussars, from which he retired with the rank of Lieutenant Colonel in 1905.[45] He was an active sportsman who lived at Brandesburton Hall in Yorkshire where he kept a large collection of trophies which he had collected all over the world. He died on March 12, 1923.[45]

AUSTIN'S SECOND EXPEDITION TO LAKE RUDOLF: THE ABYSSINIAN FRONTIERS EXPEDITION

In October 1900, Major Herbert H. Austin, who had previously made a trip to Lake Rudolf as part of the Juba Expedition in 1898, was given the command of a survey expedition in the southeastern Sudan along the border with Ethiopia (Fig 6). His second in command was Major R. G. T. Bright of the Rifle Brigade, who had been with him in Africa before and who attended Smith's presentation before the Royal Geographical Society on November 12, 1900. Austin and Bright moved up the Nile and reached Fashoda on January 11, 1901.[46] They then moved on to the Sobat River and into its headwaters, reaching the Akobo river on February 27, 1901. In early March they entered the region of the Boma Plateau and by April 4 were several miles north of Mount Nakua, not far from Lake Rudolf.[46] By this time their food supply was running extremely low and the local crops being cultivated along the Omo River Valley were still green and would not be

FIGURE 6. Major Herbert H. Austin (reproduced from *Among Swamps and Giants in Equatorial Africa*, London, C. Arthur Pearson, 1902).

ready for harvesting for two months. Because of the critical shortage of food Austin decided to travel down the western side of Lake Rudolf, "...living as best we could on our transport animals...."[46]

Austin's march down the western shores of the lake proved to be a calamity. He said of it:

> ...I frankly admit that I anticipated a loss perhaps of 25 percent of our men and about 50 percent of the animals in the undertaking; but in my most pessimistic mood I never imagined the dreadful loss of 75 percent of our personnel, which we actually suffered, nor the terrible experiences we were to undergo before reaching safety....[46]

For almost two months, as they made their way south, the expedition subsisted almost entirely on donkeys, and "...out of the fifty-nine Sudanese only fourteen survived."[47] Austin lost 45 of his 59 men. Most of them died from starvation, but others were killed in ambushes by the Turkana. Austin himself came down with scurvy. "...I noticed an extraordinary...number of purple spots all over my legs...a few days later, hemorrhage of my mucous membranes set in, and I became in course of time so seriously ill with scurvy that I almost succumbed to this disease."[46]

By early August 1901 they arrived at Lake Baringo and eventually found relief when they met Mr Hyde-Baker, the collector of the Baringo District. With proper nutrition the surviving members of the expedition rapidly recovered. Austin and Bright made their way to the railway which they reached at Lake Nakuru on August 14, 1901. They travelled to Mombasa from which they sailed on September 8, arriving in London on October 15, 1901, a year after Austin had been given responsibility for surveying the Sudan-Ethiopian borderlands.[46] Austin later published a book recounting his experiences during this expedition.[48] Butzer noted that, "...The years 1898–1900 saw an unprecedented drought across the whole of East Africa to the Nile...and the Nile volume for 1899 was the lowest since the beginning of records in 1871...."[15(p115)] Thus the paucity of food supplies was due to something more than Ethiopian raids around the course of the Omo River. The explorers who travelled through this region during those years, including Smith, all noted the paucity of water, the falling level of Lake Rudolf, and the low crop yields.

Austin's journey, despite its calamitous course, did achieve a great deal, as von Hohnel notes: "...His surveys were checked by astronomical observations and were based on positions exactly determined by triangulation."[5] His surveys and those that would follow had as their purpose the eventual establishment of the boundaries between Ethiopia and the Sudan and the British East Africa Protectorate, later known as Kenya.

COUNT WICKENBURG'S EXPEDITION TO LAKE RUDOLF

As Austin was struggling down the western shores of Lake Rudolf, the German explorer Count Wickenburg was arriving at the northern end of Lake Rudolf, having come down from Ethiopia. His purpose was to explore the area between Lake Rudolf and the Lorian Swamp, country which had been traversed in part by Smith and Chanler (see parts II and III). Wickenburg had previously led expeditions through Somaliland and was no stranger to this part of Africa. On July 27, 1901, he left the northern end of Lake Rudolf and marched toward Mount Marsabit, which up to that time had been seen only by Smith and Delamere. Wickenburg found that Lake Stefanie was drying up and that the waters were murky.[49–51] He also observed that the lake shore was full of the skeletons of numerous crocodiles.[49] These observations confirm the drought that others such as Smith had documented. Trading caravans were now regularly visiting the Lake Rudolf area from the coast, for he met one that had come up from Kismayu via El Wak to the Huri Hills and Lake Rudolf.[49] Wickenburg's account, which he published in *Petermanns Mitteilungen*, is not chronological, making it difficult to follow his exact itinerary. He visited Marsabit and provided a superb description of the crater lake in the Gof Sokorte Guda crater that Smith had discovered in 1895. Wickenburg says the following of this crater lake which the American wildlife photographers, Martin and Osa Johnson, named Lake Paradise in the early 1920s:

> The third and most southern crater is almost perfectly round with a diameter of 1,000 meters and a depth of 150 meters. At its bottom is a pond surrounded by green reeds, the walls are covered by trees and shrubs and thick forest grows up to the rim and the surrounding area is covered densely with vegetation. On Marsabit one frequently finds springs with good drinking water. Marsabit is the name the Laikop [Samburu] call the range (or chain). The Oromo who themselves never enter this range call them Haldaja. The Rendille call them Saku. Toward the south

> east Marsabit slowly descends. (It slopes towards the south-east and becomes a plain densely covered with thorn bush up to the Lorian Swamp). At the north end a last chain of hills continues to the north east. It is called Dalota. Toward the west begins the already mentioned volcanic plain which reaches up to Kulal and the Njiro Mountain.[49]

As Smith had done in 1895, Wickenburg travelled down to Laisamis and then to the Guaso Nyiro. From there he went to the Lorian Swamp which was almost completely dry and then moved on to the Tana River. He arrived on the coast at Lamu as had Smith in 1895.[49]

THE EXPEDITION OF THE VISCOUNT ROBERT DU BOURG DE BOZAS

On April 2, 1901, a French nobleman, the Viscount Robert du Bourg de Bozas, led an expedition out of Djibouti, headed for Addis Ababa, which was reached on March 2, 1902, after a circuitous itinerary around the Ethiopian highlands. The expedition then made for Lake Rudolf, the northern end of which was reached on June 22, 1902.[52(p307)] De Bozas then moved on to the Nile, following a more southerly route than that taken by Smith in 1900. He arrived on the Nile at Nimule on September 9, 1902.[52] De Bozas was the second explorer after Smith to travel east to west from the lake to the Nile. Moving westward, he entered the headwaters of the Uele River where he contracted tetanus.[52(pp410-411)] He died at Amadi on the Uele which is now in northeastern Zaire on December 24, 1902.[52(p413)] His remains were exhumed and returned to France in 1905.[53(pp55-75)] The de Bozas mission accomplished a great deal from the scientific point of view. Of significance is the fact that he was the first to note the highly fossiliferous nature of the region around Lake Rudolf and the Omo River Valley. These findings pointed the way to future discoveries in the area which have since shed much light on ancient hominids.

SURVEYORS AND TRADERS

The British were anxious to carefully survey the frontier between their East African territories and Ethiopia in order to establish a border through treaty with Emperor Menelik. With this in mind a surveying party was sent into the Lake Rudolf region in late 1902 for the purpose of surveying the frontier. It was led by Archibald Butter, who had previously travelled through the area with the Harrison-Whitehouse expedition in 1900, and included a surveyor, Philip Maud. They left Addis Ababa on November 6, 1902, and reached the northern end of the lake on May 24, 1903.[54] Maud triangulated most of the border area between Ethiopia and the British East Africa Protectorate.[54] He also seems to have shot every quadriped in sight and as Turton observes, "... turned to an orgy of big game hunting." [4(p13)] Maud's survey work constituted the grounds on which the British negotiated a border treaty with the Ethiopians in 1907. Although the precise border was not defined, it was agreed to in very general terms.[55(p30)] The precise delimitation of the border was left to another army surveyor, Major C. W. Gwynn. Meanwhile, before Gwynn got into the region, another group arrived from the south, headed by J. W. Brooke of the East Africa Syndicate. Brooke travelled up from the Mount Elgon area through the country to the west of the lake beginning in August 1903. After reaching the northern end of the lake, Brooke travelled up the course of the Kibish to the northwest of the lake, and making a loop to the west and south returned into what is now Uganda. This journey added virtually nothing new to what was already known about the region.[56] Its purpose was to assess the natural resources of the region with an eye toward the development of trade and commerce. Brooke concluded that the principal wealth of the area was ivory.[56]

The 1907 Anglo-Abyssinian treaty concerning the frontier between Ethiopia and the British East Africa Protectorate called for the establishment of a delimitation commission. In May 1908, Major C. W. Gwynn of the Royal Engineers was asked to carry out the survey work along the border. Gwynn organized a detailed expedition that covered every stretch of country between the two territories. In 1906 the British had established a post at Moyale on the frontier that was under the charge of Mr P. Zaphiro, a Greek national in their service. Zaphiro accompanied Gwynn on part of the survey. Gwynn reached Lake Rudolf on February 22, 1909, where he found an Abyssinian post just opposite the mouth of the Omo.[57] Gwynn's survey was detailed and his observations added much to what was already known about the border regions. He provided exceptionally good information about the northern end of the lake and the Omo delta which is conspicuously absent in Maud's survey.[15(p115)] Maud's survey had resulted in what came to be known as the "Red Line," the approximate border between the two territories. Gwynn's survey produced a line to the north of this known as the "Blue Line." The Ethiopians naturally held to the "Red Line" which extended their territory further south and the British to the "Blue Line" which pushed their territory further north. The combination of a disputed border and a nomadic population moving through the area plus the lure of ivory on the British side created fertile grounds for disputes between the two sides for many years to come. In the end the Ethiopians got the better part of the deal for as John Boyes, an early trader and buccaneer in Kenya, observed when he crossed the border in 1906, "... On the British side of the boundary the country is low-lying arid waste covered with thick scrub. It appeared that the Abyssinians had got everything that was worth having in that part of the country—all the land of any value being on their side of the frontier."[58(p41)]

Boyes, who was bringing down Ethiopian ponies and ivory into the British East Africa Protectorate, paid a visit to Marsabit and said of the crater lake that Smith had discovered:

> ... In the afternoon we made our way to the summit of the mountain to have a look at the Crater Lake... This mountain has recently been made famous by well-known American filmmakers, Mr. and Mrs. Martin Johnson, who have made it their headquarters for some years and have lectured on its mysteries in various parts of the world. The Crater Lake they have renamed Lake Paradise, which is quite a suitable title.[58(p50)]

Boyes was not the only trader up on the border areas. His chief competition was the Boma Trading Company which had been set up by Jack Riddell, F. Ward, and W. G. Sewell. Sewell was a wealthy American from Boston who had come out to hunt and who had stayed on and developed land. As Errol Trzebinski notes, they were gentlemen buccaneers, dealing in ivory, beeswax, ponies, donkeys, and cattle.[59(p123)] Elspeth Huxley characterized the Boma Trading Company as having "an Elizabethan air." [60(v1,p244)] It was eventually strangled in its infancy after two years or so of operations by, as Errol Trzebinski says, ". . . a network of quarantine restrictions." [59(p123)] The extension of government administration into this wild frontier land also crippled the free-wheeling style of the buccaneers because with it came strict controls on elephant hunting and access to ivory.

CHAUNCEY H. STIGAND'S EXPEDITION

A final expedition to Lake Rudolf that closes the saga of early journeys to this part of Africa was made by Chauncey H. Stigand, a British military officer who was also a superb naturalist, anthropologist, and historian. A man of superior observational skills and with a talent for communicating what he saw, Stigand added much to our knowledge of the Lake Rudolf region when he set out for Addis Ababa from Nairobi in 1908 via the lake. He was a man of much African experience who had worked as a military officer in Somaliland, Nyasaland, and British East Africa. In order to amass enough money for his Lake Rudolf expedition, he went to the Lado Enclave at the headwaters of the Nile to hunt elephant, the proceeds from the ivory furnishing him with the needed money.[61(intro ppxxiv–xxv)]

Stigand moved up the eastern shore of the lake, into the Omo river region, and then northeast to Addis Ababa. The value of Stigand's journey lies in the details he provides about peoples and places and the regions he traversed.[62] He was also the first to document the presence of tsetse fly along the Omo. He said:

> . . . a new illness had arrived last year in this neighborhood. From the description the natives gave me of it I had no doubt left in my mind that it was sleeping-sickness to which they referred . . . I am afraid that if this news is true, the banks of the Omo River will fast become depopulated . . . I am at a loss to understand how this cursed insect could have reached this neighborhood. . . .[62(p240)]

In 1913, Stigand married an American, Nancy Yulee Neff of Washington, DC, and in 1916 was appointed Governor of the Mongalla Province in the southern Sudan. His daughter, Florida, was born in 1917. In November 1919, the Dinka on the western side of the Nile revolted and Stigand was speared to death in an ambush.[61(intro ppxxxvi–xxxvii)] Stigand was a prolific writer who wrote on a variety of African subjects. Many of his books have become classics in their fields.

MODERN STUDIES OF THE LAKE RUDOLF REGION

In a sense the exploration of the Lake Rudolf region had not come to an end with Stigand's journey. Within a few decades various types of scientists entered the area to study its peoples, history, fauna, geology,[15] and fossils. Social scientists have been especially active in the region and have produced a number of definitive studies of the various peoples living there who Smith and others had first encountered so many years before. Spencer has studied the Samburu and Rendille[63] and Baxter the Boran,[64,65] Turton the Mursi,[66,67] Tornay the Murle,[68] and Nyangatom[69] and Almagor the Dassasnetch.[70] Historians such as Garretson have carefully studied the dynamics of life along the Kenya-Ethiopian frontier from the late 19th century until more recent times, providing a valuable background for understanding the border incursions and ivory poaching that afflicted northern Kenya for many years.[71] A general overview of the peoples of southwestern Ethiopia and specifically the lands around Lake Rudolf has been provided by Ernesta Cerulli.[72] In recent years some of the most exciting scientific findings to come out of the Lake Rudolf region have been in the field of paleontology. Richard Leakey, Director of the National Museum of Kenya, and his colleagues have found a number of fossils, especially along the eastern shores of the lake, that have thrown much light on the early history of hominids (see part I).

RECENT CHANGES

Much has changed in this area of the world which Smith was among the first to explore. Roads, schools, clinics, and small towns now dot the countryside. Two bridges now span the Guaso Nyiro and pastoralists have been settled along the river and on the edge of the Lorian Swamp.[73] Permanent settlement has also been encouraged on Marsabit Mountain, whose northern slopes now support a population of several thousand. A modern, paved road runs almost to Lake Rudolf from the south and similar roads cross southern Ethiopia through which Smith travelled on foot. Small settlements stand on the shores of Lake Rudolf, tourists come to fish at the lake, and Luo fishermen catch and process fish using modern technology. Further to the north modern methods of irrigation are used along the banks of the Omo River. Marsabit in 1987 is a far cry from Smith's time. Although most of the mountain is gazetted as a national reserve, one visitor said,

> . . . You would cry if you saw the state of lovely Marsabit . . . It is still beautiful at Paradise, but steadily going back to bush. The slopes are gradually being covered in corrugated iron roofs and shambas too, and one never sees a kudu now.[74]

Another visitor said:

> . . . I suppose you would be saddened at seeing Marsabit . . . tin roofs abound. The urbanization has occurred on the N.E. slopes of the mountain . . . it is there that it is all built up. They are growing coffee, mangoes, bananas, vegetables under irrigation, all down that area . . . However on the S.W. side there is no evidence of human encroachment into the forest . . . once inside the park there appears to be no change. . . .[75]

Despite the changes the challenge of Lake Rudolf's tough environment remains. Edward Rodwell recalls that:

> In the 1950s I took a small expedition to Lake Rudolf to test out a new Austin car called a Gypsy which was remarkably like a landrover. My instructions were to make or break the machine . . . there is a legend among Europeans that when at Rudolf Europeans go bananas for a time and then the best of friends become angry with one another, possibly because of the excessive heat and wind.[76]

Lake Rudolf still offers new and different challenges. Between November 1977 and January 1978, a young Englishman, Stephen Pern, became the first known person to walk around the lake, starting in the southeast and moving north into Ethiopia where he was detained by the Ethiopian authorities, and then travelling south along the western shore. He accomplished this trip in 50 days and travelled with only African companions, including a child and a pregnant woman who accompanied him for part of the way.[77,78] A more sophisticated expedition headed by Kenyan photographer Mohamed Amin drove around the lake in 1980. This expedition carried 800 liters of gasoline, 20 spare tires, 275 liters of drinking water, a quarter-ton of photographic equipment and film, and a 900-mile-range radio telephone.[79]

SMITH'S LIFE IN THE EARLY 20TH CENTURY

Arthur Donaldson Smith started the 20th century on a high point when he received the Patron's Medal of the Royal Geographical Society in 1901. There were few unknown patches of the globe left to triangulate and his financial resources were not limitless. Between 1901 and 1909, the year President Theodore Roosevelt appointed him to the US Diplomatic and Consular Service, Smith essentially followed the life pursuits of a rapidly vanishing gentry. Although there is some documentation of what he did during these eight years, it is scanty.

The Rittenhouse Club in Philadelphia was a major focal point of his life following his election to it on March 5, 1881.[80] He had been proposed for membership by A. Sydney Biddle and seconded by Judge Craig Biddle, a son of Nicholas Biddle of the Bank of the United States.[80] Smith lived on and off at the club through 1901 and changed his membership category from resident to nonresident on June 14, 1893. Some two weeks later he changed it back again to resident, perhaps because of altered travel plans as suggested by McFadden.[80] On October 14, 1907, he transferred to a nonresident status in which he remained until he resigned on September 12, 1910.[80] According to the records of the Royal Geographical Society, Smith used the address of the Rittenhouse Club for correspondence purposes in 1896 and in 1900–1901.[81] From 1894 to 1895 he used his father's address, 221 South 21st Street, Philadelphia, and from 1897 to 1898, 2227 Locust Street, Philadelphia. From 1902 to 1923, he listed 1820 Chestnut Street, Philadelphia, as his address. Two incidents involving Smith at the Rittenhouse Club, one in 1901 and the other in 1905, provide some insights into a problem that afflicted him for his remaining years. The first occurred on a Sunday evening, December 22, 1901, when he was at the club. Two other members, Gerald Holsman and his cousin, J. C. Mercer Biddle, were sitting in the East Cafe of the club when "...Smith visibly intoxicated, applied...a vile epithet to Holsman...Smith was somewhat incoherent," but apologized.[82] Holsman accepted the apology but "...at this Smith became incensed and said, 'I did not apologize, and you are a God-Damned liar...'" Holsman replied that Smith was "using Club rules to facilitate using language he would not use anywhere else."[82] At this Smith jumped up and "...struck Holsman."[82] He was restrained by others but "...broke loose and struck him again." Upon further restraint, "his fury abated." However, he remained in the club for some time "exhausting his vocabulary of vile epithets."[82]

Smith sent the club's House Committee a letter and statement giving his version of the events. In his letter he said "...while I cannot accept Mr. Holsman's version of the affair, I think it better under the circumstances to present the Board the facts as I recall them without criticism and comment on his statements."[82]

Smith's version of what happened was quite different from Holsman's. He said in his written statement to the board:

> The Messrs. Biddle, Holsman, and Smith were sitting in front of the fire in the East Cafe in general conversation and rounds of drinks were had. They were discussing the foolishness of taking offence at unjustified expressions which, if they were friends, should not be sufficient to cause a breach between them. Smith said, "Holsman, you are a friend of mine, and if I should say to you Damn your dirty little soul, that would not justify you in getting angry with me." To Smith's great surprise Holsman drew himself back very stiffly and in an insulting tone of voice said, "Smith, do you know what I think you are? You are a hound and a coward! Stung, Smith jumped up and struck him. Realizing he had done wrong, he backed away, but Holsman followed with one epithet after another to which Smith replied. The next day, Smith called on Holsman with the intention to apologize, but Holsman declined to see him stating that any apology must be in writing. This he did relating that he did not mean to be insulting and he knew Holsman did not. Nevertheless, he should not have struck him, was very sorry, and humbly apologized to the Board.[82]

The truth of the matter is very hard to arrive at as McFadden states: "...after so many years."[82] Whatever the truth, Smith was suspended from the privileges of the club for 60 days.[82]

In February 1903, Smith, apparently bearing the club no malice, loaned it an Alaskan moose-head "...which was received and acknowledged with great appreciation."[82] This indicates that Smith had gone on a hunting trip to Alaska sometime after his last African trip.

The next incident at the club concerning Smith took place on the evening of June 6, 1905, and involved Dr James Veree Ingham.[82] McFadden provides a detailed account of this incident gleaned from the club's records:

> Dr. Ingham had been reading in the Library of the Club and shortly before midnight repaired to the West Cafe where he was served a Welsh Rabbit. He heard "spasmodic mutterings" from the East Cafe but did not investigate. Preparing to leave the Club, he was overtaken by Smith who was greatly excited and shouting "profane ejaculatory sentences." These were, for the most part, somewhat indecipherable with the peculiar exception of "God damned lobster, God damned lobster everywhere." Gazing wildly at Ingham, Smith flourished his cane, brushed past him and left the Club, waited outside and confronted Ingham shouting, "Now I have you." Alarmed, convinced that he was about to be attacked, and desirous of avoiding an appearance in Police Court that might well result, Ingham returned into the Club, was joined by some other members attracted by the commotion, and was reconfronted by an enraged Dr. Smith who had come back in to the Club. Finding he could provoke no reply to his curses, Smith rushed at Ingham with uplifted cane shouting, take that damned segar out of your mouth. Expecting and preparing for a blow from Smith's cane, Ingham was not prepared for a blow from Smith's left hand to his face (known in current parlance as a "sucker punch"), but he was able to parry the following attack by the cane with his umbrella. Smith, restrained by other members (the Club was certainly alive in the wee hours) continued to curse Dr. Ingham calling him a coward and worse.

> There was no doubt to this one and Smith to his credit, in his letter to the Board, did not even attempt to offer an excuse. He said that he deplored the incident as one due solely to his own fault at his condition and with absolutely no personal animosity to Dr. Ingham. This time he was suspended for one year, and it may be a measure of kindness from either Dr. Ingham or the Board that he was not expelled for this offence. Serving out his suspension, he remained a member until 1910 before resigning his membership. . . .[82]

McFadden makes a cogent observation about Smith when he says, ". . . he may have been more at home in the great outdoors so vividly described by Robert Service, but he was. . . reared a gentlemen and obviously felt the call for the companionship which a gentlemen's club so well provides."[82] Smith, who at the time was also a member of the Philadelphia Country Club and the Radnor Hunt Club, eventually sold the moose head to the Rittenhouse Club for $250 in 1910 when he resigned and took up his consular post in Mexico.[82]

Smith may have been prompted to resign from the club for financial reasons as well. Pleasants notes that around that time ". . . he was pretty well strapped financially. . . It was expensive to live at his clubs in Philadelphia. His bankroll was pretty thin."[83] It appears that Smith was primarily living off of his inheritance. No documentary evidence has been found to date that he was practicing medicine and his name does not appear in any of the national medical directories of the day, including the first edition of the *American Medical Directory* published in 1906 by the American Medical Association or the various editions of *Polk's Medical Registry and Directory*.

In 1905 Smith and a group of men who were active or interested in exploration, the natural sciences, and prehistoric archeology formed an organizing committee that led to the establishment of the Explorers Club.[84] A subsequent group followed up on the work of this original organizing committee and are now considered the club's founders. Smith did not remain active with the Explorers Club, although he was seated on the dais at a place of honor at the club's dinner held in 1909 at the Waldorf Astoria.[85]

William Lord Smith, MD, of Massachusetts, one of Smith's old friends who had travelled with him to Norway and Somaliland in 1893, remained an active world traveller during these years. In 1903–1904 he undertook an extended shooting trip from the Aleutian Islands to Japan and from there travelled through China, Manchuria, northern India, Afghanistan, and Persia.[86] It was during this extended journey that he shot a cave tiger at Amoy in China, an event about which he wrote in detail years later.[87] His extended Asian trip made headlines primarily because he successfully treated the Shah of Persia in Tehran.[86,88] In 1909 Smith was in British East Africa collecting specimens for the Agassiz Museum of Harvard. Former president Theodore Roosevelt, who was there at the time on his now legendary hunting and collecting trip, met Smith and said of him:

> . . . as we marched into Meru we encountered the safari of an old friend, William Lord Smith—"Tiger" Smith—who with Messrs. Brooks and Allen, were on a trip which was partly a hunting trip and partly a scientific trip undertaken on behalf of the Cambridge Museum."[89]

On returning to the US William Lord Smith delivered an address on "The Peoples and Wild Animals of British East Africa" in Harvard's Assembly Hall in February 1910.[90] Dr H. Spencer Potter, Arthur Donaldson Smith's grandnephew, recalls that the two Smiths remained close friends throughout their lives.[91] He also recalls that his granduncle and Theodore Roosevelt were close friends. The latter greatly admired Smith for his talents as a sportsman and naturalist.[91]

DIPLOMATIC AND CONSULAR SERVICE

In the spring of 1909, President Roosevelt nominated Arthur Donaldson Smith to the Diplomatic and Consular Service of the United States (Fig 7).[92] Smith was appointed on March 3, 1909, from Pennsylvania as consul in Mersine, Turkey. His salary was $2,500 a year.[93] Roosevelt left office the next day when William Howard Taft became president. Pleasants indicates that by this time Smith was ". . . pretty well strapped financially."[83] His account of Smith, which is highly anecdotal in nature, lacks chronological accuracy and some of the events he recounts do not square with documentary evidence. This is especially true of his description of Smith's appointment to the Diplomatic and Consular Service. He says the following of it:

> He finally came home to Philadelphia. By this time, he was pretty well strapped financially. Not that he was actually poverty-stricken; but you know what two or three scientific expeditions are likely to cost. Something had to be done about it. I have an idea that Father mentioned the matter to his old friend, Mr. Tom Butler, who was then a pretty big man in Congress. Anyway, Uncle Donnie suddenly woke up one morning, and found himself a diplomat in a most important foreign country. It would not be cricket to say which one on account of what happened.

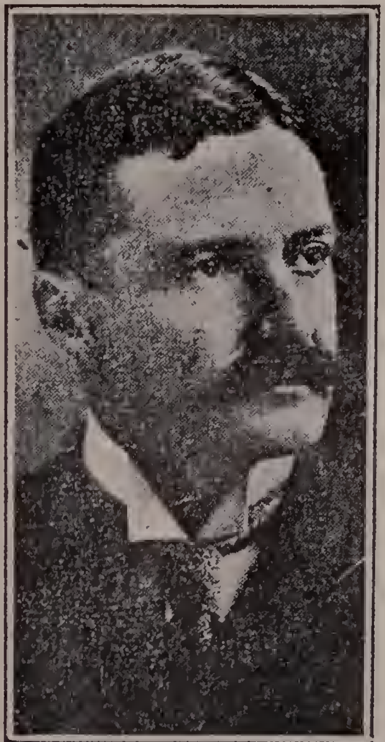


FIGURE 7. Arthur Donaldson Smith, MD, in 1909 at the time of his appointment to the US Diplomatic and Consular Service (reproduced from the *Public Ledger*, April 27, 1909).

> As a diplomat in the jungles of Abyssinia or Somaliland, Uncle Donnie was really tops. As a diplomat in the court of a European monarchy, he was an excellent African explorer. You get the idea. You see, he had been appointed by Teddy Roosevelt, who had admired his sporting qualities. In fact, Teddy was so intrigued by what he had done that he followed almost in his footsteps a few years later.
>
> Well, anyway, Uncle Donnie had barely warmed his diplomatic chair when he was invited to some sort of a formal state banquet. Other diplomats were there. Things were going famously until some minister or consul or charge d'affaires made a nasty remark about the United States. Of course, it was couched in diplomatic terms, and should not have been taken seriously.
>
> If Uncle Donnie had been a good diplomat, he would have diplomatically requested a diplomatic explanation, and would have received a diplomatic reply when the offender had sobered up. However, Uncle Donnie was impulsive, in spite of his small stature. Father never would tell me just what happened; but the papers were full of a pretty good story, whether true or not. They said that the state banquet had been broken up by a really sweet battle at the table in which champagne bottles and fists figured extensively. The last seen of a certain foreign statesman was a streak of flying coattails going up the street towards a swank hotel, with Uncle Donnie in hot pursuit brandishing a bottle of *V. Clicquot* like a war club. As neither diplomat had stopped to kiss the host good bye, the matter had assumed serious proportions diplomatically. Uncle Donnie had insisted to newspaper men that he was only trying to bring the other diplomat back to the party; but that did not explain why he had stopped to throw a full bottle of champagne after him when he found he was being outdistanced. The fact that he had missed, and that a whole quart of perfectly good wine had gone into the street only made matters worse. There came near being international complications over Uncle Donnie. I think that Father managed to get Tom Butler to see T.R. and explain things. Anyway, war was not declared, and Uncle Donnie was not shot at sunrise. Father hinted that T.R. had showed his teeth, pounded the desk and shouted "DEE-lighted!," when he heard about the affair. The only thing was that Uncle Donnie came home by the next boat.[83]

There is no record of Smith's having served as a US consul during President Theodore Roosevelt's administration. Thus the incident described by Pleasants may have taken place in Turkey which was Smith's first post. H. Spencer Potter, PhD, Smith's grandnephew, thought it took place in London.[91] If it did, then it occurred before Smith was in the Diplomatic and Consular Service.

The evidence that the incident took place in Turkey is supported by the fact that Smith was transferred to Patras, Greece, as US consul at a salary of $2,000 a year on May 7, 1909, two months after he had been appointed to Mersine.[94] This time he was appointed from North Carolina, a state to which he regularly went for quail shooting.[94]

What is one to make of this information? In the absence of documentary evidence there are several possibilities. Smith may never have gone to take up his post in Mersine, negotiating instead a more agreeable one. He may have gone for a brief period and then requested transfer. If the incident described by Pleasants and confirmed by Potter did take place in Turkey, in all probability it occurred in the capital, Constantinople, and not in Mersine which was a relatively small backwater. If it occurred in Turkey, it could have constituted grounds for Smith's transfer. Finally, there is the possibility it took place in London as Potter seems to recall. If it did then it occurred either before or after Smith was a US consul.

Smith's first post was not the most desirable one. Mersine is a relatively new port city situated on Turkey's southern coast at the extreme western end of the Cilician plain. In 1974 it had a population of only 60,000 and in Smith's day was much smaller.[95] Patras on the other hand is the third largest city in Greece and the largest in the Peloponnese. In 1981 it had a population of 112,000.[96] As the gateway to western Greece this port city had significant commercial importance when Smith was there. The $500 difference in salary between the posts in Mersine and Patras was probably due to the fact that the former was considered a hardship post for which there was an additional stipend.

Smith's stay in Patras was also brief. On April 13, 1910, he was named consul in Aguascalientes, Mexico, less than a year after he had been appointed to the consular post in Greece.[97] The US consul position in Aguascalientes carried an annual stipend of $2,000. Smith was in Aguascalientes in 1911 because a Harvard graduate, Bruno Newman, who was a consulting engineer there, wrote to the Harvard Alumni Association to say: "Received Harvard Directory. Thanks! Among addresses wanted I find Enrique Cenesta Gallardo. . . Dr. A. Donaldson Smith is Consul at Aguascalientes and not in Greece."[98]

Smith arrived in Mexico in 1910 just as the Mexican Revolution was beginning with Francisco Madero's revolt that eventually toppled Porfirio Diaz, who had ruled as dictator since 1876.[99] Aguascalientes was an important provincial town in the central part of the country. Known for its thermal and mineral waters, it was and still is an important silver mining center. Founded in the 16th century, the town is noted for its pre-Colombian subterranean catacombs.[100] Although a booming business center today, in Smith's time it was a restful and charming colonial town.

1911–1920

There is little documentary evidence as to exactly what Smith was doing during this nine-year period. He was back in Philadelphia from Mexico in 1912, and this seems to be the year he ended his career as a diplomat.[101] His grandnephew H. Spencer Potter recalls his parents as saying that Smith did practice medicine to some extent in Philadelphia.[91] During this time he became active in the Boy Scout movement out of a desire to help boys appreciate the wonders of the natural world around them.[91] Potter recalls seeing a photograph of him in a scout master's uniform.[91]

On December 11, 1914, Smith wrote to Dr E. Gordon, curator of the University of Pennsylvania Museum. He said:

> I have another African collection that I obtained on my last Africa expedition and I should like to add this also to the University Museum. It contains quite a number of interesting objects which are representative of tribes living between the northern end of Lake Rudolf and the Nile. I wish the University Museum to have these along with the collection already purchased by them. I am sure the collection, for ethnological reasons, is more than worth $100, but that is the sum for which I shall be pleased to sell it to the museum. . . There will be no necessity for sending a carpenter—I shall have it packed myself at Kaolin if you wish me to send it to you.[102]

Kaolin was the location of the large summer home of

Smith's sister, Agnes Spencer.[91] Smith spent a great deal of time there and his grandnephew recalls that while there he regularly walked through the fields at a brisk pace, chiding those younger than him for not being able to keep up with him.[91] At the Grange, which was a 5,000-acre farm belonging to his brother-in-law, Graham Spencer, he regularly showed himself to be a superb horseman, even in old age.[91]

When the United States entered World War I, Smith volunteered for the army. He was 61 years old and had to undergo surgical repair of bilateral inguinal hernias before he was accepted.[83,91] He was assigned to Fort Oglethorpe in Georgia where he spent nine months, being discharged with the rank of captain.[83] Pleasants says, ". . . he went back to his country practice. The people of the town were glad to see him again."[83] This is chronologically inaccurate because Smith did not practice in rural northern Pennsylvania until 1921.[103] Pleasants inaccurately places Smith's opening of a general practice in northern Pennsylvania before his enlistment in the army when in fact it took place three years after his discharge.

Despite the errors in chronology, Pleasants provides some background as to why Smith decided to open an office for the practice of medicine in a small rural town in northern Pennsylvania.

> . . . he had plenty of very promising investments in gold and copper mining stock. He had every expectation of their paying off handsomely in a year or two. Father had advised him differently; but he was only a conservative Philadelphia lawyer and Uncle Donnie was a famous explorer . . . as time was beginning to hang heavily on his hands, he thought he might as well explore his own country a little. Accordingly he and his valet mounted horses, and rode out from Philadelphia to San Francisco, taking plenty of leisurely side trips to interesting places en route.
>
> There was a sort of minor financial crash about that time. When he reached the Pacific Coast, he found word awaiting him that his investments had just gone to nothing. The beautifully engraved certificates that had attracted his esthetic eye were still interesting as works of art, but were no longer attractive to cold-eyed bankers. In other words, he was just plain broke. I think he sold his horse and probably a shirt or two to get home. I do not know what happened to the valet.[83]

Thus by the early 1920s Smith had become penurious. He certainly could have opened a medical practice in Philadelphia. Instead he chose rural northern Pennsylvania. H. Spencer Potter relates that Smith chose his location after reading the town's advertisement for a physician in a medical journal.[104] Pleasants says of his choice:

> . . . Father would not tell us where he was either. Actually, he had gone up to a little town in the wilds of northern Pennsylvania, and had opened an office for the practice of medicine. The people were wild about him, too. In those days, a country office visit averaged fifty cents, and a house call one dollar—if you could get it—regardless of mileage. It was not often that a community could get the services of a physician trained at Johns Hopkins, Harvard and Heidelburg, and thoroughly experienced in the treatment of tropical diseases, at such rates. Of course, he chose a place not too far from the native haunts of wild turkey and grouse, and where the trout ran really big. He also took his rifles along just in case the deer and bear became too annoying.[83]

The reason for not telling the children where Smith was is somewhat perplexing. Although Smith loved the out-of-doors he could have enjoyed them at intervals as he had done all along. This then raises the possibility that his going there was not uniquely due to his desire to be in a place where he could hunt and fish. As the inhabitants of Roulette, Pennsylvania, soon learned, Smith was afflicted with a problem which no doubt had caused his family much distress for many years.

H. Spencer Potter recalls his parents saying that Smith did some medical practice at the Chestnut Hill Hospital and at Jefferson Medical College after World War I and before opening his practice in Roulette.[105] It may be that Smith undertook refresher experiences at these institutions since he had been away from the routine practice of medicine for so long.

MOVE TO ROULETTE, PENNSYLVANIA

The *American Medical Directory* for 1921 lists Smith in the index as being in Port Allegany, Pennsylvania.[103] However under the listing of Port Allegany his name does not appear, perhaps because of a printing error.[103] The eighth edition of the same directory published in 1923 lists his address as Roulette, Pennsylvania.[106(p1439)] The archives of the Royal Geographical Society in London have 1924 as the first year he used Roulette as his address.[81] Thus it appears that Smith arrived in Roulette sometime between 1922 and 1924 and seems to have remained there until either 1933 or 1934. His address in the 13th edition of the *American Medical Directory* published in 1934 is given as Scotland Neck, North Carolina.[107] The Royal Geographical Society has 1935 as the last year he was receiving correspondence in Roulette, after which he received it in Philadelphia.[81] Thus, overall Smith spent roughly ten years or so doing general practice in Roulette.

Roulette (pronounced Raoulet) was a small town of some 1,500 people situated on the Allegheny River in Potter County in north-central Pennsylvania.[103] Today the town has only several hundred inhabitants. At the time of Smith's arrival, Roulette had only one other practicing physician, George Bigelow Mullison, who was 18 years Smith's junior and a 1912 graduate of Temple University Medical School.[106(p1439)] By 1925 Mullison had moved on to Silver City, New Mexico, and later to Albuquerque, New Mexico.[108,109] The other physician in Roulette at that time was Edith Stevens, who was born in 1848 and who graduated from Women's Medical College in Philadelphia in 1890.[103] She was retired and not in practice at the time and was the daughter of a local preacher.[37,103] Edith Stevens does not appear in the *American Medical Directory* listings after 1934.[107]

Since the late 19th century Roulette had always had one or two practicing physicians, beginning with a Dr Post who moved into the town in 1893 from Fishing Creek a short distance away.[110(p170)] He took into his home a young orphan, Charles Tauscher, who had been born in Saxony, Germany, and who later became a physician in Roulette.[110(p170)] Smith set up his first practice and rented living quarters from one of Dr Tauscher's descendants, Albert Tauscher, and his wife, Rosella, when he first came to Roulette.[111] The physician whom Smith replaced was Walter Howland Squires who had first started practice in Roulette in 1899 and whose name appeared under the town's in the first *American Medical Directory* published in 1906.[110(p179),112] He had left the town by 1921.[103] Roulette also had another physician in practice between 1906 and 1918, Fred H. Houghton, who was a graduate of the University of Vermont (1880) and who died during the influenza epidemic of 1918.[103,110(p179)]

Roulette had been established in 1816 and had been named after Jean Roulet of the Ceres Land Company.[113(p368)] Originally a farming and lumbering community, it was eventually transformed into a small industrial center in 1886 with the opening of a tannery.[110(p171)] Roulette's transformation from a purely agricultural community to a lumbering one began very early on since it was situated in the midst of one of the greatest hemlock stands in the state. Lumbering was the principal industry for more than 70 years. However, the last great stand of hemlock, the Central Pennsylvania Tract, was cut in 1912.[113(p368)] The forests around Roulette, however, provided more than logs. The bark of the hemlock was extensively used in the tanning industry which accounts for the arrival of a tannery in 1886.[110(p171)] The original tannery employed some 65 men, had a capacity of 300 hides a day, and used 3,000 cords of bark a year.[110(p171)] Many of the hides came from Africa. In 1901 the tannery closed and its old buildings were eventually converted into a bottle factory in 1911 which functioned until 1923.[110(pp171-172)] The Roulet Glass Company started operations in 1903, but closed in 1908.[110(p172)] However, one of the largest industries to operate in Roulette was the Gray Chemical Company, which started in 1902 and continued operations during the time Smith lived in the town. The plant produced acetic acid and alcohol.[110(p173)] Many of the industries located themselves in Roulette in order to be proximal to the primary resources needed in the manufacturing processes and also because of the abundance of firewood which early on was used to fire the ovens. Thus Roulette at the time of Smith's residence was far from being an idyllic spot in the country. It was in fact a microcosm of the industrial revolution that consumed some of the natural resources of its surroundings, especially the forests, and whose industries no doubt generated air and other forms of pollution that were not documented (Fig 8). Mrs Alice Morey, who grew up in Roulette in the early years of this century, recalls that the Gray Chemical Company's smokestacks produced effluents that covered the ground with soot.[114] Roulette was also situated near one of the largest natural gas fields in the state, and in 1903 a line was built from the town to Elmira in nearby New York State.[110(p177)]

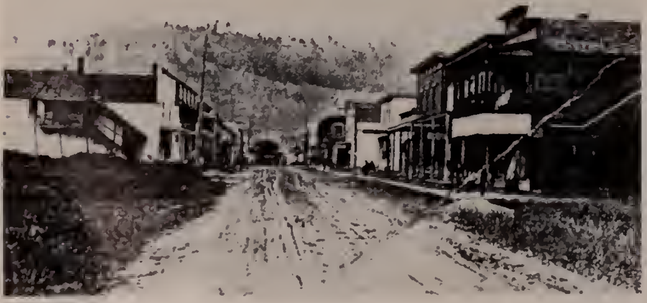


FIGURE 8. The business section of Roulette as it appeared in Dr Smith's time (reproduced from *Golden Memories 1905–1954*, Roulette, Pa, Journalism Club of Roulette Township High School, 1954 (courtesy of Dr Frank A. Rackish).

By the time Smith arrived in Roulette the deer and bear had both been hunted out and their habitat largely destroyed through logging and the cutting of wood for local industrial use.[37,115] Later on the region was restocked with deer and today they are found in large numbers, as are bears.[37] Secondary growth forests now cover much of the area.[37] The trout fishing in the Roulette area was excellent during the time Smith lived there and this no doubt had great appeal to him.[37] The Roulette that Smith entered was a very different social place from the circles in which he moved in Philadelphia and London. Basically a farming and factory town, its inhabitants were hard working people struggling to get by, a struggle that became all the more difficult after the beginning of the Great Depression in 1929.[37] A succinct description of the effect of the depression on Roulette is contained in a Pennsylvania travel guide: "...Along its single street, hemmed in by towering mountains, are many unpainted houses abandoned in the exodus caused by the 1929 depression." [113(p368)] The population of all of Potter County, where Roulette is situated, fell by almost half between 1900 and 1930, due to both the Great Depression and the ending of the lumbering boom.[116] Roulette was, in the words of one informant, "...a pretty rough town in those days." It was into this setting that Smith, a refined, well-educated, well-travelled Philadelphia aristocrat, moved and set up a medical practice.

Smith had come highly recommended to the people of Roulette by Dr Ross Holman Jones, a 1901 graduate of the University of Pennsylvania School of Medicine who practiced in Coudersport, the Potter County seat, ten miles to the east.[106(p1371)] Jones, who was 12 years younger than Smith, told the townspeople that Smith had excellent training and that he himself had examined all of his certificates and diplomas.[117] It appears that Jones, who later practiced anesthesiology in Coudersport, facilitated Smith's entry into Roulette.[107] No doubt the people of Roulette were happy to have a physician in their midst since the nearest ones were in Coudersport, ten miles to the east, or else in Port Allegany, seven miles in the other direction.[37] Smith started practice at a time when patients judged physicians more on the basis of their education and learning than on their technical skills, and according to these standards he ranked among the best.

MEDICAL PRACTICE IN ROULETTE

Smith began his medical practice in rented space on the ground floor of the house on Main Street owned by Albert and Rosella Tauscher (Fig 9).[37,111] He later moved to a house a short distance away and in his last years in Roulette lived and practiced in a small row house on North Street.[37,114,117] During the years he was in Roulette his post office box number was 216. His practice was confined to primary care and he did not perform surgery or engage in obstetrics.[118] He gained an excellent reputation for his success in treating patients with pneumonia, then a common problem in the winters.[118] Alice Morey vividly recalls that Smith treated her brother for pneumonia. He made a

FIGURE 9. The west end of Roulette, Pennsylvania, around 1910. The arrow indicates the building where Dr Smith had his first office (courtesy of Robert Currin, President of the Potter County Historical Society).

house call every morning and was caring and concerned.[114] Barbara O'Connor recalls that, "He was known to do wonderful things for people with pneumonia. He opened all the windows and let the cold air in."[118] Cold air treatment for patients with pneumonia was the accepted standard at the time Smith was in practice.[119] On one occasion he arrived at the home of a pneumonia patient and found that contrary to his orders the windows had been closed. He promptly picked up a book and threw it through the window, letting in the cold winter air. Some people in Roulette believed that his great success with pneumonia patients was due to his use of a concoction he had learned of in Africa.

In general most patients were extremely satisfied with "Doc Smith" as they came to call him. They knew that he was well educated and well trained, were confident in his abilities, and were pleased by his excellent bedside manner. He was a devoted physician who was extremely sensitive to his patient's feelings.[105] In Roulette there was a good deal of what used to be popularly termed "summer complaint" and the usual childhood communicable diseases. Smith was highly regarded for his skills in treating these.[118] Bonnie Darrin recalls that when she was a young girl Smith vaccinated all of the children in the local school including herself.[111]

Smith of course was the only doctor in Roulette and it was not easy for people to go elsewhere for medical care. When a serious problem arose people did go to Coudersport or else to Port Allegany where there was a hospital.[114,119] Smith cut a distinguished figure with his short-cropped gray hair with a dark streak down the part, his spectacles, polished manner, and neat clothes.[105,111] He either wore jackets or sweaters which, although at times a bit threadbare, were well fitted.[118] Bonnie Darrin recalls that he reminded her of a German with his gray, bush-cut hair and glasses.[111] There were some people in Roulette who would not consult Smith because of his episodic inebriation and the aberrant behavior he displayed at those times.

Smith's fees were modest and often he was paid in kind. Not infrequently he treated people free of charge when they could not afford to pay.[118] Thus "Doc Smith" was seen from a certain perspective as a refined gentleman, a well trained physician, and as sympathetic, kind, and caring. That he provided his services for nominal fees or none at all also increased his popularity. He was able to do this for two reasons. Living costs for a bachelor such as himself in Roulette were modest. In addition, the husband of his niece Margaret Spencer, Charles A. Potter, Jr, who was a successful Philadelphia businessman, regularly supported Smith with remittances during his years in Roulette.[105] Given Roulette's small population of 1,500 when Smith arrived and its sharp decline after the onset of the Great Depression, it is unlikely that a physician could have financially succeeded in medical practice, especially if he charged only nominal or no fees at all. Even if every inhabitant of the town had one bout of illness per year, which is unlikely, Smith would have seen no more than five new patients per day in that time. From all accounts he was never very busy with his practice, but the people were glad to have him just the same.[37] George C. Mosch, MD, the secretary-treasurer of the Potter County Medical Society, communicated to this writer on April 4, 1986, that ". . . Dr. Smith was remembered as being rather elderly when he got there [Roulette], that he was not very active in clinical practice and that after he left and went to Philadelphia he died within six or seven years. . . ."

SMITH'S SOCIAL PLACE IN ROULETTE

Soon after his arrival in Roulette, Smith became friends with L. L. Smith, who had been appointed principal of the local school in 1921.[110(p178)] L. L. Smith, who was called "Prof Smith" by the townspeople, had much in common with Smith. Both were well educated, they enjoyed playing bridge and other games of cards, and were excellent fly fishermen.[37,105,118,120(p28)] Prof Smith taught in the Roulette School for 32 years and the *Golden Memories 1905–1954* book published by the Journalism Club of Roulette High School is dedicated to him.[120(p7)] Frank Baxter, who came to Roulette in 1942 to work with the gas company, recalls that Prof Smith was an expert fly fisherman who taught one of his sons to fish.[121] He was also a stamp collector and built his own FM radio, the first in Roulette.[121] Prof Smith regularly taught high school children how to fish. A report from the Roulette High School Sportsmen's Club states: "Mr. Smith brought the fly tying equipment to one of the meetings, explained the uses of the different devices, and tied a few flies. . . Mr. Smith brought his casting and fly equipment to a meeting and explained how to use them. After he demonstrated for the group he let the members try their ability with them."[120(p28)] Arthur Donaldson Smith was also ever ready to teach people to fish and taught his grandnephew H. Spencer Potter to do so.[105]

The two Smiths were also very private men. However, their interests and educational backgrounds gave them much in common and they regularly played bridge at Arthur Donaldson Smith's house along with other men from the town. Prof Smith is thought by some to have been married once and to have had a daughter. However, during his years in Roulette he was, like Arthur Donaldson Smith, essentially a bachelor.

Arthur Donaldson Smith did not socialize with people in their homes. However, he regularly invited teenage boys and young men over to his house to play bridge. He was excellent at playing not only bridge but also dominoes and chess.[105] Although he did not have a housekeeper, his laundry was done by women in town and neighborhood women often brought him cakes and pies.[37,118]

Smith kept a number of hunting trophies with him at Roulette. He had no mounted heads there, but Grayel Tauscher recalls that he had a leopard skin and a tiger skin rug and python skins.[37] He also had a number of swords, daggers, spears, and guns.[37] He had a Greener shotgun which he himself did not use, but which he regularly lent to Tauscher.[37] This gun, like the Purdey weapon now in the possession of his grandnephew, was probably manufactured in England.[105,122] Smith also told Tauscher that he had a double-barreled .577 in Philadelphia,[37] no doubt the same weapon he had lent Percy Cox when the latter went out shooting rhino in 1899 during their expedition to Somaliland (see part IV). Smith also told Tauscher about his Purdey weapon, no fabrication as it is now in the possession of his grandnephew, H. Spencer Potter.[105]

Smith was not a boastful man.[105] Always modest, he rarely spoke to his family about his African experiences.[105] In Roulette he did speak of them, not in a boastful manner, but in a matter-of-fact way. A measure of the man is the fact that everything remembered and recounted by informants to this writer was absolutely accurate. Barbara O'Connor remembers that there was a mountain in Africa named after him and Grayel Tauscher remembers Smith's bitterness over the failure of the Royal Geographical Society to reimburse him for any part of his expenses from his expedition to the Nile.[37,118] Many local people were aware of his accomplishments in Africa but as Grayel Tauscher notes, "Many were not very interested. They were too busy just trying to make a living." [37] Smith lent him his book, *Through Unknown African Countries*, which he read.[37]

In addition to playing bridge, Smith spent a lot of time trout fishing. He also enjoyed swimming and diving in local creeks. On one occasion he dove into shallow water and severely scraped his face.[37] His nephew, Charles A. Potter, Jr, and Potter's wife, Margaret, Smith's niece, visited Smith on several occasions. Margaret Potter found Roulette a terrible place.[105] H. Spencer Potter, Smith's grandnephew, also visited him on a few occasions.[105] Some of the townspeople remember Charles A. Potter's visits: "His nephew who owned the Potter Linoleum Company in Philadelphia, came to visit him in a big car. Doc Smith got all dressed up for the occasion in 'high hat and tails' you might say." [117]

Smith was popular with the young boys of the town because he would entertain them with stories about his experiences in Africa and other far off areas of the world.[111] Smith's home also became a refuge for a succession of several teenage boys who for a variety of reasons had left their own homes or else had been thrown out of them. He spent a great deal of time and effort trying to educate and improve them. One boy, whom Smith was planning to adopt, ran off when Smith insisted he learn logarithms.

The Ku Klux Klan was active in Roulette and Smith was an outspoken public critic of it and of some of the men who belonged to it.[37] He even coined a brief derogatory verse about a local merchant who belonged to the Klan and recited it frequently and with satisfaction.

SMITH'S ILLNESS

There was another side to Smith's life in Roulette that reflected an illness with which he must have been afflicted since the turn of the century. In retrospect, the two incidents at the Rittenhouse Club in 1901 and 1905 indicate that his struggle with ethanolism went back a number of years. In Roulette his illness took the form of periodic binges of one to two weeks in duration, which occurred every few months. During these times Smith exhibited bizarre behavior which was often perceived as hilarious. Based on the descriptions provided to this writer by firsthand observers over 50 years later, it is difficult to ascertain whether or not he was also suffering from some of the psychiatric and physical complications of the disease and if so, to what degree. Between these bouts Smith was a totally different person: alert, courteous, coherent, and appropriate. This would tend to preclude the suspicion of some people that he was also regularly using other substances.

There are numerous anecdotal stories about Smith's behavior during these times. In general he became aggressive and word that Doc Smith was out on the street spread quickly. People usually kept out of his way and some bolted their doors. He often appeared on the street with a pistol, usually a .32 caliber weapon, and would shoot it off into the air. On one occasion he became angry while listening to a radio broadcast of "The Three Black Crows." He put three shots into the radio, one for each of the characters in the show.[37] The story of his shooting at his radio still has wide currency among those who were in Roulette at the time. One day he appeared on the street on a sunny day with an umbrella and his gun. On another he lit up a bonfire in the middle of the street and danced around it, imitating the dances of American Indians. Not infrequently he also carried a sword, and on one occasion made a young boy dance to avoid being struck by it.

In his impaired state he once held a group of young men in his house and at gun point made them play bridge for a few days. The second home he lived in had a living room with many doors, making escape difficult. Those fleeing from his pointed gun or sword often ran out the wrong door, and once a young man ended up in the cellar.[37]

Smith had an ongoing feud with an elderly widow, Mrs Jordan, who lived on the next block. She wore long skirts and was in the habit of carrying a lantern when she walked around at night as there were no street lights. Smith used to scare her by chasing her, pistol in hand. One night she stood her ground and hit him over the head with her lantern. In his impaired state he also took on some able-bodied men who in defending themselves gave him some heavy blows and knocked him to the ground.[37] At other times he would show up at public dances while impaired and would try to teach everyone to dance. His performances threw everyone into laughter. If not inebriated, he would never have attended such affairs.[37]

Once he entered a local restaurant which had a soda fountain and, pointing his gun at the 12-year-old daughter of the proprietor who was behind the counter, said, "Young lady, I will thank you to drink four cherry cokes." As she drank them he stood there smiling, with the gun pointed at her and a hat on his head. He then thanked her and left. Another time he entered the local pool hall with a sword. The proprietor was an enormously obese man whose paunch was draped over the edge of a counter. Smith went to jab his abdomen, saying, “I’m going to let some pus out of you.” Fortunately, the man pulled away.

Smith was viewed more as a prankster than a menace. Although he shot his revolver off into the air and into the radio he never actually shot anyone. Adults, however, gave him a wide berth when he was drunk. Youngsters followed him because they wanted to be entertained by whatever it was he decided to do. In Roulette, where few had radios, Smith was in a sense a one-man show. Most people had guns or rifles and regularly carried them so there was nothing unusual in Smith’s doing so.

One year Smith made the annual high school play a great success, but for reasons the director could not have foreseen. A girl who played the role of the doctor asked him to prepare a doctor’s bag. He readily agreed and she carried it out on stage. At a given time she was to open the bag and take out rolls of bandages. She reached into the bag and pulled out what she thought were bandages but which in reality were sanitary napkins. This brought the house down with laughter, much to the chagrin of the young actress.

Today, Smith would be considered an impaired physician. At the time he practiced in Roulette there was no routine peer monitoring or review of a physician’s professional performance nor were there mechanisms for referral, treatment, and follow-up of impaired physicians. Thus he was able to function in this condition for close to a decade without any intervention. Although prohibition was in effect, physicians were able to obtain alcohol for medicinal purposes by using special forms.[119] Some townspeople speculated that he brewed his own alcohol according to a formula he had learned in Africa.

Between his binges he was a perfect gentleman who on occasion invited young men to his home for dinner. He was a gourmet cook who liked to use a lot of spices. He set a table to which the young rural men of Roulette were unaccustomed, with its tablecloth, napkins, utensils properly laid out, and every glass in its place.[118]

RETIREMENT

Smith was at least 57 years old when he arrived in Roulette. When he left, probably in either 1933 or 1934, he was 67 years old. His friend, L. L. Smith, the principal of the high school, stayed on until his death. A new elementary school on North Street was opened in January 1962 and the following May was dedicated as the L. L. Smith School.[110(p178)] It has since been converted into a facility for handicapped children. The Roulette High School closed in 1954, students being taken to Port Allegany by bus.[110(p178)]

Smith moved for a while to Scotland Neck, North Carolina, where he had been quail shooting since at least the 1890s. At the time and even today, Scotland Neck had a number of private hunting clubs.[123]

Smith’s eyesight was extremely poor[105] at this time, possibly because of cataracts. Pleasants says the following of his retirement from Roulette, and provides some interesting insights about him at this stage of his life:

> He was growing blind. His old friends, including Father, had passed on. He was not making enough in his practice of medicine to live on; but a generous relative by marriage had settled a monthly allowance on him that paid his rent [Charles A. Potter, Jr]. The Veterans’ Administration was persuaded to take some action on his case, and eventually granted him eighty dollars a month as disability compensation. This was later cut to forty dollars, after he had closed out his office and sold his equipment. After all, he had only been a captain; and his blindness was not directly “service-connected.”
>
> However, Uncle Donnie had enough to start down to Florida. He stopped in to see me on his way, and spent two or three days at my house. Hank, my oldest son, was fascinated by him. He wrangled the real story of his Mongolian experiences and his second expedition to Africa out of him. Hank was then on the editorial staff of *The Evening Bulletin*, and is now a captain in the Air Corps, Military Intelligence Section, with General Mark Clark, in Italy, and could get a story out of the Sphinx itself.
>
> It was then for the first time that we found out that he had served in the Khyber Pass Campaign with the British Army, as a sort of preliminary warm-up for his later venture. In 1898, taking a body of trained troops, he had penetrated into central Africa. Eleven Sepoys, whom he had brought with him, deserted, however; and actually he had only thirty-nine men with him at the most critical period. You see, the British had acted on the advice he had given in his book regarding laying claim to that rich country. They had selected him to carry their flag, not only to Lake Rudolf, but clear on to the Nile River, through country infested with real cannibals.
>
> It was wonderful to sit there in a nice, comfortable room and listen to him. A strange, eager light had come into his mild blue eyes. The deep tired lines of his gaunt face seemed to fade as he lived again the stirring days of adventure when each tribe of savage warriors had to be dealt with carefully and tactfully so that it might assist the expedition a few miles on its way through the trackless, steaming jungle. Of one incident he said: “The chief came to visit me in camp. I was anxious to gain his good will, so I gave him my favorite leopard rug. He was delighted. He sent a runner back to his village for an elephant tusk to give me. The man returned with a small one that weighed perhaps twenty-five pounds. The chief took one disgusted look at it. The next I knew—CRASH! I can hear the tusk yet as it went through the runner’s skull.”
>
> The rest of the story of Uncle Donnie is not important. He wandered around the southern states “trying to find a warm place.” At last, he came back to Philadelphia; and passed on to explore some brighter land.[83]

Smith did not remain in Scotland Neck for long, moving on to Philadelphia, where he lived with his niece.[105] He later spent time in St Petersburg, Florida, where during his last stay in 1938 he lived at the Wilsonia on Fifth Avenue South.[124] His address with the Royal Geographical Society during the last four years of his life was 1510 Chestnut Street, Philadelphia, the office of his niece’s husband, Charles A. Potter, Jr. Smith divided his time between St Petersburg and his niece’s Philadelphia home.[105] The *St. Petersburg Times* said of his activities: “. . . He spoke here on many occasions, chiefly after Mussolini’s march into Ethiopia, a country he had explored twice. At that time he characterized the Ethiopians as ‘vulgarly immoral’ and incompetent to govern themselves.”[124]

The obituary in the *St. Petersburg Times* has a photo of Smith taken in old age. It depicts a neatly dressed man with a moustache, and hair carefully parted slightly off the center on the left.[124]

Smith’s health was poor in 1938 and on February 19, 1939, he died at 1:15 PM at the US Naval Hospital in Philadelphia, where he had been hospitalized on January

10. His cause of death was listed as coronary disease with occlusion. Contributory causes were arteriosclerosis, generalized severe nephritis, and chronic endocarditis.[125] In writing to Dr Herbert B. Carpenter, the secretary of Smith's medical school class, his niece's husband said of his long illness:

> According to Dr. White, in the Naval Hospital, who attended Donaldson, his death was caused by septic pneumonia. Donnie's condition, I would say, was a general breakdown, accompanied by definite heart trouble. I am afraid the dear boy did not show much willingness to help the doctors get him well, and the family feel, under the circumstances, his death was a blessing.[126]

Dr Carpenter in writing to Potter said: "Donny was a beloved member of the class and will be greatly missed by our fast thinning ranks."[127]

Smith's death was widely noted in a number of newspapers (see part I). The *Potter Enterprise* essentially reprinted the story that had appeared in the *Philadelphia Inquirer*. They added the subtitle, "Famous Explorer who Practiced at Roulette Following the War."[128] During the years Smith practiced in Roulette there was no town paper, its news being carried in the *Potter Enterprise* under a special column called "Roulette News."[128] A careful search of the *Potter Enterprise* for any mention of Smith was undertaken by Mr Robert Currin, President of the Potter County Historical Society. There is only one mention of him, on March 1, 1928: "Dr. Smith was called to see Mrs. George Adams, Sr. on Tuesday of this week." The Royal Geographical Society hinted at the downward turn Smith's life had taken when in its lengthy obituary notice it noted, ". . . In view of his two fine journeys and the excellent observations of which he proved himself capable, it was a pity that he was unable to continue his African travels in later years."[129] Smith was buried in the Smith family plot in Laurel Hill Cemetery on February 21, 1939. His unmarked grave lies to the right of that of Grace Helen Early Smith, his nephew's wife, who was buried there three weeks before on February 1, 1939. At her left is her husband, Henry Cavalier Smith, Jr, who died in Yonkers, New York, in August 1950.[130]

Many of Smith's papers and memorabilia have been dispersed and for the most part are considered lost at this writing. He had stored many of his things at the Grange, the Spencer's 5,000-acre farm in the Susquehanna Valley, but a fire in the early 1930s completely destroyed the house and its contents.[105] The Spencers lost most of their wealth, including the Grange, in the Great Depression.[105] H. Spencer Potter remembers a trunk of possessions being shipped up from St Petersburg, Florida, after Smith's death.[105] Some of Smith's belongings had been stored in the barn attached to the home of Albert Tauscher in Roulette where Smith first roomed. In the early 1960s the contents of the barn were auctioned in a two-day sale by Albert Tauscher's descendants. Both Bonnie Darrin and her brother, Grayel Tauscher, who were niece and nephew of Albert Tauscher, recall that Smith's books and diaries were stored in a basket with other odds and ends and auctioned off.[37,111] Smith gave the telescope that he had used in his travels to Grayel Tauscher, who still has it today.[37,111] Fortunately, the bulk of Smith's natural history and ethnographic collections are safely preserved in several museums, and his published writings and his letters in the Archives of the Royal Geographical Society and elsewhere well document his exceptional career as an explorer.

Although much of the Africa Smith knew has changed, there is much that remains the same. He would still find northern Kenya and southern Ethiopia familiar places, with their great landscapes and splendid views. And the lake is still there, a shimmering jade sea, remote and lonely, set like a jewel in a vast expanse of thorn bush and desert. Jonathan Kenworthy, the renowned British artist and sculptor, has recently reproduced this world in bronze and in sketches—the Rendille and the Boran, the Somali and the Gabbra, the shimmering heat waves and the cool springs.[131] That he has been able to do so in the 1970s and 1980s, so long after Smith's passage, says that there is something eternal about this place. Smith was among the first to bring it to others to share and enjoy. It is a world which once touched cannot be forgotten, a world whose discoverers will always be remembered.

REFERENCES

1. Letter from A. Donaldson Smith, MD, to Percy Z. Cox, London, October 26, 1898. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.
2. Wellby MS: *'Twixt Sirdar and Menelik: An Account of a Year's Expedition from Zeila to Cairo Through Unknown Abyssinia*. London and New York, Harper & Brothers, 1901, reprinted, New York, Negro Universities Press, 1969.
3. Wellby MS: King Menelik's dominions and the country between Lake Gallop (Rudolf) and the Nile Valley. *Geographical J* 1900; 16:292–306 (map p380).
4. Turton D: Exploration in the lower Omo Valley of Southwestern Ethiopia between 1890 and 1910, in Caravaglios M(ed): *L'Africa ai Tempi di Daniele Comboni*. Roma, Instituto Italo-Africano, 1981 (typescript copy courtesy of Professor David Turton, University of Manchester, Manchester, England).
5. Von Höhnel L: The Lake Rudolf region. Its discovery and subsequent exploration 1888–1909. Part I. *J of the Royal African Society* 1938; 37:21–45.
6. Captain M. S. Wellby, 18th Hussars [obituary]. *Geographical J* 1900; 16:358–359.
7. An Englishman in the Lake Rudolf region. *Geographical J* 1902; 19:197–198.
8. The Harrison-Whitehouse Expedition. *Geographical J* 1900; 16:103.
9. Jesman C: *The Russians in Ethiopia. An Essay in Futility*. London, Chatto and Windus, 1958, reprinted, Westport, Conn, Greenwood Press, 1975.
10. Zaghi C: *I Russi in Etiopia. Il Protettorato italiano, sull'Etiopia*, vol I, *Menelik e la battaglia di Adua*, vol II. Napoli, Guida Editori, 1972.
11. Sanderson GN: The foreign policy of the Negus Menelik, 1896–1898. *J African History* 1964; 5:87–97.
12. Bulatovich AK: Dall'Abissinia al Lago Rodolfo Per il Caffa. Conferenza del Capitano AK Bulatovich letta nell'adunanza generale della Imperiale Società Geografica Russa il 13 gennaio, 1899. *Bollettino della Società Geografica Italiana*. Serie IV 1900; 37:121–142.
13. D'Orléans, Prince Henri, *Une Visite à l'Empereur Mênêlick*. Paris, Librarie Dentu, 1898.
14. Boulatovich AK: *L'Armée de Sa Majestê Mênêlik II, Journal de l'expêdition des Ethiopiens au lac Rodolphe*. St Petersbourg, 1900.
15. Butzer KW: *Recent History of an Ethiopian Delta. The Omo River and the Level of Lake Rudolf*. University of Chicago Department of Geography Research Paper No. 136, Chicago, 1971.
16. Bieber FJ: *Kaffa. Ein altkuschitisches Volkstum in Inner-Afrika*. Munster-Wien, Anthropos-Bibliothek, 1920 (vol I), 1923 (vol II).
17. Pantano GV: *Ventitre Anni di Vita Africana*. Firenze, 1923.
18. Lev Koubel, Données sur les rapports socio-economiques chez les peuples de l'Ethiopie dans les oeuvres de A. K. Boulatovitch (Voyages des annees 1896–1899), in *Proceedings of the Third International Conference of Ethiopian Studies, Addis Ababa, 1966*. Addis Ababa, Institute of Ethiopian Studies, Haile Selassie I University, 1970, vol III, pp 160–166.
19. Rodd JR: *Social and Diplomatic Memories 1894–1901*. London, Edward Arnold, vol 2, 1923.
20. Leymarie H: *Un Dieppois en Abyssinie*. Dieppe, Imprimerie de Vigie de Dieppe, 1898.
21. de Léontieff N: Exploration des provinces équitoriales d'Abyssinie. *La Gêographie* 1900; 2:105–118.
22. Count Leontieff's journey south of Abyssinia. *Geographical J* 1900, 16:558.
23. de Léontieff N: *Provinces équitoriales d'Abyssinie. Expédition du Déjaz comte N de Léontieff*. Paris, Chambrelent, circa 1900.
24. Powell-Cotton PHG: *A Sporting Trip Through Abyssinia*. London, Rowland Ward, 1902.
25. Martini F: *Il diario eritreo* (4 vols). Roma, Vallecchi, 1947.

26. Vivian H: *Abyssinia, Through The Lion Land to the Court of Judah.* London, Arthur Pearson, 1901.

27. Smith AD: An expedition between Lake Rudolf and the Nile. *Geographical J* 1900; 1:600-625.

28. Graves P: *The Life of Sir Percy Cox.* London, Hutchinson and Co, 1941.

29. Letter from A. Donaldson Smith, MD, to J. Scott Keltie, Fort Berkeley, Nile, March 21, 1900. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.

30. Oldfield T: List of mammals obtained by Dr. Donaldson Smith during his recent journey from Lake Rudolf to the upper Nile. *Proc of the Zoological Society of London* 1900; 68:800-807.

31. Collins RO: *Land Beyond the Rivers, The Southern Sudan, 1898-1918.* New Haven and London, Yale University Press, 1971.

32. The Monthly Record. Africa. Dr. Donaldson Smith's Expedition. *Geographical J* 1900; 16:102.

33. Letter from A. Donaldson Smith, MD, to J. Scott Keltie, London, June 24, 1900. A. Donaldson Smith Correspondence File, Royal Geographical Society Archives, London.

34. Smith AD: An expedition between Lake Rudolf and the Nile. *The Alumnni Register*, February 1901; 148-155.

35. Smith AD: An expedition between Lake Rudolf and the Nile. *Bulletin of the Geographical Society of Philadelphia*, 1900; 2:115-138.

36. Sharpe RB: On the collection of birds made by Dr. A. Donaldson Smith on his last expedition to Lake Rudolf and the Nile. *Proceedings of the Zoological Society of London.* 1901; 69:602-622.

37. Personal communication, Grayel Tauscher, Tioga, Pa, May 15, 1987.

38. Letter from A. Donaldson Smith, MD, to J. Scott Keltie, London. Undated, 1900, A. Donaldson Smith Correspondence File, Royal Geographical Society Archives.

39. Malone D (ed): Elisha Kent Kane, in *Dictionary of American Biography.* New York, Charles Scribner's Sons, 1932, vol 5, pp 256-257.

40. Smith AD: Africa, from the Omo to the Nile. *Independent* 1900; 52:3093-3096.

41. American Explorer. Portrait. *Critic. An Illustrated Monthly Review of Literature, Art and Life*, January 9, 1897; 30:28.

42. Meetings of the Royal Geographical Society Session 1900-1901 Anniversary Meeting, May 20, 1901, Sir Clements Markham, K.C.B., F.R.S., in the Chair. *Geographical J* 1901; 18:98-100.

43. Harrison JJ: A journey from Zeila to Lake Rudolf. *Geographical J* 1901; 18:258-275.

44. The Harrison-Whitehouse expedition. *Geographical J* 1900; 16:103.

45. James J. Harrison, in *Who Was Who 1916-1928*, ed 4. London, Adam and Charles Black, 1967.

46. Austin HH: A journey from Omdurman to Mombasa via Lake Rudolf. *Geographical J* 1902; 19:669-690.

47. The Abyssinian-frontier expeditions. *Geographical J* 1901; 18:533.

48. Austin HH: *Among Swamps and Giants in Equatorial Africa. An Account of Surveys and Adventures in the Southern Sudan and British East Africa.* London, C. Arthur Pearson Ltd, 1902.

49. Wickenburg, Graf. Von. Von Dschibuti bis Lamu, *Petermanns Mitteilungen* 1903; 49:193-199.

50. Count Wickenburg's journey from Jibuti to Lamu. *Geographical J* 1901; 17:698.

51. Count Wickenburg's journey from Jibuti to Lamu. *Geographical J* 1902; 19:216.

52. *Mission Scientifique du Bourg de Bozas. De la Mer Rouge à L'Atlantique à travers L'Afrique Tropicale, Octobre 1900-Mai 1902.* Paris, F.R. de Rudeval, 1906.

53. *Mission Scientifique du Bourg de Bozas. Communications addressês au cours de route à la Sociêtê de Gêographie par le Vte R. du Bourg de Bozas et publiêes au Bulletin de cette Sociêtê dans les Nos de Juin 1902 et Fevrier 1903.* Paris, F.R. de Rudeval, 1906.

54. Maud P: Exploration of the southern borderland of Abyssinia. *Geographical J* 1904; 23:552-579.

55. Harlow V, Chilver EM, Smith A: *History of East Africa.* Oxford, Oxford University Press, 1965, vol 2.

56. Brooke JW: A journey west and north of Lake Rudolf. *Geographical J* 1905; 25:525-531.

57. Gwynn CW: A journey in southern Abyssinia. *Geographical J* 1911; 28:113-139.

58. Boyes J: *My Abyssinian Journey. A Journey Through Abyssinia from the Red Sea to Nairobi in 1906 in the Days of the Emperor Menelik* (probably published in Nairobi in the early 1940s).

59. Trzebinski E: *The Kenya Pioneers.* WW Norton, New York and London, 1985.

60. Huxley E: *White Man's Country.* London, Chatto and Windus, 1935 (vol 1), 1953 (vol 2).

61. Stigand CH: *Equatoria. The Lado Enclave*, with an introductory memoir by General Sir Reginald Wingate. London, Constable and Co, 1923.

62. Stigand CH: *To Abyssinia Through an Unknown Land. An Account of a Journey Through Unexplored Regions of British East Africa by Lake Rudolf to the Kingdom of Menelik.* Philadelphia, JB Lippincott Co, 1910.

63. Spencer P: *Nomads in Alliance. Symbiosis and Growth Among the Rendille and Samburu of Kenya.* London, Oxford University Press, 1973.

64. Baxter PTW: *Social Organization of the Boran of Northern Kenya*, shortened version of D.Phil. thesis. Lincoln College, Oxford, 1954.

65. Baxter PTW: Giraffes and poetry: Some observations on giraffe hunting among the Boran. *Paideuma* 1986:45-63.

66. Turton D: A problem of domination at the periphery: The Kwegu and the Mursi, in Donham D, James W (eds): *The Southern Marches of Ethiopia.* Cambridge, Cambridge University Press, 1986, pp 148-171.

67. Turton D, Turton P: Spontaneous resettlement after drought: An Ethiopian example. *Disasters* 1984; 8:178-189.

68. Tornay S: The Omo Murle enigma, in Bender ML (ed): *Peoples and Cultures of the Ethio-Sudan Borderlands.* East Lansing, Mich, African Studies Center, Michigan State University, 1981, pp 33-78.

69. Tornay S: The Nyangatom: An outline of their ecology and social organization, in Bender ML (ed): *Peoples and Cultures of the Ethio-Sudan Borderlands.* East Lansing, Mich, African Studies Center, Michigan State University, 1981, pp 137-178.

70. Almagor U: Institutionalizing a fringe periphery: Dassanetch-Amhara relations, in Donham D, James W (eds): *The Southern Marches of Ethiopia.* Cambridge, Cambridge University Press, 1986, pp 96-115.

71. Garretson PP: Vicious cycles: Ivory, slaves, and armies on the new Maji frontier, in Donham D, James W (eds): *The Southern Marches of Ethiopia.* Cambridge, Cambridge University Press, 1986, pp 196-218.

72. Cerulli E: *Peoples of South-West Ethiopia and Its Borderland.* London, International African Institute, 1956.

73. Hogg R: Development in Kenya: Drought, desertification and food scarcity. *African Affairs* 1987; 86:47-58.

74. Letter from Monty Brown to Pascal James Imperato, MD, Naro Moru, Kenya, March 2, 1987.

75. Letter from A. Abell to Pascal James Imperato, MD, Nairobi, Kenya, April 29, 1987.

76. Letter from Edward Rodwell to Pascal James Imperato, MD, Mombasa, Kenya, April 30, 1987.

77. Pern S: *Another Land, Another Sea. Walking 'Round Lake Rudolf.* London, Victor Gollancz Ltd, 1979.

78. Letter from Stephen Pern to Pascal James Imperato, MD, Ore Hastings, England, March 15, 1987.

79. Amin M: *Cradle of Mankind.* London, Chatto and Windus, 1981.

80. Letter from John J.W.F. McFadden to Pascal James Imperato, MD, Rittenhouse Club, Philadelphia, Pa, March 11, 1986.

81. Letter from Christine Kelly, Archivist, Royal Geographical Society, London, to Pascal James Imperato, MD, June 30, 1986.

82. McFadden JJWF: *History of the Rittenhouse Club.* Philadelphia, Pa, unpublished (courtesy of the author).

83. Pleasants H Jr: Some horses are lucky. Biographical sketch of Arthur Donaldson Smith, MD. *The General Magazine and Historical Chronicle*, University of Pennsylvania General Alumni Society, XLVIII, 1946; 4:254-261 (courtesy of H. Spencer Potter, PhD).

84. Ingersoll E: *As Explorers Go.* New York, Explorers Club, 1937.

85. Photo insert, 1909 Dinner at the Waldorf Astoria. *Explorers' J* 1980; 58.

86. Harvard doctor cured the Shah. Dr. William A. Smith honored by Persian monarch for his service. *Boston Chronicle*, September 10, 1904 (courtesy of The University Archives, Harvard University Library).

87. Smith WL: The cave tiger of Amoy. The story of a hunt for a man-eating tiger that lived in a rocky den back of Amoy. *Natural History* 1928; 28:430-438.

88. Dr. William Lord Smith. *San Francisco Chronicle*, October 5, 1904. (courtesy of the University Archives, Harvard University Library).

89. Roosevelt T: *African Game Trails. An Account of the Wanderings of an American Hunter-Naturalist.* New York, Charles Scribner's Sons, 1910, p 404.

90. Dr. William L. Smith. *Malden Mirror*, February 19, 1910 (courtesy of the University Archives, Harvard University Library).

91. Personal communication, H. Spencer Potter, PhD, East Northport, NY, September 30, 1986.

92. Philadelphians as consuls. *Public Ledger*, April 27, 1909.

93. *Diplomatic and Consular Service of the United States. Corrected to April 20, 1909.* Washington DC, 1909, p 35 (courtesy of the Library, Department of State).

94. *Diplomatic and Consular Service of the United States. Corrected to November 9, 1909.* Washington, DC, 1909, p 28 (courtesy of the Library, Department of State).

95. *Nagel's Encyclopedia-Guide, Turkey.* Geneva, Nagel Publishers, 1974, pp 618-622.

96. Rossiter S: *Blue Guides. Greece.* London, Ernest Bern, 1981, pp 353-356.

97. *Diplomatic and Consular Service of the United States. Corrected to July 15, 1910.* Washington, DC, 1910, p 28 (courtesy of the Library, Department of State).

98. Letter from Bruno Newman to the Harvard Alumni Association, Aguascalientes, Mexico, February 6, 1911. Arthur Donaldson Smith File, University Archives, Harvard University Library, Boston, Mass.

99. Alba V: *The Horizon Concise History of Mexico.* New York, American Heritage Publishing Co, 1973, pp 124-129.

100. *Fodor's Mexico 1987.* New York, Fodor's Travel Guides, 1986, pp 226-229.

101. A. Donaldson Smith in Philadelphia *Social Register.* New York, Social Register Association, 1912, p 824.

102. Letter from A. Donaldson Smith to Dr E. Gordon, Kaolin, Pa, December 11, 1914. University of Pennsylvania Museum Archives.

103. *American Medical Directory*, ed 7, Chicago, Ill, American Medical Association, 1921.

104. Personal communication, H. Spencer Potter, PhD, East Lansing, Mich, March 28, 1986.

105. Personal communication, H. Spencer Potter, PhD, Manhasset, NY, July 7, 1986.

106. *American Medical Directory*, ed 8. Chicago, Ill, American Medical Association, 1923.

107. *American Medical Directory*, ed 13. Chicago, Ill, American Medical Association, 1934.

108. *American Medical Directory*, ed 9. Chicago, Ill, American Medical Association, 1925.

109. *American Medical Directory*, ed 11, Chicago, Ill, American Medical Association, 1929.

110. *Historical Notes in the Development of Potter County*. Coudersport, Pa, Potter County Commissioners, 1949 (courtesy of Mr Robert Currin, President, Potter County Historical Society).

111. Personal communication, Mrs Bonnie Darrin, Coudersport, Pa, April 22, 1987.

112. *American Medical Directory*, ed 1. Chicago, Ill, American Medical Association, 1906.

113. *Pennsylvania. A Guide to the Keystone State*. New York, Oxford University Press, 1940, reprinted 1963.

114. Personal communication, Mrs Alice Morey, Roulette, Pa, April 8, 1986.

115. Personal communication, Mrs Sandy Stake, Port Allegany, Pa, April 6, 1986.

116. Personal communication, Mr Robert Currin, President, Potter County Historical Society, Coudersport, Pa, April 8, 1986.

117. Personal communication, Mrs Amy Baker, Roulette, Pa, April 24, 1987.

118. Personal communication, Mrs Barbara O'Connor, Port Allegany, Pa, May 30, 1986.

119. Personal communication, Alfred A. Imperato, MD, Wantagh, NY, May 1, 1987.

120. *Golden Memories 1905–1954*, Roulette, Pa, Journalism Club of Roulette Township High School, 1954 (courtesy of Dr Frank A. Rackish).

121. Personal communication, Franklyn Baxter, Roulette, Pa, April 15, 1987.

122. Peterson HL: *Pageant of the Gun*. Garden City, NY, Doubleday and Co, 1967, pp 256–261.

123. Personal communication, Mr Eric Rodgers, Scotland Neck, NC, June 12, 1984.

124. Noted Explorer, Visitor Succumbs. Dr. A. D. Smith Had Brilliant Career. *St Petersburg Times*, February 22, 1939 (courtesy of D. Strother Pope, MD).

125. Certificate of Death, Arthur Donaldson Smith, Philadelphia, Pa, File No. 13166, February 21, 1939 (obtained July 20, 1978).

126. Letter from Charles A. Potter, Jr, to Dr Herbert B. Carpenter, Philadelphia, Pa, February 24, 1939 (courtesy of H. Spencer Potter, PhD).

127. Letter from Herbert B. Carpenter, MD, to Charles A. Potter, Jr, Philadelphia, Pa, February 21, 1939 (courtesty of H. Spencer Potter, PhD).

128. Dr. Arthur Smith Dies at Age of 74. Famous Explorer Who Practiced at Roulette Following the War. *The Potter Enterprise*, February 23, 1939 (courtesy of the Coudersport Public Library).

129. Obituary. Dr. A. Donaldson Smith. *Geographical J* 1939; xciii:464.

130. Letter from Peter Kerch, Superintendent, Laurel Hill Cemetery, to Pascal James Imperato, MD, Philadelphia, Pa, November 9, 1986.

131. *Jonathan Kenworthy—Bronzes and Drawings. People of the Desert: Nomads of East Africa*. New York, Coe Kerr Gallery, 1985.

Additional Bibliography

A. Donaldson Smith exploration described. *NY Times*, November 6, 1895, p 6.

A. Donaldson Smith exploration described. *NY Times*, December 24, 1895, p 13.

African collection presented to the Philadelphia Academy of Natural Sciences. *NY Times*, May 24, 1896, p 26.

African explorer's arrival in New York. *NY Times*, December 2, 1900, p 14.

Acknowledgments. In September 1961, as I stood on the top of Mount Marsabit in northern Kenya and looked down on the crater holding Lake Paradise, I wondered who had been the first person to describe this place to the outside world. Shortly thereafter I found the answer by chance, in Elspeth Huxley's superb classic history of Kenya, *White Man's Country*. She related that Dr Arthur Donaldson Smith was probably the first European to visit Marsabit. In seeking to learn more about him I also had to learn about northern Kenya, Ethiopia, and Somalia, a discovery process that has never ended and one that has already spanned a quarter of a century. Ultimately this inquiry was conducted on three continents and brought me into contact not only with the history of this part of Africa but also with its peoples, fauna, flora, and political development.

It was my good fortune to live and work in Africa for six years and to return to the land Smith had explored so thoroughly. Discovering the Africa he knew proved easier than discovering details about his personal and professional lives, however, since he left no collection of personal papers, except letters to the Royal Geographical Society. The details of his life had to be methodically pieced together from a multiplicity of sources, an arduous process that required much time. Presenting Smith and his accomplishments without the larger geopolitical canvas against which he moved in Africa and without a full account of the other explorers who preceded and followed him would have been to tell only part of the story. This series presents the larger tale and as such is more than just a biography of Smith. While Smith's life and career are the central theme, the series comprises a detailed account of the exploration of Lake Rudolf from 1887 until 1910.

There are many individuals and institutions who have graciously helped me in this effort and to all of them I extend my sincere thanks. I am especially thankful to H. Spencer Potter, PhD, of East Lansing, Michigan, and Holmes Beach, Florida, Dr Smith's grand-nephew, for his support, interest, and valuable assistance. John Winthrop Aldrich of Barrytown, New York, kindly gave me access to the William Astor Chanler Papers at Rokeby as well as to valuable materials concerning Ludwig von Höhnel. I am also very thankful to him for carefully reading the draft of the section on Chanler and for making useful suggestions concerning it. My attempts to learn about Smith's years in Roulette, Pennsylvania, were stalled for many years until by a stroke of luck I mentioned Smith to John T. Flynn, MD, Consulting Editor of the *Journal*. A native of Pennsylvania, he immediately knew of Roulette and put me into contact with a lifelong friend, the Reverend W. Robert Griffin, Pastor of St Eulalia's Church in Coudersport, Pa. Without Father Griffin's constant support and interest this phase of the research could never have been carried out. I am grateful to him for introducing me to many helpful people in Roulette and elsewhere who knew Smith, and for his sincere concern for the success of my research.

This research effort required studying documents in five languages: English, French, Italian, German, and Hungarian. My own fluency in the first two and passable knowledge of the third assured half the battle. I am grateful to my wife, Eleanor, for translating a number of Italian language documents and for her support and understanding during the long period this project was underway. André Szabo of Raanana, Israel, a close friend and an old Africa hand, translated an enormous number of Hungarian documents into French for me, including Count Teleki's personal letters to Crown Prince Rudolf. I am extremely grateful to him for this. I want to thank Tibor Fodor, MD, of New York, who translated several Hungarian publications into English. I am very thankful to Inge Casstrissiades of Port Washington, NY, for translating a number of German documents into English.

Luckily, Frank Strovas, an old friend from my West Africa days, happened to be stationed at the US Embassy in Bucharest, Romania, as Public Affairs Officer and visited Count Teleki's former estate in Transylvania and relayed to me much useful information about it. Through him I was put into contact with Erdelyi Lajos, Count Teleki's biographer, of Tirgu Mures, Romania, who graciously sent me valuable photographs of the count and his estate from a long-overlooked archive. Erdelyi's excellent biography of Count Teleki provides much important new information on the expedition that led to the discovery of Lake Rudolf. I am especially thankful to D. Strother Pope, MD, of Columbia, SC, who has travelled extensively through much of the Africa Smith explored. Dr Pope provided me with much help and encouragement and was of great assistance in elucidating the Wyndlawn Caves Smith had discovered in Ethiopia and in uncovering information about Smith's life in Scotland Neck, NC, and St Petersburg, Fla.

I am very grateful to John J. W. F. McFadden of Philadelphia who put me in contact with Smith's grand-nephew, H. Spencer Potter, PhD. He also researched Smith's association with the Rittenhouse Club and graciously gave me access to his unpublished history of the club. I thank Robert Currin, President of the Potter County Historical Society, whose research efforts also greatly assisted me. Christine Kelly, Archivist of the Royal Geographical Society, performed a herculean task in collecting relevant letters and documents and I am most grateful to her and to the Society for their valuable help. Dr Anna Benna, Director of the Osterreichsches Staatarchiv, Vienna, graciously sent me copies of correspondence from and about Count Samuel Teleki and about Ludwig von Höhnel as well as copies of secondary source material concerning Count Teleki which proved extremely valuable. I am especially grateful to Elizabeth Somers, Secretary and Administrative Assistant, *New York State Journal of Medicine*, for carefully preparing the typescripts and proofing them, and to Carol Moore, Managing Editor of the *Journal*, for carefully copyediting and laying out the articles. I also want to express my sincere gratitude to the following individuals and institutions for their help and apologize for any names I have overlooked:

Alec and Alison Abell, Nairobi, Kenya; Ella Abney, Librarian, Library of the Medical Society of the State of New York, Lake Success, NY; Amy Baker, Roulette, PA; Robert Baker, Cultural Affairs Officer, US Consulate, Sydney, Australia; Franklyn Baxter, Roulette, PA; Professor P. T. W. Baxter, Department of Social Anthropology, University of Manchester, Manchester, England; Monty Brown, Naro Moru, Kenya; Dolores Buchsen, The Pennsylvania Lumber Museum, Coudersport, PA; Professor Karl W. Butzer, Department of Geography, University of Texas, Austin, TX; Beth Carroll Horrocks, Assistant Manuscripts Librarian, American Philosophical Society, Philadelphia, PA; Sister Antoinette Casertano, Daughters of Mary Help of Christians, Haledon, NJ; Sister Martha Casertano, Daughters of the Heart of Mary, Holyoke, MA; William Astor Chanler, Jr, Camden, ME; William Chamberlain Chanler, New York, NY (deceased); City Archives, Philadelphia, PA; Clerk of the Orphans' Court Division, Court of Common Pleas of Philadelphia County, Philadelphia, PA; Coudersport Public Library, Coudersport, PA; Bonnie Darrin, Coudersport, PA; William G. Dixson, Nairobi, Kenya; Cynthia Downey, Nairobi, Kenya; Sydney Downey, Nairobi, Kenya (deceased); Henry S. Evans, Editor, *The Explorers Journal*, Morristown, NJ; Dr Lidia Ferenczy, National Szechenyi Library, Budapest, Hungary; Free Library of Philadelphia, Philadelphia, PA; Ann E. Frontera-Rial, Arlington, VA; George E. Galvin, Jr, Albuquerque, NM; Georgette Garner, Washington, DC; John D. Garner, Washington, DC; General Society of the Sons of the Revolution; Ruxton, MD; Madeleine Gosselin, Curatorial Assistant, Harvard University Archives, Cambridge, MA; Rev Pierre Gouin, SJ, Montreal, Canada; Haile Selassie I University, Addis Ababa, Ethiopia; Gerard A. Imperato, Esq, Fresh Meadows, NY; Maryellen C. Kaminsky, Archival Specialist, University of Pennsylvania Archives, Philadelphia, PA; Dr Karl Kammel, Osterreichifche Naturialbiblothek, Vienna, Austria; Peter Kerch, Superintendent, Laurel Hill Cemetery, Philadelphia, PA; Library of the American Medical Association, Chicago, IL; Library of the American Museum of Natural History, New York, NY; Library of the College of Physicians of Philadelphia, Philadelphia, PA; Library of Congress, Washington, DC; Library, Department of State, Washington, DC; Library of the New York Academy of Medicine, New York, NY; Library of the New York Historical Society, New York, NY; Library of the State University of New York Health Science Center at Brooklyn, Brooklyn, NY; Manhasset Public Library, Manhasset, NY; Marsabit District, Administrative Authorities, Kenya; McMillan Library, Nairobi, Kenya; Medical Illustration and Photography Units, Department of Biomedical Communications, State University of New York Health Science Center at Brooklyn, Brooklyn, NY; Rev Frans Mol, Mill Hill Fathers, Lemek, Narok, Kenya; Alice Morey, Roulette, PA; George C. Mosch, MD, Secretary-Treasurer, Potter County Medical Society, Coudersport, PA; Moyale District, Administrative Authorities, Kenya; National Council for Science and Technology, Nairobi, Kenya; National Society of the Sons of the American Revolution, Louisville, KY; New York Public Library, New York, NY; Northern Frontier District Administrative Authorities, Kenya; Barbara O'Connor, Port Allegany, PA; Oxford University Press, Oxford, England; Bernard Pasqualini, Microforms and Newspapers Department, The Free Library of Philadelphia, PA; Stephen Pern, Ore Hastings, England; Sister Marguerite Petraz, Daughters of the Heart of Mary, Montreal, Canada; Port Washington Public Library, Port Washington, NY; Dr Frank A. Rackish, Executive Director, Seneca Highlands Intermediate Unit of Education, Smethport, PA; Lady Alys Reece, Bolton Old Manse, Scotland; Lord Rennell of Rodd, London, England; Eric Rodgers, Scotland Neck, NC; Edward Rodwell, Mombasa, Kenya; Mary Elizabeth Ruwell, Archivist, The University Museum, University of Pennsylvania, Philadelphia, PA; Charles Schille, Curatorial Assistant, Harvard University Archives, Cambridge, MA; Carol M. Spawn, Manuscript/Archives Librarian, The Academy of Natural Sciences of Philadelphia, Philadelphia, PA; Franz Spiegelfeld, Vienna, Austria; Sandy Stake, Port Allegany, PA; Robert J. States, All Saint's Church, Philadelphia, PA; James Stimpert, Assistant Archivist, The Ferdinand Hamburger Jr Archives, The Johns Hopkins University, Baltimore, MD; Genell Subak-Sharpe, New York, NY; Grayel Tauscher, Tioga, PA; Janos Teleki, Vienna, Austria; Karoly Teleki, Vienna, Austria; A. J. Troughear, Educational Media Service, Nairobi, Kenya; Errol Trzebinski, Mombasa, Kenya; Professor David Turton, Department of Social Anthropology, University of Manchester, Manchester, England; Barbara Wilson, Archivist, The University Museum, University of Pennsylvania, Philadelphia, PA.

Group B streptococcal osteomyelitis in an adult

ALEXANDER A. MCMEEKING, MD; ROBERT HOLZMAN, MD; DIANE DESIDERIO, MS;
BRUCE HANNA, PHD

Group B streptococcus has been recognized as a serious pathogen in neonates since the mid-1960s.[1] In the past ten years, several reviews have reported this organism to be a cause of serious infections in adults as well.[2,3] In women, group B streptococcal infections usually occur around the time of parturition.

In spite of this wider recognition, group B streptococcus remains an unusual pathogen and a rare cause of osteomyelitis in adults, with only nine prior cases reported in the world literature to our knowledge. We describe a patient with group B streptococcal vertebral osteomyelitis possibly secondary to a urinary tract infection.

CASE REPORT

The patient, an 82-year-old woman, was admitted to Bellevue Hospital on March 4, 1986, with a chief complaint of progressive lower back pain radiating down both legs. She had a history of hypertension and diabetes mellitus which had been controlled by diet alone. Three months prior to admission she had been treated with antibiotics by a private physician for a presumed urinary tract infection manifested by dysuria, urgency, and frequency. No urine culture had been obtained prior to therapy.

The patient had experienced fevers, night sweats, and a dull pain in her lower back radiating to both legs for one month prior to admission. She denied any injury to the back, weight loss, incontinence of bowel or bladder, or weakness or paresthesia in her legs. She had no known history of tuberculosis or exposure to tuberculosis.

At the time of admission the patient was afebrile and in no distress. Positive physical findings included a soft apical systolic heart murmur, mild tenderness to percussion over the lumbar spine, normal rectal sphincter tone, and a decreased right patellar reflex without other neurologic defects.

A white blood cell count was 12,900/mm^3 with normal differential; hematocrit, 32%; mean corpuscular volume, 82 μm^3; platelet count, 546,000/mm^3, and erythrocyte sedimentation rate, 78 mm/h. Serum electrolyte levels were normal, as were the creatinine and blood urea nitrogen levels. A fasting serum glucose level was 170 mg/dL. Urinalysis showed no red blood cells, white blood cells, protein, or glucose. The chest film was normal; roentgenograms of the lumbar spine showed destructive lesions at the bottom of L3 and top of L4 with a narrowed interspace, consistent with osteomyelitis. A computed tomogram confirmed this finding and showed no sign of a paravertebral abscess. An abdominal sonogram showed normal kidneys, liver, and spleen.

Cultures of blood and urine were sterile. Cultures of a needle aspirate of L4, performed under fluoroscopic guidance, grew a streptococcus, which was resistant to bacitracin, was unable to ferment mannitol or grow in 6.5% sodium chloride broth, and which demonstrated moderate beta hemolysis on sheep blood agar plates. When serotyped by the direct colony method for coagglutination, an immediate agglutination was apparent in the presence of the group B antiserum, while no reaction occurred with group A, C, or G antisera. Bone biopsy cultures for tuberculosis were negative, and pathologic specimens failed to show granulomas or acid-fast bacilli.

The patient received penicillin G intravenously, 2 million units every four hours for six weeks, followed by a four-week course of oral penicillin, 500 mg every six hours. Her back pain resolved over four weeks, and repeat radiographs prior to discharge showed no progression of disease. A repeat erythrocyte sedimentation rate on April 13, 1986, was 35 mm/h. Other tests performed during the patient's hospital stay included an echocardiogram, which was normal, and a barium enema. The patient was seen in a medical clinic several months later and continued to be in good health.

DISCUSSION

The incidence of group B streptococcal infections in pediatric populations has increased over the past 20 years. In neonates, these infections are now a leading cause of meningitis, bacteremia, and osteomyelitis.[5,6] Group B streptococcus has also been recognized as an important pathogen in adults. It is most frequently isolated from cultures of urine and from the female genital tract and is often associated with puerperal infections such as pyelonephritis, chorioamnionitis, and endometritis.[2,3] Group B streptococcus is also reported as a rare cause of pneumonitis, meningitis, endocarditis, septic arthritis, cellulitis, and other soft tissue infections.[3,4]

A review of the literature (1966–1986) yielded only nine reported cases of group B streptococcal osteomyelitis in adults. Peripheral vascular disease, orthopedic surgery, or concomitant infection such as frontal sinusitis predisposed to development of osteomyelitis in most cases.[7,8]

In only two cases was a hematogenous source proposed. McKee[9] described an 18-year-old patient with diabetes who had osteomyelitis of the left humerus, a group B streptococcal urinary tract infection, and sterile blood cultures. The infection was likely to have originated from the urinary tract or to have spread contiguously from an insulin injection site. Gordon and Oster[10] described a 55-year-old male alcoholic patient with osteomyelitis of the lumbar spine with no obvious source of infection.

The patient described here had no apparent concomitant infection such as endocarditis or urinary tract or pelvic

---

From the Division of Infectious Diseases, Department of Medicine (Drs McMeeking and Holzman), and the Department of Pathology (Ms Desiderio and Dr Hanna), Bellevue Hospital Center of New York University.

Address correspondence to Dr McMeeking, Department of Medicine, Division of Infectious Diseases, New York University Medical Center, 550 First Ave, New York, NY 10016.

infection at the time of admission. A barium enema revealed no evidence of bowel malignancy, and an abdominal sonogram showed no sign of perinephric abscess. It is possible, however, that the patient's urinary tract infection three months prior to admission was caused by group B streptococci and spread through Batson's venous plexus to her spine.

Most isolates of group B streptococci are sensitive to penicillin, although a few have been reported to show penicillin tolerance.[11] The clinical significance of this is unclear. This patient responded well to penicillin, as was generally the case in the prior reports in the literature. In some cases, however, patients have also required surgical intervention.

Patients with diabetes are more susceptible to group B streptococcal infections, although the reasons for this are poorly understood.[3,4] While this patient did not have signs of peripheral neuropathy or peripheral vascular disease, she did have fasting hyperglycemia on several repeated serum samples (range, 119–170 mg/dL). It is possible that mild diabetes mellitus predisposed this patient to a group B streptococcal infection.

Although 70–80% of group B streptococcal isolates grow in the presence of 6.5% sodium chloride,[12] this patient's isolate did not grow. This finding combined with beta hemolysis on blood agar led the microbiology laboratory personnel to use serotyping to identify the organism. In this laboratory, when an unequivocal reaction is observed with the direct plate grouping method using the Phadebact Streptococcus test kit, no further evaluations are performed. In a previously reported evaluation of this technique, as compared to the Lancefield procedure, 100% of group B streptococcal isolates were correctly identified.[13] Since a positive result occurred for this isolate with only the group B streptococcal antiserum, no further confirmatory evaluations—such as bile esculin reaction or hippurate hydrolysis—were considered necessary.

In this patient, the lack of an identifiable contiguous focus and the location of osteomyelitis would seem to make a hematogenous route of infection most likely. Adult osteomyelitis secondary to group B streptococcus remains rare but must be considered in the differential diagnosis, especially in patients with diabetes mellitus.

REFERENCES

1. Eickhoff TC, Klein JO, Daly AK, et al: Neonatal sepsis and other infections due to group B beta-hemolytic streptococci. *N Engl J Med* 1964; 271:1221–1228.
2. Anthony BF, Concepcion NF: Group B streptococcus in a general hospital. *J Infect Dis* 1975; 132:561–577.
3. Bayer AS, Chow AW, Anthony BF, et al: Serious infections in adults due to group B streptococci. Clinical and serotypic characterization. *Am J Med* 1976; 61:498–503.
4. Dworzack DL, Hodges GR, Barnes WG, et al: Group B streptococcal infections in adult males. *Am J Med Sci* 1979; 277:67–73.
5. Baker CJ: Summary of the workshop on perinatal infections due to group B streptococcus. *J Infect Dis* 1977; 136:137–152.
6. Edwards MS, Baker CJ, Wagner ML, et al: An etiologic shift in infantile osteomyelitis. The emergence of the group B streptococcus. *J Pediatr* 1978; 93:578–583.
7. McGuire T, Gerjarusak P, Hinthorn DR, et al: Osteomyelitis caused by beta-hemolytic streptococcus group B. *JAMA* 1977; 238:2054–2055.
8. Stevens DL, Haburchak DR, McNitt TR, et al: Group B streptococcal osteomyelitis in adults. *South Med J* 1978; 71:1450–1451.
9. McKee KT Jr: Osteomyelitis due to group B streptococcus in a diabetic adolescent. *J Pediatr* 1978; 92:677–678.
10. Gordon DM, Oster CN: Hematogenous group B streptococcal osteomyelitis in an adult. *South Med J* 1984; 77:643–645.
11. Kim KS, Anthony BF: Penicillin tolerance in group B streptococci isolated from infected neonates. *J Infect Dis* 1981; 144:411–419.
12. Facklam RR, Padula JF, Thacker LG, et al: Presumptive identification of group A, B and D streptococci. *Appl Microbiol* 1974; 27:107–113.
13. Slifkin M, Engwall C, Pouchet GR: Direct-plate serological grouping of beta-hemolytic streptococci from primary isolation plates with the Phadebact streptococcus test. *J Clin Microbiol* 1978; 7:356–360.

See also pp 428–430.

Unusual thoracic myelopathy in two patients with Hodgkin disease

ROBERT L. RUFF, MD, PhD, ROBERT SCHELL, MD

Spinal cord dysfunction occurs in approximately 7.5% of patients with Hodgkin disease.[1] In 20% of these cases the spinal lesion is the presenting manifestation of the disease.[2] We describe two patients with Hodgkin disease who presented with painful myelopathy. The clinical course and radiologic findings in these cases suggested that direct spinal cord compression may not have been the cause of the myelopathy. Computed tomography (CT) was useful in imaging paravertebral lymphoma that may have contributed to the spinal cord dysfunction.

CASE REPORTS

Case 1. In August 1978, a 32-year-old man experienced posterior mid-thoracic pain brought on by bending forward or by the Valsalva maneuver; numbness and incoordination of the lower extremities; and urinary urgency. He had a spastic paraparesis and decreased pinprick sensation below T_8, but position and vibration sensation were preserved. He did not have percussion tenderness over the spine or lymphadenopathy. An iophendylate myelogram with prone, supine, and both lateral decubitus views was repeatedly normal. The complete blood count, antinuclear antibody levels, serum electrolyte concentrations, and serum protein electrophoresis studies were within normal limits. The cerebrospinal fluid (CSF) cell count and protein and glucose concen-

---

From the Departments of Neurology, Cleveland and Seattle Veterans Administration Hospitals, and Case Western Reserve Medical School (Dr Ruff), Cleveland, Ohio, and Medical Service, Coney Island Hospital (Drs Ruff and Schell), Brooklyn, NY.

Address correspondence to Dr Ruff, Chief, Neurology Service 127(W), Cleveland Veterans Administration Medical Center, 10701 East Blvd, Cleveland, OH 44106.

trations were also within normal limits, with gamma globulin constituting 2.7% of the total CSF protein.

Within several weeks the patient's pain and spasticity had subsided, and his coordination and urinary urgency improved. The numbness in the lower extremities persisted. In June 1979, he had a three-week episode of back pain and lower extremity incoordination and spasticity. Symptoms and signs were similar to those of the initial episode. The pain and ataxia resolved in several weeks. In February 1980, the patient had a third episode of severe thoracic back pain and spastic paraparesis. Pain now increased with neck flexion. Repeat iophendylate prone and supine myelograms were again reported to be normal. The CSF protein and glucose concentrations and cell count were also normal, but since the gamma globulin was 17% of the total CSF protein, a diagnosis of multiple sclerosis was considered. The patient's condition improved spontaneously over several months and he returned to normal activity, though the numbness in his lower extremities persisted.

The fourth episode of painful myelopathy occurred in June 1980. The pain was in the same location as before, but was now associated with the Lhermitte sign. The patient had lower extremity ataxia, spastic paraparesis with nocturnal extensor spasms, urinary urgency, and impotence. Because his condition failed to improve, he was admitted to the Seattle Veterans Administration Hospital in August 1980.

There was no history of back injury, fevers, chills, skin rash, arthralgia, sweats, or weight loss. The patient had numerous firm, matted, supraclavicular, cervical, and axillary lymph nodes. His skin and extremity joints were normal. Percussion at T_8 produced pain, which radiated to the anterior chest. He had a mild spastic paraparesis. Pinprick was poorly felt below T_6, and vibration sensation was diminished below T_{10}. Proprioception in the lower extremities was impaired. The patient had hyperactive lower extremity reflexes with bilateral Babinski signs and ankle clonus. Cutaneous abdominal and cremasteric reflexes were absent. His gait was spastic with a positive Romberg sign. Anal sphincter tone was diminished. The complete blood count, serum electrolyte concentrations, VDRL, antinuclear antibody levels, erythrocyte sedimentation rate, serum B_{12} concentration, and liver function tests were within normal limits. A chest film showed mediastinal adenopathy.

Iophendylate myelography demonstrated an extradural defect extending from T_3 through T_9, displacing the spinal cord slightly from left to right, as well as slight enlargement of the spinal cord outline from T_4 to T_6 (Fig 1). The opening pressure was 110 mm CSF. The CSF protein concentration was 78 mg/dL; there was less than one white blood cell/mm^3; cytologic studies were negative, and oligoclonal bands were not present. A thoracic CT scan delineated a posterior mediastinal mass overlying the vertebral column from T_4 to T_8 (Fig 1). An abdominal CT scan demonstrated moderate para-aortic adenopathy. Brainstem auditory and visual evoked responses were normal. Supraclavicular biopsy revealed nodular sclerosing Hodgkin lymphoma.

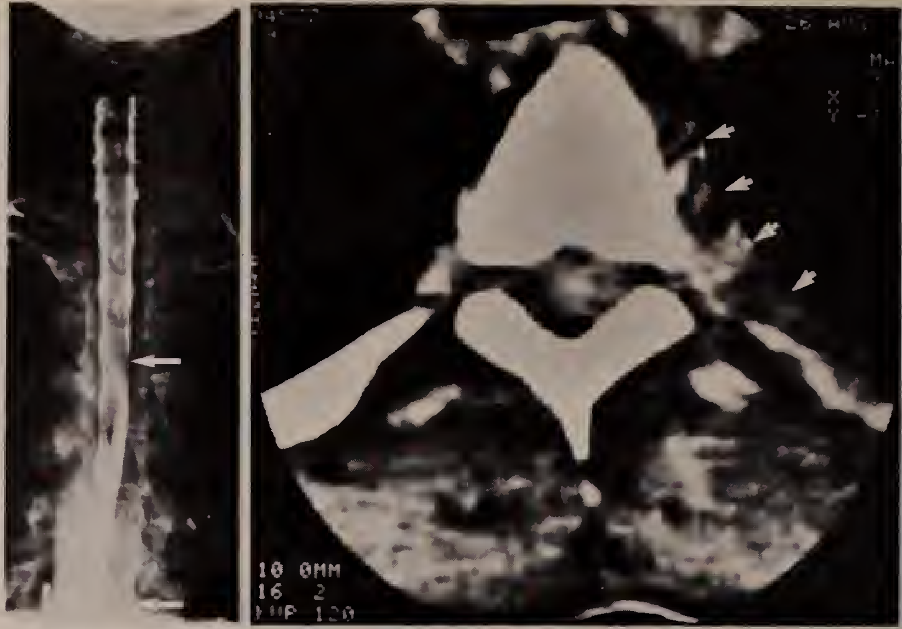


FIGURE 1. Radiologic studies from Case 1. **Left**, iophendylate myelogram showing slight displacement of the subarachnoid space from left to right from T_3 through T_8 (arrows) and an enlarged spinal cord outline from T_4 to T_6. **Right**, CT scan at the level of T_8 demonstrating a posterior mediastinal soft tissue mass on the left (arrows).

The patient was treated with 100 mg of intravenous dexamethasone, with rapid improvement of his painful myelopathy, and he began chemotherapy consisting of mechlorethamine, vincristine, procarbazine, and prednisone. Radiation therapy was withheld in view of the need for chemotherapy and the continued improvement in the patient's condition. Within three months, he was free of pain. His strength returned to normal; his spasticity was reduced, and his bladder and bowel symptoms resolved. His sensation improved, and his mediastinal adenopathy decreased. Myelopathy had not recurred to the time of his death in November 1986.

Case 2. In 1983, a 28-year-old man suddenly experienced mid-thoracic pain, paraparesis, and urinary hesitancy. The pain projected anteriorly and was increased with the Valsalva maneuver but not with spinal percussion. The patient had flaccid paraparesis, a T_6 sensory level to all modalities, and decreased anal sphincter tone, and cutaneous abdominal and cremasteric reflexes were absent. There was no palpable lymphadenopathy. The skin and joints were normal. Thoracic spine radiograms, a prone and supine iophendylate myelogram (Fig 2), and laboratory tests including complete blood count, erythrocyte sedimentation rate, antinuclear antibody levels, and serum protein electrophoresis, serum electrolyte concentrations, and liver function tests were within normal limits. A CSF evaluation revealed a protein level of 58 mg/dL, and one white blood cell/mm^3. On cytologic analysis, no malignant cells and no oligoclonal bands were found. CT demonstrated a small mass in the left posterior mediastinum from T_3 to T_4 (Fig 2). Mediastinal biopsy revealed nodular sclerosing Hodgkin lymphoma. The patient was treated with high-dose dexamethasone according to the protocol of Greenberg et al[3] and was then started on chemotherapy with mechlorethamine, vincristine, procarbazine, and prednisone. The myelopathy resolved over two weeks and did not recur during the subsequent three years.

DISCUSSION

In these cases, painful myelopathy developed without significant spinal cord compression or displacement on myelography. The causes of these myelopathies have not been determined. In most patients with lymphoma, myelopathy is caused by epidural compression.[1,2,4,5] In Case 1, the initial two myelograms failed to demonstrate an epidural mass, and the third myelogram showed a left-sided epidural mass extending from T_3 to T_8. Possibly, the epidural lesion was too small to be detected by the earlier myelograms. Myelography did not demonstrate an epidural lesion in Case 2. Consequently, the myelopathy in Case 2 and possibly the initial episodes of myelopathy in Case 1 did not appear to be caused by epidural spinal cord compression. Multiple sclerosis was initially considered in Case 1. Occasionally, the central nervous system complications of lymphoma may have a temporal course characterized by exacerbations and remissions, suggesting a diagnosis of multiple sclerosis.[6,7] However, physical examination and evoked response testing did not suggest injury to the central nervous system outside of the spinal cord, and immunological studies of the CSF also did not support a diagnosis of multiple sclerosis. Vasculitis can produce myelopathy; however, neither patient had laboratory or dermatologic evidence of systemic vasculitis. Transverse myelitis has not been recognized as a paraneoplastic complication of Hodgkin lymphoma. Paraneoplastic subacute myelitis usually has a steadily progressive course and does not respond to treatment of the primary malignancy.[8]

In both cases computed tomography demonstrated paravertebral lymphoma that anatomically correlated with the levels of spinal cord dysfunction. The relationship of the paravertebral tumor to the spinal cord dysfunction is not clear. The myelopathies may have resulted from compromise of the spinal cord's vascular supply due to the paraspinal Hodgkin disease. Thoracic spinal cord segments one through four are very vulnerable to ischemia, because the radicular arterial supply to the thoracic cord is inconstant, and circumflex vessels uniting anterior and posterior spinal arteries are least numerous in this region.[9,10] Reduction in blood flow through a thoracic radicular artery could compromise the function of the anterior two thirds of the spinal cord,[9,10] creating paraparesis, loss of bladder and/or bowel control, and impaired pinprick sensation, as was seen in the initial episodes of myelopathy in Case 1. Alternatively, occlusion of a radicular artery may produce complete transverse ischemia of the cord over several segments, resulting in a clinical picture similar to that of Case 2. Spinal cord ischemia can produce radiating radicular pain and elevated CSF protein concentration without a CSF pleocytosis.[9,10] Ischemia can produce spinal cord edema, but a myelogram is normal in 20% of patients with spinal cord infarction.[10] Hodgkin disease can extensively involve intrathoracic lymph nodes,[11] laying the foundation for multilevel radicular artery compression, resulting in spinal cord vascular insufficiency. Bahgwah and McKissock[4] suggested that the myelopathy in some patients with Hodgkin disease may be caused by spinal cord ischemia rather than compression. Goldhahn and Goldhahn[12] described myelopathy in patients with Hodgkin disease without demonstrable compression or intramedullary spinal cord metastasis.

Myelopathy could also have resulted from direct extension of the paravertebral lymphoma to the spinal cord parenchyma via a spinal nerve root.[13] Intramedullary spinal cord metastases are rare. They account for only 1–5% of central nervous system parenchymal metastases. Only seven cases of intramedullary spinal cord metastasis associated with malignant lymphoma were noted in a review of the literature through 1978.[13] The clinical presentation of intramedullary spinal cord metastasis usually includes pain, weakness, autonomic dysfunction, and sensory loss. The presentation may resemble epidural spinal cord compression or spinal cord vascular insufficiency. CSF changes usually consist of elevated protein concentration, normal cell count, and no malignant cells.[13] The third myelogram in Case 1 showed a slight enlargement of the spinal cord outline from T_4 to T_6, which could have been consistent with an intramedullary metastasis. There did not appear to be any spinal cord enlargement in Case 2. Magnetic resonance imaging (MRI) and CT performed with a water soluble contrast agent in the subarachnoid space could have provided more precise imaging of the spinal cord to determine if an intramedullary mass was present. MRI may also have helped determine if spinal cord swelling was due to a tumor or to edema associated with ischemia. MRI of the thoracic spinal cord was not available at the time these cases were evaluated.

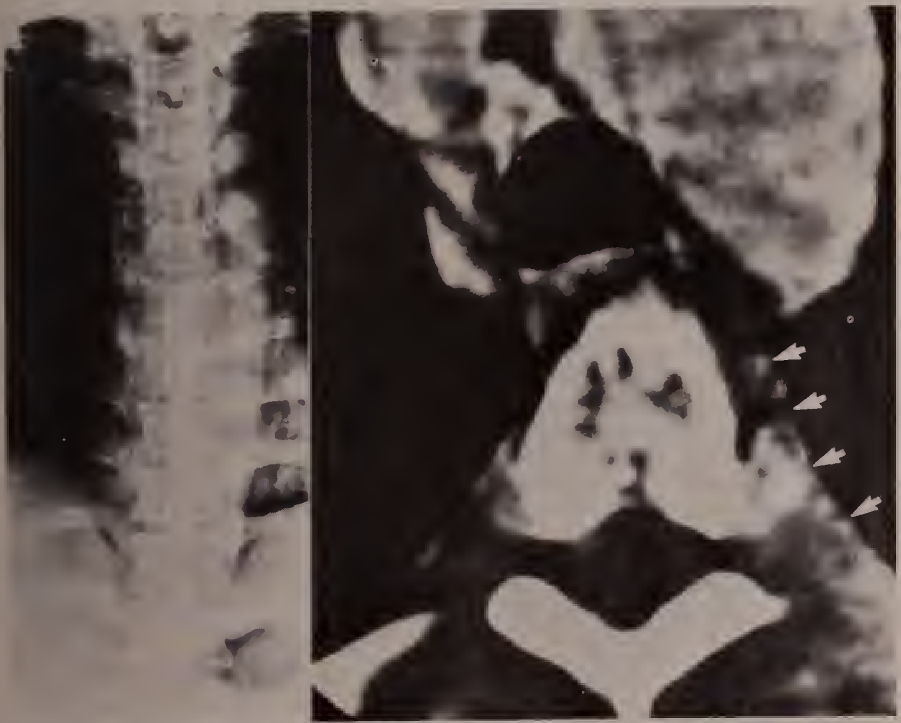


FIGURE 2. Radiologic studies from Case 2. **Left**, normal iophendylate myelogram. **Right**, CT scan at the level of T_4 demonstrating a posterior mediastinal soft tissue mass overlying the neural foramen on the left (arrows).

These two cases indicate that myelography alone may not be adequate to evaluate cases of painful thoracic myelopathy. If myelography is normal, CT or MRI at the appropriate level may detect tumors outside the spinal canal. CT with a water soluble contrast agent in the subarachnoid space or MRI may also indicate changes in spinal cord size or subtle extradural defects.

Acknowledgment. This study was supported by Merit Review Funding from the Veterans' Administration of the United States to Dr Ruff.

REFERENCES

1. Somasundaram M, Posner JB: Neurologic complications of Hodgkin's disease, in Lacher MJ (ed): *Hodgkin's Disease*. New York, John Wiley, 1976, pp 325–370.

2. Mullins GM, Flynn JP, el-Mahdi AM, et al: Malignant lymphoma of the spinal epidural space. *Ann Intern Med* 1971; 74:416–423.

3. Greenberg HS, Kim JH, Posner JB: Epidural spinal cord compression from metastatic tumor: Results with a new treatment protocol. *Ann Neurol* 1980; 8:361–366.

4. Bahgwah SN, McKissock W: Spinal cord compression in Hodgkin's disease: A review of 10 cases. *Brit J Surg* 1961; 48:672–676.

5. Ward JH, Kjeldsberg CR: Spinal cord compression in mycosis fungoides. *Cancer* 1982; 50:2510–2512.

6. Williams RS, Crowell RM, Fisher CM, et al:

Clinical and radiologic remission in reticulum cell sarcoma of the brain. *Arch Neurol* 1979; 36:206-210.

7. Ruff RL, Petito CK, Rawlinson DG: Primary cerebral lymphoma mimicking multiple sclerosis [letter]. *Arch Neurol* 1979; 36:598.

8. Spence AM, Sumi SM, Ruff RL: Paraneoplastic syndromes that involve the nervous system. *Curr Probl Cancer* 1983; 8:4-43.

9. Henson RA, Parsons M: Ischaemic lesions of the spinal cord: An illustrated review. *Quart J Med* 1966; 36:205-222.

10. Silver JR, Buxton PH: Spinal stroke. *Brain* 1974; 97:539-550.

11. Rostock RA, Giangreco A, Wharam MD, et al: CT scan modification in the treatment of mediastinal Hodgkin's disease. *Cancer* 1982; 49:2267-2275.

12. Goldhahn W-E, Goldhahn G: Neoplasms of the spinal epidural space with special reference to lymphogranulomatosis, in Vinken PJ, Bruyn GW (eds): *Handbook of Clinical Neurology*. Amsterdam, North Holland, 1976, vol 20, pp 103-136.

13. Jellinger K, Kothbauer P, Sunder-Plassmann E, et al: Intramedullary spinal cord metastasis. *J Neurol* 1979; 220:31-41.

Malignant angioendotheliomatosis manifesting as ascending spinal cord dysfunction

PATRICK CHEN, MD, PHD; ERIC GAETJENS, PHD; JOANNA SHER, MD; MAHENDRA SOMASUNDARAM, MD;
TI-KAI LEE, MD; SHU-WEN HOW, MD; NAN-HWA CHO, MD

Malignant angioendotheliomatosis (neoplastic or systemic angioendotheliosis)[1] is an uncommon disease characterized by a proliferation of neoplastic cells within the lumina of small and intermediate sized vessels in a variety of organs. Patients usually suffer from multifocal neurologic symptoms during the course of the illness, which is rapidly fatal. We describe a patient who presented with the symptoms of a conus medullaris lesion, accompanied by anemia and thrombocytopenia. The neurologic manifestations progressed in an episodic, seemingly ascending fashion for one year before final dissemination and the patient's demise.

CASE REPORT

A 68-year-old woman in Taiwan first noticed numbness over both plantar surfaces in April 1983, accompanied by progressive difficulty in walking and emptying her bladder, and constipation. A month later she noted mild fever, skin nodules, and ecchymoses over the medial aspect of both thighs lasting for 12 days. At that time, she was found to have anemia and thrombocytopenia. In June, pain developed over the posterolateral aspect of the left lower extremity, which was not relieved by traction. In July she experienced similar pain in her right leg and was admitted to the National Taiwan University Hospital.

On admission, the patient was alert, with no impairment of mental functions. No rash or lymphadenopathy was present. The cranial nerves and upper extremities were unremarkable. Hip extension and plantar flexion of the feet were weak. There was sensory impairment to pain and touch in both feet, on the lateral aspect of the left leg, and in the perianal area. There were disturbances of position and vibration sense of the lower legs, with a positive Romberg sign. Ankle reflexes were absent, while knee reflexes were hyperactive. Babinski sign was elicited bilaterally. The anal and detrusor reflexes were absent.

Analysis of the cerebrospinal fluid showed 27 lymphocytes and 2 neutrophils; the protein level was 255 mg/dL and the glucose level was 51 mg/dL. The patient had normocytic anemia with hemoglobin (Hb) of 9.7 g/dL; the red blood cell count was $3.33 \times 10^6/\mu L$; white blood cell count, $6{,}600/\mu L$; platelet count, $114{,}000/\mu L$; reticulocytes, 2%; and the erythrocyte sedimentation rate (ESR) was 55 mm/h. (The red blood cell count one year before had been $4.5 \times 10^6/\mu L$). The Coombs test and test for antinuclear antibody (ANA) were negative. The serum albumin level was 3.0 g/dL; globulin, 3.1 g/dL. The only other blood abnormality was a persistent elevation of lactic dehydrogenase (LDH), from 600 to 1,000 SI units, throughout her illness.

Various radiologic studies failed to reveal any tumor. A bone marrow smear showed erythroid hyperplasia. The myelogram was normal. Nerve conduction studies showed decreased amplitude of evoked muscle responses in bilateral peroneal and posterior tibial nerves. An electromyogram (EMG) showed fibrillation in the anterior tibialis, gastrocnemius, and gluteus maximus, and no anal sphincter activity. No scalp evoked potential was recorded on stimulation of peroneal and posterior tibial nerves.

The patient's condition did not change until September 1983, when her symptoms rapidly progressed, with complete urinary and fecal retention and increased weakness with inability to stand. She was transferred to State University of New York Health Science Center at Brooklyn. Examination on admission showed severe wasting of both legs without fasciculation. An LDH isoenzyme study revealed a general increase in all the fractions, with more prominence in isoenzymes 3 and 4. The serum haptoglobulin level was 0. Nuclear magnetic resonance imaging (MRI) of the lower spinal cord performed at New York Hospital revealed a normal contour. Cerebrospinal fluid studies showed negative cytologic findings; protein, 91 mg/dL; glucose, 68 mg/dL; IgG, 25.4 mg% with oligoclonal bands.

By October, the patient could hardly move her lower extremities, which were flaccid. Pain and sensory impairment extended upwards to involve L4 on the right side and L3 on the left. The knee jerk reflexes were gradually lost. EMG demonstrated denervation in all muscles of both lower extremities, with fibrillations in the lumbar paraspinal muscles up to the L2 level. The nerves in the lower extremities were inexcitable, while motor and sensory conduction in the upper extremities remained normal.

A diagnosis of progressive polyradiculoneuropathy was made, and the patient was started on prednisone therapy, 40 mg/day, increasing to 60 mg/day. Azathioprine was then started at 50 mg daily, increasing to 150 mg daily. Twenty days later, the patient felt slight return of movement of the lower extremities. Contractions of the right iliopsoas and quadriceps were noted, as well as feeble contractions of the left anterior thigh. Pain subsided and, subjectively, she also felt better. There was also improvement of the blood studies: the hemoglobin level rose to 12 g/dL, and the red blood cell count rose to

---

From the Departments of Pathology (Drs Chen, Gaetjens, and Sher) and Neurology (Dr Somasundaram), State University of New York Health Science Center at Brooklyn, and the Departments of Internal Medicine (Dr Lee) and Pathology (Drs How and Cho), National Taiwan University Hospital, Taipei, Taiwan, Republic of China.

Address correspondence to Dr Chen, Box 37, SUNY Health Science Center at Brooklyn, Brooklyn, NY 11203.

4 × $10^6/\mu L$. The dose of prednisone was gradually reduced to 40 mg every other day.

In January 1984, pain developed in the left flank area. Thereafter, the patient lost all movements and reflexes in her lower extremities and experienced loss of sensation up to L1 on the right side and T12 on the left. Again, the hemoglobin level dropped to 9.1 g/dL and the red blood cell count dropped to 2.97 × $10^6/\mu L$. A computed tomographic (CT) scan of the abdomen showed moderate splenomegaly and prominent adrenals. The dose of prednisone was stepped up to 50 mg daily. Pain subsided, and the hemoglobin level again rose to 11.5 g/dL. However, there was no improvement of neurologic function. The patient was transferred back to Taiwan and was readmitted to the National Taiwan University Hospital in February 1984.

Azathioprine therapy was discontinued, and the dosage of prednisone was gradually tapered to 40 mg daily. At the end of March, the patient complained of toothache, although a dental examination was normal. She also complained of epigastric pain. In the beginning of April, she became drowsy, with decreased mental abilities. Weakness of the upper extremities developed, and general petechiae were noted. The platelet count dropped to 35,000/μL, and pleural effusion followed. The patient then suffered respiratory failure, and was transferred to the intensive care unit and placed on a respirator. She remained unresponsive most of the time. The leukocyte count dropped to 1,600/μL, the sputum culture grew *Pseudomonas* and *Bacteroides*, and the patient died a short time later.

Autopsy Findings. Gross examination showed multifocal petechial hemorrhages of the cerebral cortex; marked softening of the spinal cord below T10; fibrin deposits in the aorta, vena cava, portal veins, and other great vessels; and a tumor mass measuring 2.8 × 2.0 × 1.5 cm in the left adrenal gland. Other findings included splenomegaly (270 g), chronic pancreatitis with fibrosis and fat necrosis, pneumonia, and mild atherosclerosis.

Microscopic examination showed intraluminal proliferation of neoplastic cells in the small arteries, capillaries, and veins throughout the body, a feature characteristic of malignant angioendotheliomatosis.[1-12] The cells were three to five times the size of lymphocytes and had a large nuclear to cytoplasmic ratio (Fig 1). The nuclei were pleomorphic and showed frequent mitoses. The malignant cells did not contain mucin, melanin, or argyrophilic granules. In the larger blood vessels, the tumor cells formed thrombi incorporating platelets, fibrin, and other blood cells. Extravascular infiltrations of tumor cells were found focally in the myocardium and epicardial fat, as well as in the left adrenal gland, where the tumor cells formed the visible mass and proliferated in a medullary pattern. No carcinoma, sarcoma, or lymphoma was found.

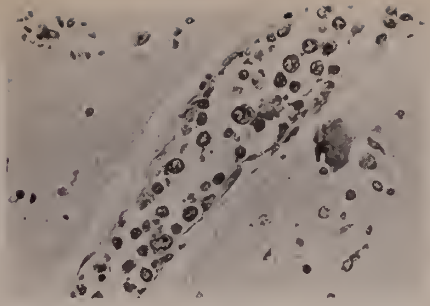


FIGURE 1. Section of brain showing the malignant cells inside an artery (hematoxylin-eosin stain; original magnification × 100).

The spinal cord showed old necrotizing and hemorrhagic myelomalacia involving the gray and white matter in the lower segments (Fig 2). Below the level of T11 the cord was cystic; many blood vessels in the meninges showed recanalization of their lumens and thickened walls. At the level of T10, there was severe dorsal and lateral column degeneration with old and fresh hemorrhages in the anterior and dorsal horns and loss of neurons and gliosis in the anterior horns. No infarcts were detected other than those in the lower spinal cord. Nerve roots showed foci of myelin degeneration. Throughout the central nervous system, the blood vessels also showed intravascular malignant cells and hemorrhage.

Immunohistochemical Findings. Sections from paraffin blocks were rehydrated and stained with various antibodies using the PAP (peroxidase anti-peroxidase) kits from DAKO Co (Santa Barbara, Calif), together with positive and negative controls. The tumor cells from three different sites did not react with antibodies to epithelial membrane antigens, cytokeratin, vimentin, Factor VIII related antigen, S-100, lysozyme, or CEA. About 5% of the cells showed staining with the monoclonal antibody to common leukocyte antigen. This antibody also uniformly stained the small lymphocytes. The reactions to anti-immunoglobulins were equivocal.

DISCUSSION

Malignant angioendotheliomatosis is a rare disease not commonly considered in the differential diagnosis of neurologic syndromes and therefore often missed. Most cases have been diagnosed at autopsy. There are now more than 60 cases reported in the literature.[3] Although the clinical presentation varies, a common pattern has emerged: an older patient presents with neurologic symptoms, often multifocal and affecting various cerebral functions with signs of dementia,[1-11] together with a moderate degree of anemia, elevated LDH and ESR, and a downhill course within one year. Cerebral infarcts may appear in various scans. Sometimes there is a history of nodular skin rash. The disease is rather refractory to treatment, although there was one report of spontaneous remission,[4] and steroid therapy sometimes caused symptomatic relief.[1,5]

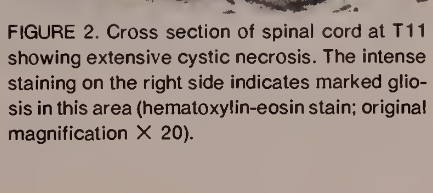


FIGURE 2. Cross section of spinal cord at T11 showing extensive cystic necrosis. The intense staining on the right side indicates marked gliosis in this area (hematoxylin-eosin stain; original magnification × 20).

One frequent finding is tumor formation in the adrenal gland,[2,3,6] which may have pathogenetic significance. The high LDH probably represents the destruction of the nervous tissue. Thrombocytopenia may be the consequence of repeated loss from thrombus formation, and the anemia may be angiopathic/hemolytic in nature.[11] In the case reported here, the hematologic abnormalities seemed parallel to the disease activity.

The clinical symptoms are caused by the presence of malignant cells in small blood vessels, resulting in ischemia, microthrombi, and infarction. The nature of the malignant cells has been extensively studied. It has been previously reported that the cells appear to be derived from the endothelial cells, for they often attached to or replaced the endothelial cells, and contained Factor VIII-related antigens and Weibel-Palade bodies.[2,6] Several recent reports indicate that the cells are "angiotropic" lymphoma cells, based on the findings that they possess lymphoid cell antigens,[7-10] and reclassification of the disease as "intravascular lymphomatosis" has been proposed.[9] In some cases, no known tissue-specific antigens were detected.[2]

The immunoperoxidase staining pattern in the present case was generally consistent with the lymphocytic nature of the tumor cells, although only 5% of the cells were stained. This may be due to the poor preservation of the lymphoid antigens with the autopsy material. Alternatively, anaplastic lymphomas sometimes exhibit no definite antigens or aberrant antigen such as epithelial membrane antigens.[13]

The distinctive feature in this case is the localization of the initial neurologic symptoms to the lower lumbar and sacral region, which later spread upward as the disease progressed. Although the spinal cord is often involved in malignant angioendotheliomatosis,[2,3,6] only one case of isolated spinal cord lesion has been reported, in which the patient suddenly died before the lesion spread.[12] The patient had weakness and then loss of use of both legs. Autopsy revealed hemorrhagic necrosis of the lumbar cord, and malignant cells were found only in the arteries and veins of the spinal cord. The patient described here demonstrates the pattern of infarction and degeneration of the nervous tissue and the recanalization of the blood vessels in the lower cord, which could be the long-term result of the initial involvement. Where spontaneous remission was reported,[4] the blood vessels in the old cerebral lesion showed recanalization without tumor cells, even though intravascular malignant cells were present in the biopsy specimen two years earlier.

The case reported here illustrates that any form of neurologic presentation is possible in malignant angioendotheliomatosis, and this disease should therefore be included in the differential diagnosis of neurologic symptoms, especially in the presence of hematologic abnormalities and the absence of demonstrable tumor. Meningeal biopsy has been proposed as a means of obtaining a definite premortem diagnosis.[4,6]

REFERENCES

1. Ansbacher L, Low N, Beck D, et al: Neoplastic angioendotheliosis: A clinicopathological entity with multifocal presentation. Case report. *J Neurosurg* 1981; 54:412-415.
2. Kitagawa M, Matsubara O, Song SY, et al: Neoplastic angioendotheliosis. Immunohistochemical and electron microscopic findings in three cases. *Cancer* 1985; 56:1134-1143.
3. Wick MR, Banks PM, McDonald TJ: Angioendotheliomatosis of the nose with fatal systemic dissemination. *Cancer* 1981; 48:2510-2517.
4. LeWitt PA, Forno LS, Brant-Zawadzki M: Neoplastic angioendotheliosis: A case with spontaneous regression and radiographic appearance of cerebral arteritis. *Neurology* 1983; 33:39-44.
5. Reinglass JL, Muller J, Wissman S, et al: Central nervous system angioendotheliosis: A treatable multiple infarct dementia. *Stroke* 1977; 8:218-221.
6. Petito CK, Gottlieb GJ, Dougherty JH, et al: Neoplastic angioendotheliosis: Ultrastructural study and review of the literature. *Ann Neurol* 1978; 3:393-399.
7. Wrotnowski U, Mills SE, Cooper PH: Malignant angioendotheliomatosis. An angiotropic lymphoma? *Am J Clin Pathol* 1985; 83:244-248.
8. Sheibani K, Battifora H, Winberg CD, et al: Further evidence that "malignant angioendotheliomatosis" is an angiotropic large-cell lymphoma. *N Engl J Med* 1986; 314:943-948.
9. Wick MR, Mills SE, Scheithauer BW, et al: Reassessment of malignant "angioendotheliomatosis": Evidence in favor of its reclassification as "intravascular lymphomatosis." *Am J Surg Pathol* 1986; 10:112-123.
10. Carroll TJ, Schelper RL, Goeken JA, et al: Angioendotheliomatosis: Immunopathologic and morphologic evidence for intravascular malignant lymphomatosis. *Am J Clin Pathol* 1985; 85:169-175.
11. Arnn ET, Yam LT, Li CY: Systemic angioendotheliomatosis presenting with hemolytic anemia. *Am J Clin Pathol* 1983; 80:246-251.
12. Ojeda VJ: Neoplastic angioendotheliosis of the spinal cord. Case report. *Acta Neuropathol (Berl)* 1983; 62:164-166.
13. Gonzalez-Crussi F, Mangkornkanok M, Hsueh W: Large-cell lymphoma. Diagnostic difficulties and case study. Case reports. *Am J Surg Pathol* 1987; 11:59-65.

FROM THE LIBRARY

ACUTE OSTEOMYELITIS

. . . I am in agreement with Dr. Harris' presentation of the picture of acute osteomyelitis of the long bones, and am in accord with his feeling that the condition is a surgical emergency that calls for immediate operation. When one has seen a child with acute osteomyelitis that has been operated upon within a few hours after the onset of the clinical symptoms, as I have on several occasions, and has noted the immediate change in the patient's condition so that directly there is an abrupt drop in the temperature, with subsidence of toxic symptoms, one is necessarily impressed . . .

In very young children the condition seems milder than in the older ones. For the past several years I have been very much interested in acute osteomyelitis of the long bones in adults, and whereas the clinical picture may be the same as one sees in childhood, it frequently is different, and in the early stages is a relatively mild affair and tends to remain more localized. If untreated surgically, the process spreads.

The picture as seen in the adult is frequently a very indefinite affair. There is usually no history of preceding injury, or of a preceding or concomitant infection elsewhere in the body. The patient's complaint is one of pain, often poorly localized. At the time of onset and for days the adult frequently shows a temperature approximately normal and a white blood count approximately normal without a material increase in the polymorphonuclear leukocytes.

The pathologic picture is different. The changes are essentially local periosteal ones associated with central involvement of the shaft. This is due to a more even blood supply to the bone, in contrast to a greater blood supply about the metaphyseal region in children. There is greater likelihood of joint involvement in the adult because the cartilaginous barrier of the epiphysis has disappeared. At operation one may find a local cortical thickening bathed in a couple of cubic centimeters of pus and, in this contingency, the surgeon should not be misled into believing that there is a suppurative periostitis present. This is part of the picture of an osteomyelitis, and the shaft of the bone should be opened. In an adult I prefer removal of a small window of cortical bone rather than making drill holes as I do in children. Sequestrum formation in adults is rare. The organism is usually a staphylococcus aureus. It may be a streptococcus.

Realizing how poorly osteomyelitis is treated, and I think it is the most poorly treated of all surgical conditions, acute osteomyelitis in adults (fortunately infrequent) is more poorly handled.

DR ISADORE ZADEK
(*NY State J Med* 1939; 1556-1557)

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Review of *Once a Doctor, Always a Doctor*

TO THE EDITOR: I was thrilled when I opened my April copy of the *Journal* and found the review of my book, *Once a Doctor, Always a Doctor: The Memoirs of a German-Jewish Immigrant Physician*, by Dr Naomi Bluestone,[1] whose articles in various publications I have read and admired for many years. I am looking forward to reading Dr Imperato's *Doctor in the Land of the Lion* and Dr Bluestone's own book, *So You Want to Be a Doctor*, which are referred to in the review. Yet, when it comes to my own book, Dr Bluestone's empathy is somewhat lacking. In two long paragraphs she discusses an introductory remark of mine, made in jest, that after so many autobiographies of famous men and women the public might want to hear from an ordinary man for a change. She disagrees with this attitude completely, but contradicts herself at the end of the essay by saying, "I have always found the biographies of the ordinary docs of this world more interesting than those of the Schweitzers, perhaps because there is more with which I can identify."[1]

Too bad Dr Bluestone was unable to identify with *Once a Doctor, Always a Doctor*, in contrast to the numerous people, Jews and non-Jews, who communicated with me. I was "fortunate enough to have escaped the Holocaust," but have "virtually nothing to say about it." I was still a prisoner of Buchenwald concentration camp, and I was able to emigrate to the United States in 1939, years before those annihilation camps of Auschwitz and Treblinka were set in motion. There were very few survivors, and they have given us dozens of volumes about the Holocaust. My own book is not a book about the Holocaust, but my autobiography, which I felt compelled to write. Two-thirds of my life have been spent in America as a practicing physician, struck also by tragedy here. Although I devoted "only several pages" to our vaccine-damaged son Michael, I tried to come to grips all through my book with the Holocaust, the loss of our close relatives, and constant illness, to search for meaning in all our lives. I don't have all the answers, but who has? How could Dr Bluestone have missed all this? How could she have failed to find my autobiography "worth the time of a busy reader"? Well, it is not easy to write about four physicians' memoirs in a long essay, which included comments about Dr Arthur Hertzler's *The Horse and Buggy Doctor* and Sir Thomas Browne's *Religio Medici*. She probably had a deadline to meet, too.

I completely agree with Dr Bluestone's last paragraph concerning my book: "Still, I commend Dr Hartmann warmly. He has troubled to write a chronicle for his family that coming generations will rightfully cherish... I wish him a long and happy life."

A happy and long life to you, Dr Bluestone! And let us hope that the manuscript of your own revered grandfather, Dr Joseph Isaac Bluestone, will find a publisher soon.

HEINZ HARTMANN, MD
PO Box 508
Syracuse, NY 13214

---

[1] Bluestone NR: Taking stock: Physicians' memoirs. *NY State J Med* 1987; 87:239-241.

Review of physicians' memoirs

TO THE EDITOR: I want to tell you how much I enjoyed reading Dr Naomi Bluestone's essay on physicians' memoirs in the April issue of the *Journal*.[1] I have always been fascinated by medical autobiographies and biographies.

I am now retired from my New York City practice, living in a wonderful Quaker retirement continuing-care community. I miss my patients but keep up with medical meetings and music.

With appreciation, thanks and best wishes.

CHARLES A. PERERA, MD
Kendal at Longwood #17
Kennett Square, PA 19348

---

[1] Bluestone NR: Taking stock: Physicians' memoirs. *NY State J Med* 1987; 87:239-241.

Dr Bluestone's essay review

TO THE EDITOR: I congratulate Dr Bluestone for that great statement in her essay review, "Nothing of interest to the philosphers of mankind can come from physicians who function as agents of the bottom line." [1] Superb!!

FRANK A. DISNEY, MD
Elmwood Pediatric Group
125 Lattimore Rd
Rochester, NY 14620

---

[1] Bluestone NR: Taking stock: Physicians' memoirs. *NY State J Med* 1987; 87:239-241.

Malpractice by nonphysician healers

TO THE EDITOR: We found your July 1986 issue on medical malpractice to be timely and of interest. Although comprehensive in scope, none of the articles in the issue addressed the problem of ineffective and inappropriate care provided by nonphysician healers. We recently treated a young woman who succumbed to a disease of which she might otherwise have been cured had she not been in the care of a variety of "healers."

Case Report. A 29-year-old woman presented with a large, fungating lesion on the anterior aspect of the right lower leg, which had first appeared in 1975. She consulted a number of nonphysician healers while avoiding contact with physicians. A variety of medicines, potions, and injections were employed, the pharmaceutical natures of which were either unclear or kept secret. Over the years, the lesion grew in size, and the patient finally sought medical care at a hospital 11 years later, in April 1986, because of intense pain and a worsening of her general physical condition.

She had a 14 × 19-cm fungating mass of the lower right leg that was infected, necrotic, and foul smelling (Fig 1). On admission, she was febrile, with a temperature of 39°C, and complained of coughing and diffuse chest pain. The relevant diagnostic findings

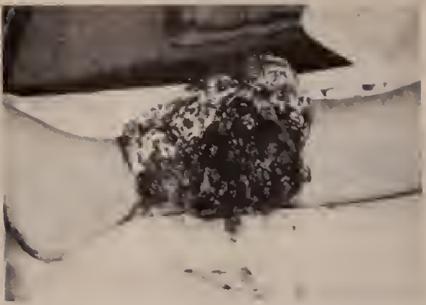


FIGURE 1. Large, fungating mass of the right lower leg.

included tender right inguinal lymphadenopathy, and a chest film that showed right hilar adenopathy as well as bilateral parenchymatous nodular shadows suggestive of metastatic disease.

A biopsy of the lesion on the patient's leg showed it to be a fibrosarcoma (fibroblastic sarcoma). Microscopic examination revealed a proliferation of fusiform cells in the dermis and hypodermis, clustered into interwoven bundles, and a paucity of collagen fibrils. In addition to normal vessels, there were also vascular lacunae devoid of any endothelial lining and bordered by tumor cells. A test for tumor markers was negative.

The patient refused surgical treatment and was thus only symptomatically treated with antibiotics and analgesics. Her condition rapidly deteriorated, with extension of the pulmonary metastases, and she died in June 1986, six weeks after admission.

At autopsy, extensive pulmonary metastases were found, and the diagnosis of fibrosarcoma was confirmed. In retrospect this rather rare tumor presented in a characteristic manner, being located at the distal portion of a limb.[1]

Discussion. This patient's course illustrates not only the natural history of untreated fibrosarcoma of a limb but also the ineffectiveness and inappropriateness of the care she received from a variety of healers. Wide resection of this type of tumor during its early stages combined with appropriate radiotherapy often gives impressive survival rates. This patient's unfortunate course was directly due to the medical malpractice of the healers who treated her. The elements for a judgment of medical malpractice were present, in that these healers had a duty to the patient, breached this duty through negligent care, and in so doing caused injury.[2,3] From another perspective, they assumed the care of a patient whose illness they were not competent to treat. Although this case is a dramatic example, it serves to alert both physicians and society in general to the existence in industrialized societies of healers, who usually practice illegally, beyond the fringes of the medical care system. They frequently escape both legal and societal accountability.

JEAN-MARC NORES, MD
JEAN-MICHEL REMY, MD
ANDRE D. NENNA, MD
Department of Internal Medicine
University of Paris
Hopital Raymond Poincare
92380 Garches, France

PASCAL REYGAGNE, MD
Department of Dermatology
University of Paris
Hopital Saint-Louis
75010 Paris, France

1. Pritchard DJ, Soule EH, Taylor WF, et al: Fibrosarcoma—A clinicopathologic and statistical study of 199 tumors of the soft tissues of the extremities and trunk. *Cancer* 1974; 33:888-897.
2. Kramer C: Medical malpractice: The patient's point of view. *NY State J Med* 1986; 86:348-351.
3. Clark DW, MacMahon B: *Preventive and Community Medicine*, ed 2. Boston, Little Brown & Co, 1986, p 656.

Colostomy bag simulating bony metastases

TO THE EDITOR: Intrinsic and extrinsic artifacts causing radiographic abnormalities may result in unnecessary and expensive investigation to rule out serious underlying disease. This can be prevented if the primary care provider, radiologist, and technician keep in mind the possibility that an artifact may exist. Nipple shadows mimicking a pulmonary nodule are the most common radiologic artifact and can easily be corrected by radiographic marking. We describe a case of a radiologic artifact in which a colostomy bag mimicked pelvic bony metastases.

Case Report. A 60-year-old man presented with a two-week history of lumbar and left posterior hip pain. He had a longstanding history of intermittent low back pain, but recently reported a constant, dull pain in the lower lumbar and left buttock region which radiated circumferentially around the left thigh to the knee. The pain was slightly worse with lumbar motion, but unrelated to posture or position. He denied any history of trauma, claudication, and abdominal or urinary symptoms. The medical history was significant for a total colectomy for ulcerative colitis 16 years previously and a lumbar laminectomy for a herniated disc 12 years previously.

An ileostomy bag with fecal contents was noted over the right lower quadrant and there was a well-healed scar from the previous abdominal surgery. The musculoskeletal and neurologic examinations, including gait and straight leg raising tests, were normal. Anteroposterior pelvic and lumbosacral radiographs revealed lumbosacral disc degeneration and osteosclerotic changes, or possible osteoblastic metastases in the right ilium and acetabular area (Fig 1). These findings were not present on a prior radiograph. Further laboratory tests revealed an alkaline phosphatase level of 86 IU/L (normal, 40-115 IU/L): acid phosphatase, 0.46 IU/L (normal, 0.0-0.8 IU/L); and sedimentation rate, 6 mm/h. In order to rule out metastatic bone disease, a bone scan and joint imaging study was obtained. This study demonstrated uptake at the third lumbar vertebra and right hip joint compatible with arthritis.

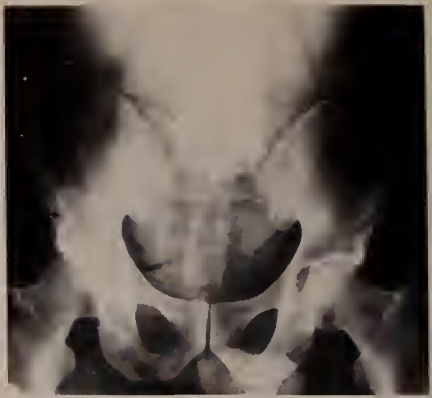


FIGURE 1. Anteropelvic radiograph showing osteosclerotic changes or possible osteoblastic metastases in right ileum (arrow).

The patient continued to have pain, and a repeat pelvic radiograph was obtained while the colostomy bag was elevated. The colostomy device was now noted over the right iliac bone (Fig 2). The possible bony metastases visible in Figure 1 were not seen on the repeat films, and were obviously due to the fecal contents in the colostomy bag. In retrospect, the patient's symptoms were most likely secondary to the degenerative disc disease.

Discussion. Various skin lesions and surface foreign bodies are well-known causes of radiologic artifact, if they are not marked or removed. Chewing gum "stuck" to the skin of the anterior chest, like nipple shadows, can mimic a solitary pulmonary nodule. Skin tumors, especially pedunculated lesions such as sebaceous cysts, lipomas, multiple skin warts, and neurofi-

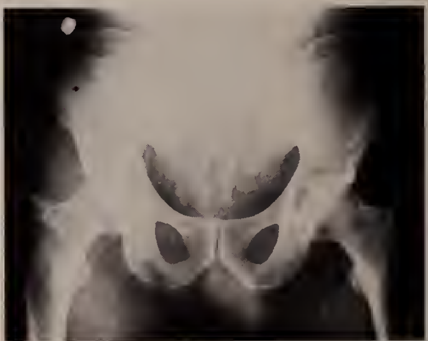


FIGURE 2. Arrow shows colostomy bag over the right iliac bone, with no visible bony metastases now present.

bromatosis, can cast round shadows simulating nodules in the lung.[1] Braided hair has been reported to mimic apical scarring.[2]

There is a paucity of information on nonpulmonary radiologic artifacts. The button on one patient's robe simulated a gallstone[3] and ingested medication has mimicked renal stones.[4] In addition, matted fabrics lying under the patient can appear similar to calcified arteries in the abdomen.[3] This case clearly illustrates how a simple oversight can lead to expensive diagnostic evaluation. However, such occurrences can easily be prevented if the colostomy bag is properly marked with radiopaque material or is elevated away from the pelvic area.

KUSUM L. KUMAR, MD
DAVID L. SMITH, MD
LINDA M. LUCAS, MD
Division of General Medicine
Oregon Health Sciences University
Ambulatory Care and Medical Service
Veterans Administration Medical Center
Portland, OR 97207

1. Sutton D: Some differential diagnoses, in *A Textbook of Radiology and Imaging*, ed 3. New York, Churchill Livingstone Inc, 1980, pp 425-445.
2. Buccini RV, Rubin II: Radiographic artifact from braided hair mimicking tuberculosis [letter]. *N Engl J Med* 1985; 313:1227-1228.
3. Sweeney RJ: Artifacts caused by failing to attire the patient properly, in *Radiographic Artifacts: Their Cause and Control*, ed 1. Philadelphia, JB Lippincott Co, 1983, pp 133-186.
4. Benforado JM: Pills as radiologic artifacts. *GP* 1969; 40:135.

FROM THE LIBRARY

THE ILLEGALITY OF CULT PRACTICE

Physicians need to have a clear conception of what is illegal and reprehensible in the practice of the healing cults, and to be able to present their arguments in a form which is clear and non-technical, and which can be understood by the average layman and legislator.

It is not illegal or reprehensible for anyone to visit the sick; to speak words of encouragement to them; to pray with them; or to administer food, baths, or massage to them in accordance with the present every-day standards of such acts. It is not illegal and may not be reprehensible for anyone to make it a practice to offer his services in these lines, or even to accept pay for services rendered, provided the services are not at variance with similar services in vogue in the community. A nurse or a masseur, for example, may practice such a calling for pay, and a social worker may speak motherly words of encouragement which might not be justified by a strict scientific analysis of their import, because each of the above has presumably been schooled in some type of education along the lines of modern scientific thought. But these basic principles of certain acts being not illegal nor reprehensible are incomplete and misleading as is the oft-quoted statement—that "all men are created free and equal."

When do encouraging words, or prayers, or physical manipulations cease to be desirable and become reprehensible and dangerous?

The answer is—when they either cause direct harm, or invite a patient to neglect a standard plan of treatment evolved in accordance with the present-day sciences and which would bring relief or cure. . . .

Cultists of all kinds put forth with a stream of words the monotonous argument that anyone has an inalienable freedom of choice of a healer or a system of healing. The truth is, that few persons would seek the cultist if the cultists themselves did not actively seek patients and, through their advertisements, hold out rosy promises of cure for every person who would come under their treatment. The patients of cultists are irresistibly drawn to them by dazzling promises of cure. They throw judgment to the winds and place a blind faith in the cultist because of the promises of sure cures—such promises as no reputable physician would dare to make after a careful and scientific physical examination. Would that the patients of the cultists publish their results of failure as freely as do the few who have apparently received benefit after the remission of their disease! . . .

EDITORIAL
(*NY State J Med* 1924; 24:27-28)

Schedule for children beginning immunization in infancy

TABLE 1. Recommended schedules for active immunization of normal infants and children

Recommended age†	Vaccine (s)§	Comments
2 months	DTP-1¶,OPV-1**	Can be given earlier in areas of high endemicity.
4 months	DTP-2, OPV-2	6-week to 2-month interval desired between OPV doses to avoid interference.
6 months	DTP-3	An additional dose of OPV at this time is optional for use in areas with a high risk of polio exposure.
15 months††	MMR,§§DTP-4, OPV-3	Completion of primary series of DTP and OPV.
24 months	Hib¶¶	Can be given at 18-23 months for children in groups who are thought to be at increased risk of disease, e.g., day-care-center attendees.
4-6 years***	DTP-5, OPV-4	Preferably at or before school entry.
14-16 years	Td†††	Repeat every 10 years throughout life.

†These recommended ages should not be construed as absolute, i.e., 2 months can be 6-10 weeks, etc.

§ For all products used, consult manufacturer's package enclosure for instructions for storage, handling and administration. Immunobiologics prepared by different manufacturers may vary, and those of the same manufacturer may change from time to time. The package insert should be followed for a specific product. See chart on back. Recommendations for storage and handling

¶ DTP-Diphtheria and Tetanus Toxoids and Pertussis Vaccine Adsorbed

**OPV-Poliovirus Vaccine Live Oral; contains poliovirus strains Types 1, 2 and 3.

††Provided at least 6 months have elapsed since DTP-3 or, if fewer than three DTPs have been received, at least 6 weeks since last previous dose of DTP or OPV. MMR vaccine should not be delayed just to allow simultaneous administration with DTP and OPV. Administering MMR at 15 months and DTP-4 and OPV-3 at 18 months continues to be an acceptable alternative

§§ MMR-Measles, Mumps and Rubella Virus Vaccine, Live

¶¶Haemophilus b Polysaccharide Vaccine for use in the prevention of Haemophilus influenzae type b (Hib) infection.

***Up to the seventh birthday

†††Td-Tetanus and Diphtheria Toxoids Adsorbed (For adult use) — contains the same dose of tetanus toxoid as DTP or DT and a reduced dose of diphtheria toxoid

PRECAUTIONS AND CONTRAINDICATIONS Consult the manufacturer's package insert for the specific product used. *In general,* contraindications include:

1. current illness with something more serious than a cold;
2. immunosuppressive therapy (radiation, corticosteriods, antimetabolites, alkylating agents and cytotoxic agents);
3. recent (within 3 months) immune globulin (IG), plasma or blood transfusion;
4. immunodeficiency disorders;
5. leukemia, lymphoma or generalized malignancy;
6. prior allergic reaction to the same vaccine;
7. specific severe allergic conditions must be considered for each antigen. Consult manufacturer's package insert.

REQUIREMENTS FOR SCHOOL ATTENDANCE

Under section 2164 of the New York State Public Health Law, all children attending a school or day care center must be immunized against diphtheria, polio, measles, mumps and rubella. Minimum requirements include:

- 3 doses of diphtheria toxoid (usually administered as either DTP or Td)
- 3 doses of TOPV (or, as an alternative, 4 doses of IPV administered after 1968)
- 1 dose of live measles vaccine administered after the age of 12 months
- 1 dose of live mumps vaccine administered after the age of 12 months
- 1 dose of live rubella vaccine administered after the age of 12 months

USE OF INACTIVATED POLIO VACCINE (IPV), where indicated

The primary series consists of 4 doses vaccine (volume and route specified by the manufacturer). In infancy, the primary schedule is usually integrated with DTP: 3 doses given at 8 week intervals with a fourth dose 6-12 months after the third. Booster doses should be given at school entry and thereafter every 5 years through age 18.

TETANUS PROPHYLAXIS IN WOUND MANAGEMENT

HISTORY OF TETANUS IMMUNIZATIONS	CLEAN, MINOR WOUNDS		ALL OTHER WOUNDS	
	Td (Adult)	TIG[4]	Td (Adult)	TIG
UNCERTAIN	YES	NO	YES	YES
0-1	YES	NO	YES	YES
2	YES	NO	YES	NO[1]
3 OR MORE	NO[2]	NO	NO[3]	NO

[1] Unless wound more than 24 hours

[2] Unless more than 10 years since last dose

[3] Unless more than 5 years since last dose

[4] TIG - tetanus immune globulin

NOTE: Tetanus immune globulin and Td may be given simultaneously but in different sites. Substitute DTP vaccine for children under 7 years of age

---

* Guidelines prepared by the New York State Department of Health, David Axelrod, MD, Commissioner.

Schedules for children not immunized as infants

TABLE 2. Recommended immunization schedule for infants and children up to 7th birthday not immunized at the recommended time in early infancy*
(See individual ACIP recommendations for details.)

Timing	Vaccine(s)	Comments
First visit	DTP-1, OPV-1 (If child is ≥ 15 mo. of age, MMR)	DTP, OPV and MMR can be administered simultaneously to children ≥ 15 mo of age
2 mo. after first DTP, OPV	DTP-2, OPV-2	
2 mo. after second DTP	DTP-3	An additional dose of OPV at this time is optional for use in areas with a high risk of polio exposure
6-12 mo. after third DTP	DTP-4, OPV-3	
Preschool** (4-6 yr.)	DTP-5, OPV-4	Preferably at or before school entry
14-16 yr.	Td	Repeat every 10 years throughout life

Please note: Hib vaccine is routinely administered at 24 months of age and may be given in combination with any other vaccine(s) presented above. Hib vaccine may be administered to children as young as 18 months of age if they are at increased risk, i.e., day-care attendees.

*If initiated in the first year of life, give DTP-1, 2 and 3, OPV-1 and 2 according to this schedule and give MMR when the child becomes 15 months old

**The preschool dose is not necessary if the fourth dose of DTP and third dose of OPV are administered after the fourth birthday

TABLE 3. Recommended immunization schedule for persons 7 years of age or older
(See individual ACIP recommendations for details.)

Timing	Vaccine(s)	Comments
First visit	Td-1*, OPV-1, and MMR§	OPV not routinely administered to those ≥ 18 years of age
2 mo. after first Td, OPV	Td-2, OPV-2	
6-12 mo. after second Td, OPV	Td-3, OPV-3	OPV-3 may be given as soon as 6 weeks after OPV-2
10 years after Td-3	Td	Repeat every 10 years throughout life

*Td-Tetanus and diphtheria toxoids (adult type) are used after the seventh birthday. The DTP doses given to children under 7 who remain incompletely immunized at age 7 or older should be counted as prior exposure to tetanus and diphtheria toxoids (e.g., a child who previously received 2 doses of DTP, only needs 1 dose of Td to complete a primary series)

§ MMR - Live measles, mumps and rubella virus vaccines combined. Persons born prior to 1957 may generally be considered immune to measles and mumps and may not need routine immunization. Rubella vaccine may be given to persons of any age, particularly women of childbearing age. Prior to administering rubella vaccine to females past menarche, the patient and/or her parent/guardian must be asked if she is pregnant. Pregnant patients should not be given rubella vaccine due to theoretical risks to a fetus. Females receiving vaccine should be informed of the importance of not becoming pregnant for three months following vaccination.

INTERRUPTION OF IMMUNIZATION SCHEDULE

A delay between doses does not interfere with final immunity and does not necessitate starting the series over again, regardless of the interval elapsed. Simply resume the schedule where it left off.

FOR FURTHER INFORMATION, CALL (518) 474-4284

RECOMMENDATIONS FOR THE HANDLING AND STORAGE OF BIOLOGICALS*

BIOLOGICAL	SHIPPING REQUIREMENTS	CONDITION ON ARRIVAL	STORAGE REQUIREMENTS	SHELF-LIFE EXPIRATION	INSTRUCTIONS ON RECONSTITUTION OR USE	SHELF-LIFE AFTER RECONSTITUTION/ THAWING OR OPENING	SPECIAL INSTRUCTIONS
D.T.P. Diphtheria Tetanus Pertussis	Ship in insulated container Maintain temperature at 2° - 8° C (35° - 46°F).	Refrigerate immediately.	Refrigerate immediately upon arrival - store at 2° - 8° C (35° - 46°F). DO NOT FREEZE - this reduces potency.	Up to 18 months Check date on vial or container	Shake vial vigorously before withdrawing each dose	Until outdated, if not contaminated	Rotate stock so that most current expiration dated stock is used first
T.D. - Adult Tetanus - Diphtheria Tetanus Toxoid	Ship in insulated container Maintain temperature at 2° - 8° C (35° - 46°F)	Refrigerate immediately	Refrigerate immediately upon arrival - store at 2° - 8° C (35° - 46°F). DO NOT FREEZE - this reduces potency	Up to 2 years Check date on container or vial	Shake vial vigorously before withdrawing each dose When used on jet injector gun, shake vial before each injection	Until outdated, if not contaminated Multidose vials for jet injector discard remainder of contents on day of use	Rotate stock so that most current expiration dated stock is used first Multidose vials for jet injector use should not be utilized in needle/syringe method of immunization
Rubella Virus **Measles Virus** **Measles/Rubella** - combined virus **Measles/Mumps/ Rubella** combined virus **Mumps**	Use insulated container Must be shipped with refrigerant. Maintain at 10° C (50°F) or less. If shipped with dry ice, diluent must be shipped separately	Should be below 10° C (50°F) If above this temperature, refuse shipment or return to shipper DO NOT USE WARM VACCINE. Refrigerate on arrival	If possible, separate vaccine from diluent. Store as follows: **Vaccine** - refrigerate immediately upon arrival. Store at 2° - 8° C (35° - 46°F). PROTECT FROM LIGHT AT ALL TIMES, since such exposure may inactivate the virus. **Diluent** - Store at 15° - 30° C (59° - 86°F), room temperature. **Special Note:** Freeze-dried vaccines may be maintained at freezer temperatures.	**Vaccine** — Up to 2 years Check date on container or vial **Diluent** — Check date on container or vial	RECONSTITUTE JUST BEFORE USING Use only diluent supplied to reconstitute **Singles** — inject into the vial of lyophilized vaccine and agitate to ensure thorough mixing Withdraw entire contents into syringe and inject total volume of vaccine subcutaneously **Multidose Vials** — withdraw ALL diluent from ampul into syringe, inject into vial of lyophilized vaccine and agitate to ensure thorough mixing **10-Dose Vial** — withdraw 0 5 cc of reconstituted vaccine into separate sterile syringe and needle for each immunization. **50-Dose Vial** — place on jet injector gun with dosage at 0 5 cc	After reconstitution, use immediately or store in a dark place at 2° - 8° C (35° - 46°F). DISCARD IF NOT USED WITHIN 8 HOURS	Rotate stock so that most current expiration dated stock is used first Use only the diluent supplied to reconstitute the vaccine. DO NOT use diluents from other vaccines or manufacturers **50-Dose Vials** - for jet injector use should not be utilized in needle/syringe method of immunization. **10-Dose Vials** - may be used for both jet injector and needle/syringe methods of immunization
Poliovirus	Pack with dry ice. Should be delivered within 3 days.	Should be frozen If thawed but still cold - below 8° C (46°F) - refreeze immediately If thawed and warm - above 8° C (46°F) - or vaccine is cloudy, refuse shipment or return to shipper DO NOT USE THAWED, WARM OR CLOUDY VACCINE	Maintain continuously in the frozen state, -14° C (17°F) or lower The **vaccine may be refrozen** - if not opened, a maximum of 10 freeze-thaw cycles is permissible, provided the total cumulative duration of thaw does not exceed 24 hours, and provided the temperature does not exceed 8° C (46°F) during the period of thaw	Up to 1 year Check date on container or vial	Thaw before using - may be rubbed between hands for rapid thawing	Vaccine in liquid state in unopened vials may be used for up to 30 days provided it has been stored at 2° - 8° C (35° - 46°F). Once the vial is opened, it must be refrigerated at 2° - 8° C (35° - 46°F) and used within 7 days.	If the dropper becomes contaminated by touching the mouth, do not reuse dropper or reintroduce into the vial Sterilize dropper by boiling in water (cool before replacing dropper in vial) or use a new dropper before continuing administration. USE ONLY THE DROPPER SUPPLIED IN THE PACKAGE
Influenza	Should be delivered in shortest possible time Should not be exposed to excessive temperatures	Should not have been frozen Refrigerate on arrival	Refrigerate immediately upon arrival Store at 2° - 8° C (35° - 46°F) DO NOT FREEZE - this reduces potency	Check date on vial or container	Shake vial vigorously before withdrawing each dose	Until outdated, if not contaminated	Rotate stock so that the most current expiration dated stock is used first
Hib Vaccine Haemophilus b Polysaccharide Vaccine	Ship in insulated container to prevent freezing	Should not have been frozen Refrigerate on arrival	Refrigerate immediately on arrival Store at 2° - 8° C (35° - 46°F) Do not freeze - this reduces potency	Up to 2 years Check date on container	Reconstitute before use Record date of reconstitution on vial Use only diluent supplied Mix thoroughly before use	**Multiple dose** - stable for 30 days after reconstitution if stored at 2° - 8° C (35° - 46°F) when not in use **Single dose** - discard unused reconstituted vials after 8 hours	Rotate stock so that the shortest dated vaccine is used first

*These recommendations are not a substitute for the package insert included with each different biologic.

NOTE All materials used for administering live virus vaccines should be burned boiled or autoclaved prior to disposal

REVISED MARCH 1987

American Medical Association endorses mandatory AIDS testing for two groups

The House of Delegates of the American Medical Association (AMA) voted approval on June 23 of recommendations concerning mandatory AIDS testing of all prisoners and those immigrating to the United States. The federal government had adopted similar policies two weeks before as part of a program announced by US Attorney General Edwin Meese III. The US government program also requires mandatory testing of members of the military, Foreign Service officers, and applicants for certain programs administered within the Department of Labor by the Job Corps. The AMA House of Delegates also approved:

- Routine AIDS testing of patients at clinics that treat drug abusers and those with sexually transmitted diseases provided patient consent is given.
- An increase in educational efforts targeted to physicians and the public.
- An increase in federal financing for AIDS testing, for counseling of infected individuals, and for research into counseling methods.
- Implementation of education programs among elementary and young adult students regarding the mode of transmission of AIDS.
- The creation of a national commission on AIDS, comprising national, state, and local leaders, to develop a consensus on how best to combat AIDS.

The AMA's call for a national AIDS commission is seen by many as an important step forward in coordinating the nation's efforts against the disease. Similar such commissions already exist in a number of countries.

FDA licenses Western blot kit for HIV

The FDA has licensed a Western blot commercial test kit for confirming the presence of antibodies to human immunodeficiency virus (HIV) in donated blood samples that have yielded positive results on an ELISA screening test. This in vitro assay, developed and produced by Biotech Research Laboratories and the Dupont Company, combines electrophoretic and immunoblotting techniques to identify HIV antibodies. The US Department of Health and Human Services suggests the following precautions when using the kit: handle all specimens and assay materials as if capable of transmitting an infectious agent; wear gloves throughout the test procedure; dispose of all gloves, specimens, and assay materials as biohazard waste.

FDA compliance policy guide for condoms

The Food and Drug Administration (FDA) issued a compliance policy that outlines the criteria for seizure actions against condom manufacturers who fail to comply with the maximum voluntary standard concerning defect levels. The FDA has modified its inspection sampling criteria to correspond with the voluntary standard established by the American Society for Testing Materials. According to this standard, the acceptable quality level for leakage is 0.4% (4 leaking condoms/1,000 condoms). On inspection, the FDA will accept 0.4% as "the maximum percent defective for leakage that will be considered satisfactory as a process average."

New prescription format

Replacement of triplicate and nontriplicate prescription forms with a new, one-signature, "dispense-as-written" prescription format went into effect on July 1. The 1986 legislation that provides for pharmaceutical insurance coverage for the elderly prompted the change in format. The new forms permit physicians to request the use of brand-name pharmaceuticals. July 1 also marked the date on which benzodiazepines, including Valium, require the triplicate prescription forms used for Schedule II substances.

The MSSNY opposed the change in prescription format according to President Samuel Gelfand, who described the plan as "a wasteful expenditure of health care dollars."

New drug approach against ovarian cancer

A radioactive drug that mimics the female hormone estrogen has proved effective in killing ovarian cancer cells in rats. The drug, developed by researchers at the US Department of Energy's Argonne National Laboratory and the University of Chicago, is scheduled to undergo clinical trials in humans within three years.

Traditional forms of treatment, including surgery, chemotherapy, and radiation, are relatively ineffective against advanced ovarian cancer, which often spreads to multiple sites. This new drug takes advantage of the rapid reproduction rate of cancer cells, and the fact that ovarian tumor cells frequently have increased numbers of estrogen receptors and require estrogen for growth. Once taken up by the cell, the drug concentrates in the genetic material, and its radioactive component damages the DNA, thus killing the cell. With its specificity for ovarian cells and a short half-life of four hours, the drug poses little danger to other parts of the body.

Is breast self-examination an effective means of screening for breast cancer?

The positive attributes of breast self-examination (BSE) are apparent: It is an easy, noninvasive, and inexpensive procedure that can result in the early detection of breast cancer. In a review of the current literature on BSE, O'Malley and Fletcher (*JAMA* 1987; 257:2197–2203) address the issue of "what role BSE should play—a major detection method or a supplement to clinical breast examination (CBE) and mammography."

The authors first reviewed the effectiveness of treatment following early detection of breast cancer. "There is good evidence that early treatment following screening by

---

NEWS BRIEFS are compiled and written by Vicki Glaser.

CBE and/or mammography is associated with reduced breast cancer mortality for women 50 years of age and older." Previous studies have reported a reduction in breast cancer mortality of about 33% in the 50–59-year age group and at least 20% in women aged 60 years and older. Alternatively, for women younger than 50 years, there appears to be no reduction in mortality associated with early treatment following screening by CBE and/or mammography.

Studies that focus on the effectiveness of BSE suggest that women trained in the technique may discover slightly smaller tumors, and that among women who perform BSE, there is an apparent reduction in mortality due to breast cancer and an improved estimated five-year survival rate.

In the United States, BSE is recommended as a supplement to CBE and mammography in screening for breast cancer. In assessing the acceptability of BSE, the authors reached the following conclusions: It is a safe and relatively simple procedure; women seem to be willing to learn BSE; and, although most women do not practice BSE on a monthly basis—as recommended by the American Cancer Society—they are more likely to perform the technique if they receive some training, and the sensitivity of the procedure is enhanced following such training. The authors propose that "the problem with BSE is not evidence of a lack of effect, but lack of evidence. With strong evidence already available for . . . CBE and mammography, efforts to get older women screened for breast cancer currently should be focused on these methods."

Aortic valve rupture following the Heimlich maneuver

The Heimlich maneuver, used to clear an air passage in a choking victim, has been associated with complications including pharyngeal abrasion, esophageal laceration, retinal detachment, and fractured ribs. Passik et al (*Arch Pathol Lab Med* 1987; 111:469–470) describe a patient who experienced a ruptured aortic bioprosthesis believed to have occurred as a result of the Heimlich maneuver.

The 74-year-old female patient had received an Ionescu-Shiley bovine pericardial bioprosthesis in 1981 due to extensive degenerative calcification of a tricuspid aortic valve. A 17-month follow-up evaluation showed normal function of the prosthesis. In 1984, she was rescued from a severe choking episode with the Heimlich maneuver. Subsequently, orthopnea, severe dyspnea, pedal edema, and a grade 3/6 diastolic blowing murmur developed. Angiography revealed severe aortic insufficiency. Surgical evaluation in 1985 showed a perforation in the noncoronary cusp near one commissure and a calcified tear at the adjacent left cusp. Valve replacement corrected the problem.

The authors theorize that the aortic valve ruptured as a result of the sharp increase in intrathoracic pressure created by the Heimlich maneuver. "Most nonpenetrating trauma produces avulsion at points of fixation, with tearing of commissural attachments and resultant prolapse of cusps," the authors note. The effects of the Heimlich maneuver should be considered in a patient with unexplained acute prosthetic insufficiency.

New treatment approach reduces morbidity and mortality associated with toxic epidermal necrolysis

Toxic epidermal necrolysis (TEN) is a severe sloughing of the epidermis accompanied by fever, toxic reactions, intense pain, conjunctivitis, and often mucositis affecting the mouth, upper airway, and esophagus. TEN is associated with a mortality rate of 25–100%, commonly caused by sepsis that originates in the denuded skin or in the lungs. Survivors are at high risk of long-term morbidity including scarring, contractures, blindness, and esophageal strictures.

Heimbach et al (*JAMA* 1987; 257:2171–2175) report on a novel, standardized treatment protocol in which patients receive intensive care in a hospital burn center. The portocol is based on the following hypothesis: "the epidermal separation leaves denuded dermis that is completely viable and contains intact skin appendages, and if the dermal surface is meticulously protected, the patient will be rendered pain free, and the dermis will reepithelialize in about 14 days without developing infection or subsequent scarring . . . the associated conjunctivitis and mucositis will also heal without complications if the eyes are meticulously protected, the patient nourished, and sepsis prevented."

Under general anesthesia, the patient's wounds are washed, and all loose skin and blisters are removed. The denuded surfaces are then covered with porcine xenograft, which is stapled in place. The patient is maintained on a warmed, air-fluidized bed in the intensive care area of the burn unit. Daily care includes monitoring of fluid and electrolyte levels, enteral feeding through a nasogastric tube, cessation of any steroid therapy if possible, meticulous eye care on an hourly basis, control of pain with a mixture of methadone, hydroxyzine, and acetaminophen, and trimming of the xenograft in areas of healing.

The authors recommend against inserting central venous and bladder catheters, as they can provide a breeding ground for bacteria. Common topical antimicrobial agents should also be avoided, including silver sulfadiazine, mafenide acetate, and sulfonamide drugs. Whereas cadaver allograft and the less costly and more readily available porcine xenograft work equally well, synthetic dressings have not proven as effective.

Among the 19 patients described by Heimbach et al—with a mean skin loss of 75% ± 20% of total body surface area—16 survived, for a mortality rate of 16%. One of the three fatalities died of sepsis caused by an infected ventriculovenous shunt; two patients died after the epithelium had completely regrown. The authors report no major long-term morbidity among the survivors. TEN leaves "totally viable dermis and intact skin appendages. If the dermis can be protected from toxic detergents, salves, or desiccation, rapid resurfacing by proliferation of epithelium from the skin appendages will occur."

BOOKS RECEIVED

CARDIOVASCULAR SURGERY

Open Heart Surgery. Edited by John G. Callaghan, MD, and Joseph Wartak, MD. 178 pp, illustrated. New York, Praeger Publishers, 1986. $42.95 (hardcover)

HEALTH CARE

Employers' Guide to HMOs—Health Maintenance Organizations. By Leon J. Warshaw, MD, John Monaghan, EDM, and Preston A. Trombly, MMA. 64 pp. New York, The New York Business Group on Health, Inc, 1987. $15.00 (softcover)

Health Care and Its Costs: Can the U.S. Afford Adequate Health Care? Edited by Dr Carl J. Schramm. 301 pp, illustrated. New York, W.W. Norton & Co, 1987. $18.95 (hardcover)

The Physician as Manager. Second Edition. By John J. Aluise, PHD. 260 pp, illustrated. New York, Springer-Verlag, 1987. $35.00 (hardcover)

Financing Health Care. Economic Efficiency and Equity. By Steven R. Eastaugh. 720 pp, illustrated. Dover, Mass, Auburn House Publishing Co, 1987. $17.95 (paperback)

Health for the 80's: A Question of Survival. Volume 1. By Joseph M. Mauceri, MD. 222 pp. Effingham, Ill, National Holistics, Inc, 1980. $7.95 (paperback)

HISTORY OF MEDICINE

Johannes Müller and the Nineteenth-Century Origins of Tumor Cell Theory. Edited by L. J. Rather, Patricia Rather, and John B. Frerichs. 193 pp, illustrated. Canton, Mass, Watson Publishing International, 1986. $15.00 (hardcover)

The Great Break. A Short History of the Separation of Medical Science from Religion. By Joseph Mauceri, MD. 148 pp. Barrytown, NY, P-U-L-S-E Books, 1986. $9.95 (paperback)

The Development of American Physiology. Scientific Medicine in the Nineteenth Century. By W. Bruce Fye, MD. 308 pp. Baltimore, Md, The Johns Hopkins University Press, 1987. $35.00 (hardcover)

INFECTIOUS DISEASES

Infectious Diseases in Child Day Care: Management and Prevention. Edited by Michael T. Osterholm, Jerome O. Klein, Susan S. Aronson, and Larry K. Pickering. 175 pp, illustrated. Chicago, Ill, University of Chicago Press, 1987. $30.00 (hardcover)

INTERNAL MEDICINE

DeGowin and DeGowin's Bedside Diagnostic Examination. Fifth Edition. By Richard L. DeGowin, MD. 1,053 pp, illustrated. New York, Macmillan Publishing Co, 1987. $25.95 (hardcover)

LABORATORY MEDICINE

Interpretation of Diagnostic Tests. A Synopsis of Laboratory Medicine. By Jacques Wallach, MD. 825 pp, illustrated. Boston, Little, Brown & Co, 1981. $17.50 (paperback)

Studying a Study and Testing a Test. How to Read the Medical Literature. By Richard K. Riegelman, MD. 283 pp, illustrated. Boston, Little, Brown & Co, 1981. $17.50 (paperback)

LEGAL MEDICINE

The Deposition Guide. A Practical Handbook for Witnesses. By Larry G. Johnson, JD. 49 pp. Seattle, Wash, Law Forum Press, 1986. $7.95 (paperback)

Malpractice Depositions. Avoiding the Traps. By Raymond M. Fish, PHD, and Melvin E. Ehrhardt, MD. 160 pp. Oradell, NJ, Medical Economics Books, 1987. $24.95 (paperback)

MEDICAL ETHICS

Ethics in Emergency Medicine. By Kenneth V. Iserson, MD, Arthur B. Sanders, MD, Deborah R. Mathieu, PHD, and Allen E. Buchanan, PHD. 285 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1986. $39.50 (softcover)

MEDICAL WRITING

Medical Journalism. The Writing, Design and Production of Local Medical, Pharmaceutical and Hospital Publications. Edited by Craig D. Burrell, MD. 250 pp. New York, Sandoz Corporation. 1986. $15.00 (hardcover)

MISCELLANEOUS

Encounters with Qi. Exploring Chinese Medicine. By David Eisenberg, MD. 254 pp, illustrated. New York, Penguin Books, 1985. $6.95 (paperback)

The Parents' Emergency Medical Guide. By Robert Kaplan, MD. 274 pp, illustrated. New York, McGraw-Hill Book Co, 1987. $9.95 (paperback)

Webster's New World Medical Speller/Divider. By Joy H. Walworth. 265 pp. New York, Prentice Hall Press, 1987. $6.95 (softcover)

NEPHROLOGY

Self-Assessment in Nephrology. By Sanford E. Warren, CDR, MC, USNR; Gary M. Rabetoy, Capt, MC, USN; Coleman A. Mosley, Jr, CDR, MC, USN; and Mark S. Duke, LCDR, MC, USN. 258 pp. New York, Plenum Publishing Corp, 1987. $35.00 (softcover)

NUTRITION

Jean Carper's Total Nutrition Guide. Complete and Up-to-Date. By Jean Carper. 424 pp. New York, Bantam Books. $12.95 (paperback)

OBSTETRICS-GYNECOLOGY

Endocervical Carcinoma: A Cervicoscopic Atlas. By Minoru Ueki, MD, and Takashi Sano, MD. 88 pp, illustrated. St Louis, Mo, Ishiyaku Euro-America, Inc, 1987. $35.00 (hardcover)

Induced Abortion: A World Review 1986. Sixth Edition. By Christopher Tietze and Stanley K. Henshaw. 143 pp, illustrated. New York, The Alan Guttmacher Institute, 1986. $10.00 (softcover)

ONCOLOGY

The Basic Science of Oncology. Edited by Ian F. Tannock, MD, and Richard P. Hill, PHD. 398 pp, illustrated. Elmsford, NY, Pergamon Press, Inc, 1987. $37.50 (softcover)

Women and Cancer. Edited by Steven D. Stellman, PHD. 277 pp, illustrated. New York, The Haworth Press, Inc, 1987. $39.95 (hardcover)

PHARMACOLOGY

Drug Interactions Decision Support Tables. By Philip D. Hansten, PharmD. 152 pp. Spokane, Wash, Applied Therapeutics, Inc, 1987. $25.00 (softcover)

PUBLIC HEALTH

The State of the World's Children, 1987. Edited by James P. Grant. 148 pp, illustrated. New York, Oxford University Press, 1987. $8.95 (softcover)

PSYCHIATRY

The Good News About Depression. Cures and Treatment in the New Age of Psychiatry. By Mark S. Gold, MD. 328 pp, illustrated. New York Villard Books, 1986. $18.95 (hardcover)

Inpatient Group Psychotherapy. A Psychodynamic Perspective. By Cecil A. Rice, PHD, and J. Scott Rutan, PHD. 180 pp. New York, Macmillan Publishing Co, 1987. $19.95 (hardcover)

Sexual Aversion, Sexual Phobias, and Panic Disorder. By Helen Singer Kaplan, MD. 158 pp. New York, Brunner/Mazel Publishers, 1987. $25.00 (hardcover)

The Hyperactive Child, Adolescent, and Adult. Attention Deficit Disorder Through the Lifespan. By Paul H. Wender, MD. 162 pp. New York, Oxford University Press, 1987. $6.95 (paperback)

RADIOLOGY

A Multimodality Approach to Breast Imaging. By Saar Porrath, MD. 333 pp, illustrated. Rockville, Md, Aspen Publishers, Inc, 1986. $63.50 (hardcover)

SUBSTANCE ABUSE

Cocaine: Clinical and Biobehavioral Aspects. Edited by Seymour Fisher, PHD, Allen Raskin, PHD, and E. H. Uhlenhuth, MD. 256 pp, illustrated. New York, Oxford University Press, Inc, 1987. $24.95 (hardcover)

Alcohol and Drug Abuse Handbook. Edited by Roland E. Herrington, MD, George R. Jacobson, PHD, and David G. Benzer, DO. 490 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1987. $47.50 (hardcover)

SURGERY

Atlas of Advanced Surgery. Edited by Martin S. Litwin, MD. 198 pp, illustrated. New York, Yorke Medical Books, 1986. $95.00 (hardcover)

TOXICOLOGY

Handbook of Toxicology. Edited by Thomas J. Haley, PHD, and William O. Berndt, PHD. 697 pp, illustrated. New York, Hemisphere Publishing Corp, 1987. $110.00 (hardcover)

FROM THE LIBRARY

LITERATURE AND MEDICINE

The doctor should be an all-around man. It is not enough to be learned in medicine, for the knowledge of the physician has this quality that distinguishes it from the knowledge of all other professional men, except, perhaps, the soldier, or the navigator, that it must be readily available; and no man can have the necessary suppleness of intellect whose mind is muscle-bound, as it were, by a too limited application to one line of thought. Therefore, it is desirable that the medical man should indulge, at least moderately, in the recreation that produces the greatest intellectual broadening, which is literature. It is scarcely necessary to refer to the singular ability, in medicine, of literary doctors, Charcot, Marion Sims, our own Mitchell and a host of others, of the added influence that accrues to an attractive literary style, of which nearly all the classics are examples. But there is no reason why, in reading general literature, a man should not add to his medical information. Clinical histories of the most charming and accurate nature are observed in the autobiographies and memoirs of the last century, and some of the cases described, of undoubted authenticity, are both rare and instructive. Take, for example, the remarkable cure of Pepys, whose lithotomy wound reopened after many years, the excellent description of cirrhosis of the liver in Fielding's "Journal of a Voyage to Lisbon," the curious histories of poisonings and sickness often accompanied by the autopsy records, in Saint Simon's memoirs, or the cases recorded in romances, undoubtedly obtained from good authorities. The works of the great realists, De Foe, Smollett, Balzac, Zola and a hundred others abound in such reports. "Reading makes a full man," and the physician who has filled his mind with the understanding of the masters of literature will be better able to handle the human side of his patients; if he has studied the subtleties of their observation, he will be better able to cope with the mysteries of diagnosis. Medicine, any more than statecraft, will never be an exact science, and the greatest physician, therefore, will not be the men of mere technical training, but those who add to this knowledge of the human character that is only acquired by the study of its highest production.—*Philadelphia Medical Journal.*

(*NY State J Med* 1904; 4:11–12)

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Ludwig Auerbach, MD, Largo, Florida. Died April 27, 1987; age 87. Dr Auerbach was a 1926 graduate of Medizinische Fakultaet der Julius Maximilians Universitaet, Wurzberg, Bayern, Germany. He was a member of the Medical Society of the County of Saint Lawrence and the Medical Society of the State of New York.

Maxwell Berkowitz, MD, Brooklyn. Died January 19, 1987; age 90. He was a 1918 graduate of New York University School of Medicine, New York. Dr Berkowitz was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Manfred Braun, MD, New York. Died February 28, 1987; age 82. Dr Braun was a 1929 graduate of Universitaet Graz, Medizinische Fakultaet, Graz, Austria. He was a Fellow of the American Psychiatric Association and a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Walter Brinitzer, MD, New York. Died May 12, 1987; age 77. Dr Brinitzer was a 1935 graduate of Medizinische Fakultaet der Christian Albrechts Universitaet, Kiel Schleswig Holstein, Germany. He was a member of the American Psychiatric Association, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Monroe M. Broad, MD, Jamaica. Died May 19, 1987; age 78. He was a 1933 graduate of the University of St Andrews School of Medicine, Dundee, Scotland. Dr Broad was a member of the American Society of Abdominal Surgeons, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Mitchell A. Brucker, MD, Pompano Beach, Florida. Died May 5, 1987; age 72. He was a 1941 graduate of Louisiana State University School of Medicine, New Orleans. Dr Brucker was a Fellow of the American College of Obstetricians and Gynecologists, a Diplomate of the American Board of Obstetrics and Gynecology, and a member of the New York County Medical Society and the Medical Society of the State of New York.

Jacob Clahr, MD, New York. Died April 8, 1987; age 83. Dr Clahr was a 1928 graduate of the State University of New York Health Science Center at Brooklyn. He was a Fellow of the American College of Obstetricians and Gynecologists and a Diplomate of the American Board of Obstetrics and Gynecology. His memberships included the Academy of Medicine, the American Public Health Association, the Bronx County Medical Society, and the Medical Society of the State of New York.

Ralph Clarence Drews, MD, Morrisville, Vermont. Died May 10, 1987; age 83. Dr Drews was a 1930 graduate of Columbia University College of Physicians and Surgeons, New York. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Maurice Abraham Egand, MD, Spring Valley. Died April 22, 1987; age 100. Dr Egand was a 1921 graduate of Kazan Medical Institute, Kazan, USSR. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Leo David Freydberg, MD, Hollywood, Florida. Died April 10, 1987; age 77. He was a 1936 graduate of Cornell University Medical College, New York. Dr Freydberg was a Fellow of the American College of Physicians, a Diplomate of the American Board of Internal Medicine, and a member of the Medical Society of the County of Rensselaer and the Medical Society of the State of New York.

Asa Ben Friedmann, MD, New York. Died April 24, 1987; age 86. He was a 1924 graduate of the State University of New York Health Science Center at Brooklyn. Dr Friedmann was a Fellow of the American College of Radiology and a Diplomate of the American Board of Radiology. His memberships included the New York Roentgen Society, the Academy of Medicine, the American Radium Society, the Radiological Society of North America, Inc, the New York County Medical Society, and the Medical Society of the State of New York.

Morris Goldberger, MD, New Rochelle. Died May 6, 1987; age 101. Dr Goldberger was a 1907 graduate of New York University School of Medicine, New York. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Martin Gerhard Goldner, MD, Palo Alto, California. Died April 1987; age 85. Dr Goldner was a 1926 graduate of Medizinische Fakultaet der Johann Wolfgang Goethe Universitaet, Frankfurt-am-Main, Hessen, Germany. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Solon H. Gottlieb, MD, New York. Died May 11, 1987; age 61. Dr Gottlieb was a 1952 graduate of State University of New York at Buffalo School of Medicine, Buffalo. Dr Gottlieb was a Diplomate of the American Board of Obstetrics and Gynecology and a member of the American Fertility Society, the Pan American Medical Association, the New York County Medical Society, and the Medical Society of the State of New York.

Emil Granet, MD, New York. Died May 15, 1987; age 88. He was a 1923 graduate of Columbia University College of Physicians and Surgeons, New York. Dr Granet was a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Colon and Rectal Surgery. His memberships included the Academy of Medicine, the American Society of Colon and Rectal Surgeons, the New York County Medical Society, and the Medical Society of the State of New York.

John E. Horn, MD, Painted Post. Died April 8, 1987; age 65. Dr Horn was a 1947 graduate of the University of Rochester School of Medicine-Dentistry, Rochester. He was a member of the Steuben County Medical Society and the Medical Society of the State of New York.

Samuel C. Johnson, MD, Buffalo. Died May 4, 1987; age 83. He was a 1931 graduate of Meharry Medical College, Nashville. Dr Johnson was a member of the Academy of Medicine, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Joseph P. Kasnetz, MD, Rumson, New Jersey. Died April 28, 1987; age 90. Dr Kasnetz was a 1921 graduate of New York Medical College, New York. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Barnard Kleiger, MD, Scarsdale. Died April 25, 1987; age 76. He was a 1936 graduate of the University of Michigan Medical School, Ann Arbor. Dr Kleiger was a Fellow of the American College of Surgeons, a Diplomate of the American Board of Orthopedic Surgery, and a member of the Academy of Medicine, the American Academy of Orthopaedic Surgeons, the New York County Medical Society, and the Medical Society of the State of New York.

Louis H. Klinger, MD, New York. Died May 13, 1987, age 83. Dr Klinger was a 1930 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Dudley Leibowitz, MD, Boynton Beach, Florida. Died May 13, 1987; age 66. Dr Leibowitz was a 1945 graduate of Tulane University School of Medicine, New Orleans. He was a Diplomate of the American Board of Pediatrics and a member of the American Academy of Pediatrics, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Charles Lerner, MD, Cuyahoga Flats, Ohio. Died February 22, 1987; age 92. Dr Lerner was a 1921 graduate of New York University School of Medicine, New York. He was a member of the Sullivan County Medical Society and the Medical Society of the State of New York.

Samuel G. Levin, MD, Brooklyn. Died March 26, 1987; age 85. He was a 1933 graduate of George Washington University School of Medicine, Washington. Dr Levin was a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Orthopedic Surgery. His memberships included the American Academy of Orthopaedic Surgeons, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Irving Edwin Lieberman, MD, Brooklyn. Died February 21, 1987; age 78. Dr Lieberman was a 1933 graduate of New York University School of Medicine, New York. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Malcolm James Magovern, MD, Saratoga Springs, NY. Died May 9, 1987; age 89. He was a 1928 graduate of Georgetown University School of Medicine, Washington. Dr Magovern was a member of the Medical Society of the County of Saratoga and the Medical Society of the State of New York.

Raymond W. Mitchell, Jr, MD, Hamburg. Died May 9, 1987; age 70. He was a 1943 graduate of State University of New York at Buffalo School of Medicine. Dr Mitchell was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Babette Neuberg, MD, Albany. Died April 11, 1987; age 96. Dr Neuberg was a 1916 graduate of Medizinische Fakultaet, Albert Ludwigs Universitaet, Freiburg in Breisgau, Baden-Wurttenberg, Germany. She was a member of the Medical Society of the County of Rockland and the Medical Society of the State of New York.

Isidore I. Perlmutter, MD, Rochester. Died May 4, 1987; age 75. He was a 1940 graduate of Medizinische Fakultaet der Universitaet Bern, Bern, Switzerland. Dr Perlmutter was a member of the American Geriatrics Society, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Daniel Joseph Riordan, MD, Hamburg. Died April 22, 1987; age 63. Dr Riordan was a 1947 graduate of State University of New York at Buffalo School of Medicine. He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the New York State Society of Internal Medicine, the American Rheumatism Association, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Edward I. Salisbury, MD, Manhasset. Died April 19, 1987; age 94. He was a 1915 graduate of Jefferson Medical College of Thomas Jefferson University, Philadelphia. Dr Salisbury was a Fellow of the American College of Surgeons and a member of the New York Society of Tropical Medicine, the American Society of Tropical Medicine and Hygiene, the New York County Medical Society, and the Medical Society of the State of New York.

Harry Shapiro, MD, Johnson City. Died May 22, 1987; age 82. Dr Shapiro was a 1933 graduate of University of Michigan Medical School, Ann Arbor. He was a member of the Broome County Medical Society and the Medical Society of the State of New York.

Augusta Skodnick, MD, Savannah, Georgia. Died May 26, 1987; age 96. Dr Skodnick was a 1922 graduate of Medical College of Pennsylvania, Philadelphia. She was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Frank Spaeth, MD, Binghamton. Died May 24, 1987; age 78. He was a 1939 graduate of the State University of New York Health Science Center at Brooklyn. Dr Spaeth was a member of the Academy of Medicine, the Bronx County Medical Society, and the Medical Society of the State of New York.

Ralph F. Traver, MD, Tulsa, Oklahoma. Died April 28, 1987; age 84. He was a

1928 graduate of Harvard Medical School, Boston. Dr Traver was a Fellow of the American College of Surgeons, a Diplomate of the American Board of Surgery, and a member of the Steuben County Medical Society and the Medical Society of the State of New York.

John O. Vieta, MD, New York. Died May 10, 1987; age 75. Dr Vieta was a 1938 graduate of Northwestern University Medical School, Chicago. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the Academy of Medicine, the New York State Surgical Society, the Society of Surgical Oncology, the New York County Medical Society, and the Medical Society of the State of New York.

Laurence E. Viola, MD, Savannah, Georgia. Died May 13, 1987; age 80. He was a 1934 graduate of New York Medical College, New York. Dr Viola was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Qualified Fellow of the International College of Surgeons. He was a member of the Richmond County Medical Society and the Medical Society of the State of New York.

Selma L. Wehl, MD, Brooklyn. Died April 1, 1987; age 89. She was a 1923 graduate of Medizinische Fakultaet der Universitaet Hamburg, Hamburg, Germany. Dr Wehl was a Diplomate of the American Board of Pediatrics and a member of the American Academy of Pediatrics, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Paul A. Weinrauch, MD, Hartsdale. Died December 18, 1986; age 74. Dr Weinrauch was a 1938 graduate of Faculte de Medecine de l'Universite de Lausanne, Lausanne, Switzerland. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1983; 83:1089-1094). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith[2] reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al*. Names of journals should be abbreviated according to *Index Medicus* and underlined.

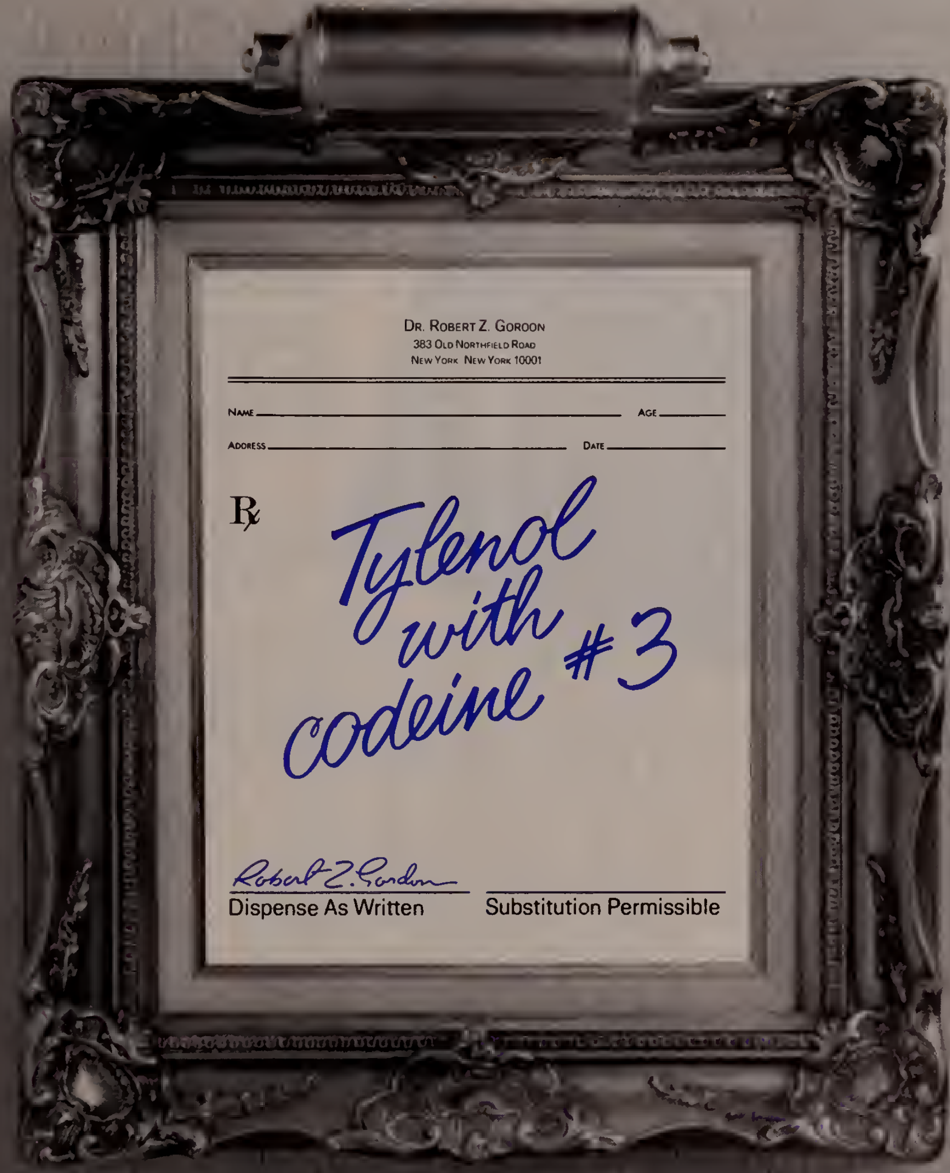
Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83: 925-927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337-338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, P.O. Box 5404, 420 Lakeville Road, Lake Success, NY 11042.



DR. ROBERT Z. GORDON
383 OLD NORTHFIELD ROAD
NEW YORK NEW YORK 10001

NAME ______ AGE ______

ADDRESS ______ DATE ______

℞

Tylenol
with
codeine #3

Robert Z. Gordon
Dispense As Written

Substitution Permissible

CREATE A SIGNED ORIGINAL

to be sure your patients get...

Tylenol®
ACETAMINOPHEN
with codeine
phosphate
TABLETS Ⓒ III ELIXIR Ⓒ V

Tablets: Contain Codeine Phosphate*: No. 3 — 30 mg; No. 4 — 60 mg — plus Acetaminophen 300 mg.
Elixir: Each 5 mL contains 12 mg Codeine Phosphate* plus 120 mg Acetaminophen (Alcohol 7%).
*Warning: May be habit forming.

GIVE ADDED CHANCE TO YOUR RESPIRATOR DEPENDENT PATIEN TO IMPROVE DIFFUSION AND GET OFF THE RESPIRATOR

with
Effective Lobe to Lobe
Aspirations with the
Rockey-Thompson Bronchia
Suction Catheters will help.

AVAILABLE IN SETS OF 4:
F14, F16, F18, F20

For more information call or write:

from:
R.E.P. DIVISION PECK PACKAGING COR
528 Carter Street, New Canaan,
CONN. 06840 Tel. (203) 972-0119

tions. Harvard School of Public Health. Contact: Office of Continuing Education, Department A, Harvard School of Public Health, 677 Huntington Ave, Boston, MA 02115. Tel: (617) 732-1171.

NEW MEXICO

Sept. 27–30. **Plastic Surgery of the Breast—The Art and Science—1987 Update.** 18 Cat 1 Credits. Eldorado Hotel, Santa Fe. Contact: Plastic Surgery Educational Foundation, 233 North Michigan Ave, Suite 1900, Chicago, Illinois 60601.

OHIO

Sept. 14–17. **Medical Oncology—Principles of Patient Management.** Hyatt Regency Hotel, Columbus. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

PENNSYLVANIA

Sept. 9–11. **Health Promotion and Preventive Care for the Elderly: A Concept Whose Age Has Come.** Penn Tower Hotel, Philadelphia. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

Sept. 28–30. **Community Re-entry Following Head Injury.** Vista Hotel, Pittsburgh. Contact: Training Department, The Rehabilitation Institute of Pittsburgh, 6301 Northumberland St, Pittsburgh, PA 15217. Tel: (412) 521-9000.

TENNESSEE

Sept. 14–18. **Recent Advances in Internal Medicine.** Vanderbilt University School of Medicine, Nashville. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

VIRGINIA

Sept. 28–30. **Impact on the Fetus of Parental Sexually Transmitted Disease.** Marriott Crystal Gateway, Arlington. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd Street, New York, NY 10021.

AROUND THE WORLD

BANGKOK, HONG KONG, SINGAPORE

Sept. 12–15. **Family Medicine—Old and New.** 26 Cat 1 Credits. Bangkok, Hong Kong, and Singapore. Contact: Dawne Ryals, Ryals and Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

CANADA

Sept. 28–30. **An Update on Cerebral Vascular Disease.** University Hospital, London, Ontario. Info: Dr V. C. Hachinski, Rm Pl-106, The John P. Robarts Research Institute, PO Box 5015, London, Ontario, Canada N6A 5K8. Tel: (519) 663-3787. □

REAL ESTATE FOR SALE OR RENT—CONT'D

OFFICE SPACE AVAILABLE: Throgs Neck section of Bronx. Modern medical complex, 1500 square feet, part time/full time. Call (212) 409-9494.

EAST SIXTIES, PROFESSIONAL BUILDING. The preferred location for internists, cardiologist, rheumatologist. 7 treatment rooms, 2 reception areas to share with three other internists. Share NYC licensed lab, radiology, thermography. Full or part time. (212) 838-2860.

PRACTICE LOCATION: Private Home with Duplex Apartment, spacious grounds, detached garage, and a fully-equipped centrally air-conditioned, 9 room Medical Suite; including large waiting room, business office, consultation room, nurses station, pharmacy, therapy room, 3 complete examining rooms, x-ray, clinical laboratory, 2 bathrooms. Apartment and office each suitable for sub-lease and added income. Turn-key operation ready for active solo or multi-specialty group. Large middle and upper class residential community, South Shore, Long Island. Write: Box 414, West Lawrence, NY 11690.

WESTCHESTER NEW YORK RESIDENCE, 26 minutes to NYC. Substantial English Georgian 6 br., sun rm., artist alcove, circa 1920, stripped, replastered, refinished mahogany, brick-stone-slate, 1/3 green acre, giant oaks. Central a/c, manageable-safe-secure-secluded. Turn of century area. $850K, Dr. Skalafuris (914) 668-9108, (216) 832-5077 or (202) 462-3344.

PRIME MIDTOWN LOCATION. Office space for rent in newly refurbished Madison Medical Building, New York City, Madison Avenue between 32-33rd Streets. 800 usable sq. feet. Long term lease and sublet clause available. Secretarial area, waiting room, two exam rooms. Call Dr. Siegel at (212) 340-6431.

SUBLET THREE ROOMS just renovated office near all transportation (North/South bus, East/West bus, Express bus and subway all on corner). M. S. Goldenberg, 157 East 86th Street, New York, NY 10028. (212) 860-2311.

SPACIOUS PARK AVENUE and 72nd Street office. Sale or share. Newly renovated with operating room, consultation and examining rooms, reception area, etc. (212) 517-4410.

FOR RENT—MASPETH QUEENS. Busy location, modern ground level complex. 10 treatment rooms, 2 recovery rooms, lab, staff lounge, reception area and office. 2900 sq. feet, 2 a/c and heating zones. Can share with other professionals. No fee. Call owner (718) 786-3405 for more information.

NEW PENTHOUSE MEDICAL OFFICE for lease on Long Island Expressway in Fresh Meadows, Queens, New York. Close proximity to all major highways and transportation. 2,000 sq. ft. available. Will build to suit. Elevators, solarium and on-site parking. Call (718) 463-8800.

PARADISE MINI-ESTATE White Plains Vicinity. Thirty minutes Mid-Town Manhattan, custom built home on Cul-de-Sac with 4 bedrooms, 3 baths. Secluded, lushly landscaped, includes gourmet eat in kitchen with skylights, cathedral living room and formal dining room, in ground pool, greenhouse, steamroom, 2 car remote control garage, family room with wet bar, fireplace wall and entertainment wall. Original owner, $575,000.00. Call (914) 592-9455.

"SELLING PRACTICES IS OUR BUSINESS"

Want to maximize the return on your investment? Before you buy or sell, call for an appraisal to assure the best financial and transfer terms. We are a full service practice broker, not simply a listing service. Since 1981, Countrywide has been instrumental in helping hundreds of doctors like yourself buy and sell their practices. We guide you through the entire sales process from initial meeting to closing. Our offices serve New York, New Jersey and New England.

Countrywide Business Brokerage, Inc.
319 East 24th Street, Suite 23-G, New York, NY 10010
(212) 686-7902 (203) 868-3666 (617) 232-1075

Medical Society of the State of New York
ANNUAL CME ASSEMBLY
APRIL 22-24, 1988
The New York Hilton, New York City
Combined With The Annual Meeting of
The House of Delegates
APRIL 21-24

MISCELLANEOUS

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta, GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuarial certification and employee statements of participation—partnership agreements and professionals incorporation—No insurance required—references upon request—Wachstock and Dienstag Attorneys at Law, 122 Cutter Mill Road, Great Neck, NY 11021 (516) 773-3322.

FIRM OF GERALD GOLDBERG, CPA-ATTORNEY, formerly with IRS, announces his availability to prepare taxes and plan under changing tax environment, accounting, tax appeals, legal representation or practice transfers. (212) 406-1156 and (516) 565-6260.

SIXTH ANNUAL ELLA T. GRASSO MEMORIAL CONFERENCE, November 18, 1987, The Park Plaza Hotel, New Haven, Conn. For registration and further information (after August 1, 1987): Yale University School of Medicine, Office of Graduate and Continuing Education, 333 Cedar Street, P.O. Box 3333, New Haven, CT 06510, (203) 785-4578.

MISCELLANEOUS—CONT'D

HAVE YOU RECEIVED ANY COMMUNICATION FROM THE OFFICE OF PROFESSIONAL MEDICAL CONDUCT? If affirmative, contact Susan Kaplan, Attorney-at-Law, (212) 877-5998. Practice limited to the crucial legal problems directly affecting your license to practice medicine—with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professions), and as an Assistant District Attorney in Nassau County. Susan Kaplan, Esq., 165 West End Avenue, Suite 27P, New York, N.Y. 10023.

ADVICE, COUNSEL & REPRESENTATION of physicians and other health care professionals in licensure and professional conduct matters, reimbursement issues and practice-related litigation. David E. Ruck, Esq., former Chief, Criminal Division, Office of the New York State Special Prosecutor for Medicaid Fraud Control and Alain M. Bourgeois, Esq., former Acting Justice, Supreme Court of the State of New York, announce the opening of their offices for the practice of law under the name of Bourgeois & Ruck, 101 Park Avenue, New York, New York 10178. Telephone 212-661-8070.

PROFESSIONAL MISCONDUCT ATTORNEYS. William L. Wood Jr., formerly Executive Director of the New York State Office of Professional Discipline and Anthony Z. Scher, formerly Director of Prosecutions, announce the opening of their law office, Wood & Scher. Our recent tenure as chief enforcement officers has given us experience which will allow us to represent physicians in professional misconduct proceedings, malpractice licensure, license restoration, controlled drug proceedings, reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practitioners. Wood & Scher, Attorneys at Law, One Chase Road, Scarsdale, New York 10583. Telephone 914-723-3500.

In New York City, for the Newest and Finest Medical Space, Remember:

THE 4 ESSENTIAL REQUIREMENTS

1. LOCATION!
Liberty Residences at Battery Park City

2. LOCATION!
Windsor Pavilion
(3rd Avenue & 32nd Street)

3. LOCATION!
205 East 64th Street (3rd Avenue)

AND

4. LOCATION!
Normandie Pavilion
(3rd Avenue & 95th Street)

For Further Information Contact Lester Schwalb
(212) 708-0846
Milstein Properties Corp.

It's Here At Last...
A Choice of Two Low-Cost Practice Evaluations.

Basic Practice Evaluation
Provides a benchmark for your practice.

Expanded Option Evaluation
Provides 15 different option categories, with 180 evaluations, under a variety of conditions.

Here's Why You Should Consider a Pro-Tech Practice Evaluation:

- Analysis can be used in buying, selling, merging or dissolving of a practice.
- Analysis can serve to confirm other appraisals.
- Analysis is processed quickly — usually within 10 days.

Get the hard facts.

Name ______ Phone ______
Address ______
City ______ State ______ Zip ______

Pro-Tech Information Systems
© 1986 All Rights Reserved

240 Cedar Knolls Rd., Suite 310, Cedar Knolls, NJ 07927-9990

Exclusive Marketing Agents: Sarantos & Co., Inc.

Doctor . .

Do you, or a physician you know, have a problem with alcohol, drugs, or emotional illness?

If so, please contact the Medical Society of the State of New York's Committee for Physicians' Health, whose function is to assist, *confidentially.*

Telephone: 516-488-7777.

Index to Advertisers

Ayerst Laboratories	13A, 14A, 15A, 16A
St. Clare's Hospital	7A
Classified Advertising	6A, 11A
Family Practice Recertification Magazine	4A
Knoll Pharmaceuticals	5A, 6A
Eli Lilly and Company	8A
Marion Laboratories	1A, 2A
McNeil Laboratories	9A
Milstein Properties Corp.	12A
Peck Packaging Corp.	10A
Pro-Tech Information Systems	12A
Roche Laboratories	3rd & 4th Cove
Upjohn Company	2nd Cove

To show you how many hypertensives stayed on INDERAL® LA (PROPRANOLOL HCl) after a major nationwide trial...

...we had
to find
just the
right room.

60,073 patients (90%) who started on INDERAL® LA stayed on INDERAL LA.[1*]

Surprising? Not really.

Because most patients on INDERAL LA (propranolol HCl) don't even know it's working.

A recent double-blind, placebo-controlled, crossover study in 138 hypertensive patients[2] revealed that INDERAL LA has a side effects profile unsurpassed by atenolol or metoprolol—which shows how well-tolerated once-daily INDERAL LA can be.

Sole therapy or concomitant therapy? Fifty-nine percent of the time, INDERAL LA stood on its own.

The patients in the nationwide compliance trial were no different from yours. Generally when the antihypertensive regimen is complicated, compliance may become a problem. So, the effectiveness of INDERAL LA as once-daily monotherapy is a big plus. Of the remaining hypertensives in the program, 36% were treated merely with the addition of a diuretic to INDERAL LA.

For the noncompliant patients in your practice, INDERAL LA may well be the answer.

Almost 20,000 of the patients in the nationwide compliance trial were identified as having been noncompliant with their previous antihypertensive therapy. Their physicians reported that 88% showed improved compliance when placed on once-daily INDERAL LA.

Control, comfort, and compliance

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl) LONG ACTING CAPSULES

Like conventional INDERAL Tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree, and bronchial asthma.

*After a 30-day trial with INDERAL LA, physicians reported that 90% of the patients would remain on INDERAL LA.

The one you know best keeps looking better

NOW AVAILABLE...

NEW LOW DOSE

INDERAL® LA 60 mg

(PROPRANOLOL HCl) LONG ACTING CAPSULES

Please see next page for brief summary of prescribing information

The one you know best keeps looking better

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION SEE PACKAGE CIRCULAR)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. INDERAL LA is formulated to provide a sustained release of propranolol hydrochloride INDERAL LA is available as 60 mg, 80 mg 120 mg, and 160 mg capsules

CLINICAL PHARMACOLOGY. INDERAL is a nonselective, beta-adrenergic receptor-blocking agent possessing no other autonomic nervous system activity It specifically competes with beta-adrenergic receptor-stimulating agents for available receptor sites When access to beta-receptor sites is blocked by INDERAL the chronotropic, inotropic and vasodilator responses to beta-adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (60, 80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL Tablets The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg-for-mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose When changing to INDERAL LA from conventional propranolol a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product INDERAL LA can provide effective beta blockade for a 24-hour period

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina palpitations, and syncope INDERAL LA also improves exercise performance The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first-degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

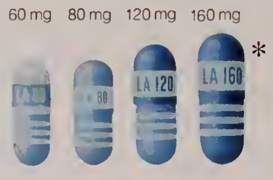
WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl)

LONG ACTING CAPSULES



IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible)

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following *abrupt* discontinuance of INDERAL therapy Therefore, when discontinuance of INDERAL is planned, the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema) — PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, eg, dobutamine or isoproterenol However, such patients may be subject to protracted severe hypotension Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers

DIABETES AND HYPOGLYCEMIA Beta blockers should be used with caution in diabetic patients if a beta-blocking agent is required Beta blockers may mask tachycardia occurring with hypoglycemia, but other manifestations such as dizziness and sweating may not be significantly affected Following insulin-induced hypoglycemia, propranolol may cause a delay in the recovery of blood glucose to normal levels

THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm Propranolol may change thyroid function tests, increasing T_4 and reverse T_3, and decreasing T_3

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker In one case, this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. GENERAL Propranolol should be used with caution in patients with impaired hepatic or renal function INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that INDERAL may interfere with the glaucoma screening test Withdrawal may lead to a return of increased intraocular pressure

CLINICAL LABORATORY TESTS Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension

Caution should be exercised when patients receiving a beta blocker are administered a calcium-channel-blocking drug, especially intravenous verapamil, for both agents may depress myocardial contractility or atrioventricular conduction On rare occasions, the concomitant intravenous use of a beta blocker and verapamil has resulted in serious adverse reactions, especially in patients with severe cardiomyopathy, congestive heart failure or recent myocardial infarction

Aluminum hydroxide gel greatly reduces intestinal absorption of propranolol

Ethanol slows the rate of absorption of propranolol

Phenytoin, phenobarbitone, and *rifampin* accelerate propranolol clearance

Chlorpromazine, when used concomitantly with propranolol, results in increased plasma levels of both drugs

Antipyrine and *lidocaine* have reduced clearance when used concomitantly with propranolol

Thyroxine may result in a lower than expected T_3 concentration when used concomitantly with propranolol

Cimetidine decreases the hepatic metabolism of propranolol, delaying elimination and increasing blood levels

Theophylline clearance is reduced when used concomitantly with propranolol

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

PREGNANCY Pregnancy Category C INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose

There are no adequate and well-controlled studies in pregnant women INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

NURSING MOTHERS: INDERAL is excreted in human milk. Caution should be exercised when INDERAL (propranolol HCl) is administered to a nursing woman.

PEDIATRIC USE Safety and effectiveness in children have not been established

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy

Cardiovascular Bradycardia, congestive heart failure, intensification of AV block, hypotension, paresthesia of hands, thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type

Central Nervous System Light-headedness, mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia, visual disturbances; hallucinations, vivid dreams, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics. For immediate formulations, fatigue, lethargy, and vivid dreams appear dose related

Gastrointestinal: Nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis

Allergic Pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress

Respiratory Bronchospasm

Hematologic: Agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune In extremely rare instances, systemic lupus erythematosus has been reported

Miscellaneous: Alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily If patients are switched from INDERAL Tablets to INDERAL LA Capsules, care should be taken to assure that the desired therapeutic effect is maintained INDERAL LA should not be considered a simple mg-for-mg substitute for INDERAL INDERAL LA has different kinetics and produces lower blood levels Retitration may be necessary, especially to maintain effectiveness at the end of the 24-hour dosing interval.

HYPERTENSION — *Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic The dosage may be increased to 120 mg once daily or higher until adequate blood-pressure control is achieved The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage of 640 mg may be required The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks

ANGINA PECTORIS — *Dosage must be individualized.* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three- to seven-day intervals until optimal response is obtained. Although individual patients may respond at any dosage level, the average optimal dosage appears to be 160 mg once daily In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS)

MIGRAINE — *Dosage must be individualized* The initial oral dose is 80 mg INDERAL LA once daily The usual effective dose range is 160-240 mg once daily. The dosage may be increased gradually to achieve optimal migraine prophylaxis If a satisfactory response is not obtained within four to six weeks after reaching the maximal dose, INDERAL LA therapy should be discontinued It may be advisable to withdraw the drug gradually over a period of several weeks

HYPERTROPHIC SUBAORTIC STENOSIS — 80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE — At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use

*The appearance of these capsules is a registered trademark of Ayerst Laboratories

---

REFERENCES:

1. INDERAL LA National Compliance Evaluation Program Data on file, Ayerst Laboratories
2. Ravid M, Lang R, Jutrin I: The relative antihypertensive potency of propranolol, oxprenolol, atenolol, and metoprolol given once daily *Arch Intern Med* 1985, 145 1321-1323

1R7119/887

Ayerst

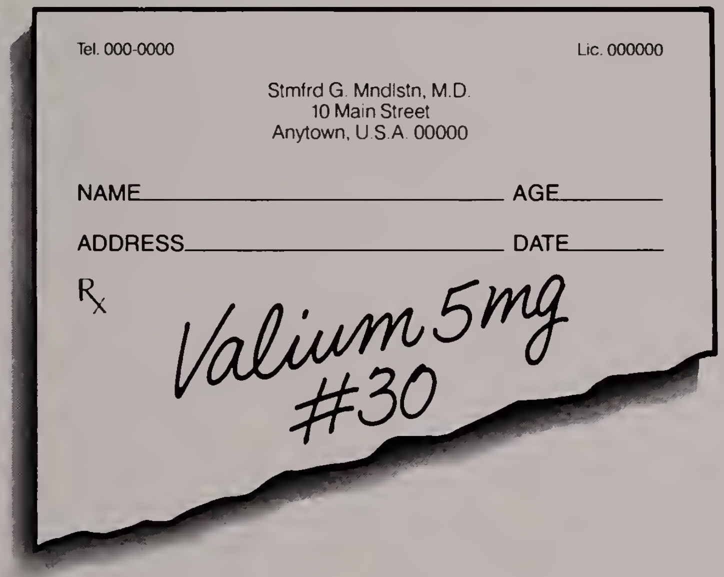
AYERST LABORATORIES
New York, NY 10017

© 1987 Ayerst Laboratories

Do Not Substitute

VALIUM®
brand of
diazepam/Roche Ⓘⅴ

The One You Know Best


Tel. 000-0000 Lic. 000000

Stmfrd G. Mndlstn, M.D.
10 Main Street
Anytown, U.S.A. 00000

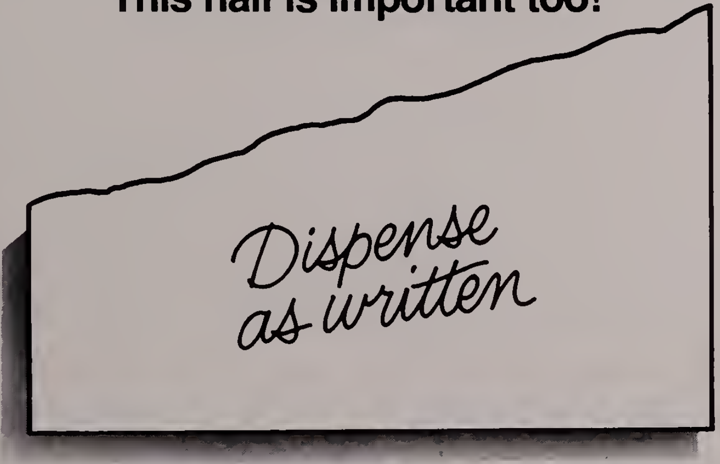
NAME________ AGE____

ADDRESS________ DATE____

℞
Valium 5mg
#30

Are you writing only half a prescription for it?

This half is important too!


Dispense
as written

Be sure to write a complete prescription.

Specify "Dispense as written."

ROCHE
Roche Products Inc.
Manati, Puerto Rico 00701

Copyright © 1987 by Roche Products Inc.
All rights reserved.

State flag of New York

Flag It.

To complete your prescription,
be sure to specify
"Dispense As Written."
This "flags" both pharmacist and patient
that you want the *brand* to be dispensed.
And it protects your decision.

VALIUM®
brand of
diazepam/Roche Ⓘⓥ

scored tablets

2 mg 5 mg 10 mg

The one you know best.

The cut out "V" design is a registered trademark
of Roche Products Inc.

ROCHE®

STACKS

NEW YORK STATE JOURNAL OF MEDICINE

SEPTEMBER 1987 • Volume 87, Number 9

MEDICAL SOCIETY OF THE STATE OF NEW YORK ORGANIZED 1807

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA
SEP 21 1987
M.D.S.

Contents

COMMENTARIES

Fibronectin deficiency following cardiopulmonary bypass 487
THOMAS M. SABA, PHD

Corticosteroid treatment of sarcoidosis—who needs it? 490
HAROLD L. ISRAEL, MD, MPH

The role of the physician as the patient's advocate 491
LEON J. WARSHAW, MD

RESEARCH PAPERS

Fibronectin levels during cardiopulmonary bypass 493
ALEXANDER W. GOTTA, MD; STEVEN CARSONS, MD; LAWRENCE ABRAMS, MD; ANN E. KEANY, CRNA

Corticosteroid therapy in sarcoidosis: A five-year, controlled follow-up study 496
MAHFOUZ H. ZAKI, MD, DRPH; HAROLD A. LYONS, MD; LINDA LEILOP, MD; CHIN TANG HUANG, MD

ESSAYS IN PEDIATRICS

The discovery of the first drug effective in treating acute lymphoid leukemia in children 500
CARL POCHEDLY, MD

HISTORY

Physician framers of the Constitution 504
FLORENCE KAVALER, MD, MPH; DAVID METZ, MPA; ANN B. COHEN, MSL

SPECIAL ARTICLE

Mr Keane's tree 509
PASCAL JAMES IMPERATO, MD

CASE REPORTS

Diaphragmatic rupture due to blunt trauma: The case for timely intervention 512
LATHAM B. MURRAY, MD; ROGER W. MACMILLAN, MD; TURNER OSLER, MD; JOHN E. OLSON, MD

Bilateral diaphragm paralysis associated with topical cardiac hypothermia 514
MANUEL R. CABRERA, MD, JOHN R. EDSALL, MD

Bochdalek hernia in adulthood 516
RICHARD FINE, MD; EDGAR BORRERO, MD; ALEX STONE, MD

Radionuclide imaging in the diagnosis of diaphragmatic hernia associated with neonatal group B streptococcal sepsis 518
ROBERT CHECOLA, MD; STEPHEN R. KANDALL, MD; FUKIAT ONGSENG, MD; SYLVAIN M. WEINBERGER, MD; RICHARD GOLDFARB, MD

LETTERS TO THE EDITOR

Diaphragmatic hernia 520
ROBERT H. POE, MD, CHARLES D. MASKIELL, MD

Computed tomography of Morgagni hernia 520
WILLIAM H. SUTRO, MD, STEVEN J. KING, MD

Tissue injury and breast carcinoma 521
A N PAPAIOANNOU, MD

Subcutaneous nonparenchymal hemangioma of the breast 521
JULIE S. MITNICK, MD; MATTHEW HARRIS, MD; HELEN FEINER, MD

BOOK REVIEWS 523

NEWS BRIEFS 527

MEDICAL SOCIETY NEWS 529

MEDICAL MEETINGS AND LECTURES 9A

Motrin® 800 TABLETS mg

ibuprofen

Extra strength

Convenience

Economy

Upjohn

A Century of Caring

1886·1986

© 1986 The Upjohn Company

J-6138 January 1986

Give your angina patients what they're missing...

CARDIZEM®: FEW SIDE EFFECTS

diltiazem HCl/Marion

90 mg GREATER DOSAGE FLEXIBILITY
NEW 120 mg

- ***Antianginal action includes dilatation of coronary arteries, a decrease in vascular resistance/afterload, and a reduction in heart rate***
- ***Proven efficacy when used alone in angina[1]***
- ***Compatible with other antianginals[2,3]****
- ***A safe choice for angina patients with coexisting hypertension, asthma, COPD, or PVD[4,5]***

****See Warnings and Precautions.***

Please see brief summary of prescribing information on the next page.

CARDIZEM® FEW SIDE EFFECTS IN ANTIANGINAL THERAPY

diltiazem HCl/Marion

60 mg tid or qid

Brief Summary
Professional Use Information

CARDIZEM®
(diltiazem HCl)
30 mg, 60 mg, 90 mg, and 120 mg Tablets

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** *CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1,243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.*
2. **Congestive Heart Failure.** *Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt).*
   Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** *Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.*
4. **Acute Hepatic Injury.** *In rare instances, significant elevations in enzymes such as alkaline phosphatase, CPK, LDH, SGOT, SGPT, and other symptoms consistent with acute hepatic injury have been noted. These reactions have been reversible upon discontinuation of drug therapy. The relationship to CARDIZEM is uncertain in most cases, but probable in some. (See PRECAUTIONS.)*

PRECAUTIONS

General. *CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.*

Drug Interaction. *Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)*

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. *A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.*

Pregnancy. *Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.*

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. *Diltiazem is excreted in human milk. One report suggests that concentrations in breast milk may approximate serum levels. If use of CARDIZEM is deemed essential, an alternative method of infant feeding should be instituted.*

Pediatric Use. *Safety and effectiveness in children have not been established.*

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences as well as their frequency of presentation are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%). In addition, the following events were reported infrequently (less than 1%):

Cardiovascular:	*Angina, arrhythmia, AV block (first degree), AV block (second or third degree — see conduction warning), bradycardia, congestive heart failure, flushing, hypotension, palpitations, syncope.*
Nervous System:	*Amnesia, gait abnormality, hallucinations, insomnia, nervousness, paresthesia, personality change, somnolence, tinnitus, tremor.*
Gastrointestinal:	*Anorexia, constipation, diarrhea, dysgeusia, dyspepsia, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH (see hepatic warnings), vomiting, weight increase.*
Dermatologic:	*Petechiae, pruritus, photosensitivity, urticaria.*
Other:	*Amblyopia, dyspnea, epistaxis, eye irritation, hyperglycemia, nasal congestion, nocturia, osteoarticular pain, polyuria, sexual difficulties.*

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: alopecia, gingival hyperplasia, erythema multiforme, and leukopenia. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established. *Issued 9/86*

See complete Professional Use Information before prescribing.

References: 1. *Pepine CJ, Feldman RL, Hill JA, et al: Clinical outcome after treatment of rest angina with calcium blockers: Comparative experience during the initial year of therapy with diltiazem, nifedipine, and verapamil. Am Heart J 1983; 106(6):1341-1347.* **2.** *Shapiro W: Calcium channel blockers: Actions on the heart and uses in ischemic heart disease. Consultant 1984;24(Dec):150-159.* **3.** *Johnston DL, Lesoway R, Humen DP, et al: Clinical and hemodynamic evaluation of propranolol in combination with verapamil, nifedipine and diltiazem in exertional angina pectoris: A placebo-controlled, double-blind, randomized, crossover study. Am J Cardiol 1985;55:680-687.* **4.** *Cohn PF, Braunwald E: Chronic ischemic heart disease, in Braunwald E (ed): Heart Disease: A Textbook of Cardiovascular Medicine, ed 2. Philadelphia, WB Saunders Co, 1984, chap 39.* **5.** *Schroeder JS: Calcium and beta blockers in ischemic heart disease: When to use which. Mod Med 1982;50(Sept):94-116.*

Another patient benefit product from
PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC.
KANSAS CITY, MO 64137

1403E7

NEW YORK STATE JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
ELIZA H. CALDWELL, MD
JOSEPH F. MURATORE, MD
JOHN T. PRIOR, MD
GITA S. SINGH*
STANFORD WESSLER, MD

**Medical student*

Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Consulting Editor and Book Review Editor	RICHARD B. BIRRER, MD
Consulting Editor	NAOMI R. BLUESTONE, MD, MPH
Consulting Editor	CARL POCHEDLY, MD
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Managing Editor	CAROL L. MOORE
Advertising Director	ROBERT MILLER
Consulting Medical Writer	VICKI GLASER
Editorial Assistant	MILDRED J. AREMANN
Secretary	ELIZABETH J. SOMERS
Librarian	ELLA ABNEY
Assistant Librarian	ELEANOR BURNS

ASSOCIATE EDITORIAL BOARD 1987

MICHAEL E. BERLOW, MD
RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MYLES S. GOMBERT, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
JAMES M. MORRISSEY, ESQ
STEPHEN NORDLICHT, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. United Media Associates, Inc, 16 Bruce Park Ave, Greenwich, CT 06830. Telephone: (203) 661-9702.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

SAMUEL M. GELFAND, MD,	*President*
JOHN A. FINKBEINER, MD,	*Past-President*
CHARLES D. SHERMAN, JR, MD,	*President-Elect* *Vice-President*
JOHN H. CARTER, MD,	*Secretary*
GEORGE LIM, MD,	*Assistant Secretary*
MORTON KURTZ, MD,	*Treasurer*
ROBERT A. MAYERS, MD,	*Assistant Treasurer*
CHARLES N. ASWAD, MD,	*Speaker*
SEYMOUR R. STALL, MD,	*Vice-Speaker*

Councilors

Term Expires 1988

JAMES H. COSGRIFF, JR, MD,	Erie
RICHARD A. HUGHES, MD,	Warren
ANTONIO F. LASORTE, MD,	Broome
SIDNEY MISHKIN, MD,	Nassau

Term Expires 1989

ROBERT E. FEAR, MD,	Suffolk
STANLEY L. GROSSMAN, MD,	Orange
THOMAS D. PEMRICK, MD,	Rensselaer
RALPH E. SCHLOSSMAN, MD,	Queens

Term Expires 1990

DAVID M. BENFORD, MD,	New York
DUANE M. CADY, MD,	Onondaga
WILLIAM A. DOLAN, MD,	Monroe
ROBERT E. GORDON, MD,	Kings

Resident Councilor (representing the resident physician membership)

KATHLEEN E. SQUIRES, MD,	New York

Student Councilor (representing the medical student membership)

MICHAEL PACIOREK,	Onondaga

Trustees

RICHARD D. EBERLE, MD, (*Chairman*)	Onondaga
EDGAR P. BERRY, MD,	New York
JAMES M. FLANAGAN, MD,	Wayne
ALLISON B. LANDOLT, MD,	Westchester
DANIEL F. O'KEEFFE, MD,	Warren
BERNARD J. PISANI, MD,	New York
VICTOR J. TOFANY, MD,	Monroe

Executive Vice-President	DONALD F. FOY
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Vice-President Emeritus	GEORGE J. LAWRENCE, JR, MD
Director, Division of Policy Coordination	IRMA A. ERICKSON
Director, Division of Scientific Publications	PASCAL JAMES IMPERATO, MD
Director, Division of Communications	EDWARD A. HYNES
Director, Division of Computer Services	IVAN H. NAJMAN
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Governmental Affairs	GERARD L. CONWAY, ESQ
General Counsel	DONALD R. MOY, JD
Director, Division of Management and Meeting Services	ROBERT MILLER
Director, Division of Membership Support Services	EUNICE M. SKELLY
Director, Division of Scientific and Educational Activities	PETER B. FARNSWORTH, MD
Director, Division of Socio-Medical Economics	MARIO V. MENGHINI
Medical Director, Committee for Physicians' Health	BARTON A. HARRIS, MD
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York. Copyright © 1987, Medical Society of the State of New York. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.

There's never been a better time for her...

and PREMARIN®

Proven benefits beyond relief of vasomotor symptoms

No other estrogen proven effective for osteoporosis

Only conjugated estrogens tablets have established efficacy in both osteoporosis[1] and vasomotor symptoms* at 0.625 mg/day. No other estrogen, oral or transdermal, has established clinical evidence or minimum effective dose in both indications.

No estrogen proven safer

PREMARIN is the most extensively tested estrogen, with an unsurpassed record of long-term safety.
And clinical evidence shows a significantly reduced risk of endometrial hyperplasia when cycled with a progestin.[2]

PREMARIN®
(conjugated estrogens tablets)

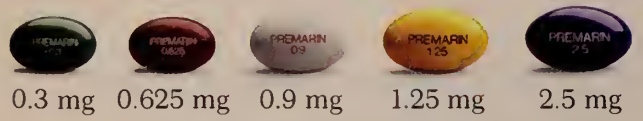
Most trusted for more reasons

*PREMARIN is indicated for moderate-to-severe vasomotor symptoms.

Please see following page for brief summary of prescribing information.

For moderate-to-severe vasomotor symptoms and for osteoporosis

PREMARIN®
(conjugated estrogens tablets)



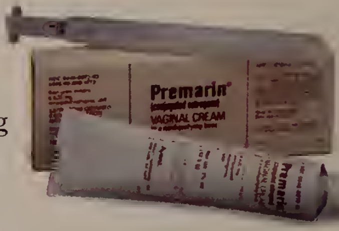
The appearance of these tablets is a trademark of Ayerst Laboratories.

For atrophic vaginitis

PREMARIN®
(conjugated estrogens)

Vaginal Cream

0.625 mg/g



BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION AND PATIENT INFORMATION, SEE PACKAGE CIRCULARS.)*

PREMARIN® Brand of conjugated estrogens tablets, USP
PREMARIN® Brand of conjugated estrogens Vaginal Cream, in a nonliquefying base

> 1. ESTROGENS HAVE BEEN REPORTED TO INCREASE THE RISK OF ENDOMETRIAL CARCINOMA.
> Three independent, case-controlled studies have reported an increased risk of endometrial cancer in postmenopausal women exposed to exogenous estrogens for more than one year This risk was independent of the other known risk factors for endometrial cancer These studies are further supported by the finding that incidence rates of endometrial cancer have increased sharply since 1969 in eight different areas of the United States with population-based cancer reporting systems, an increase which may be related to the rapidly expanding use of estrogens during the last decade. The three case-controlled studies reported that the risk of endometrial cancer in estrogen users was about 4 5 to 13 9 times greater than in nonusers. The risk appears to depend on both duration of treatment and on estrogen dose. In view of these findings, when estrogens are used for the treatment of menopausal symptoms, the lowest dose that will control symptoms should be utilized and medication should be discontinued as soon as possible When prolonged treatment is medically indicated, the patient should be reassessed on at least a semi-annual basis to determine the need for continued therapy Although the evidence must be considered preliminary, one study suggests that cyclic administration of low doses of estrogen may carry less risk than continuous administration; it therefore appears prudent to utilize such a regimen. Close clinical surveillance of all women taking estrogens is important In all cases of undiagnosed persistent or recurring abnormal vaginal bleeding, adequate diagnostic measures should be undertaken to rule out malignancy There is no evidence at present that "natural" estrogens are more or less hazardous than "synthetic" estrogens at equi-estrogenic doses
> 2 ESTROGENS SHOULD NOT BE USED DURING PREGNANCY
> The use of female sex hormones, both estrogens and progestogens, during early pregnancy may seriously damage the offspring It has been shown that females exposed in utero to diethylstilbestrol, a nonsteroidal estrogen, have an increased risk of developing, in later life, a form of vaginal or cervical cancer that is ordinarily extremely rare This risk has been estimated as not greater than 4 per 1,000 exposures. Furthermore, a high percentage of such exposed women (from 30% to 90%) have been found to have vaginal adenosis, epithelial changes of the vagina and cervix Although these changes are histologically benign, it is not known whether they are precursors of malignancy Although similar data are not available with the use of other estrogens, it cannot be presumed they would not induce similar changes Several reports suggest an association between intrauterine exposure to female sex hormones and congenital anomalies, including congenital heart defects and limb-reduction defects. One case-controlled study estimated a 4 7-fold increased risk of limb-reduction defects in infants exposed in utero to sex hormones (oral contraceptives, hormone withdrawal tests for pregnancy, or attempted treatment for threatened abortion). Some of these exposures were very short and involved only a few days of treatment The data suggest that the risk of limb-reduction defects in exposed fetuses is somewhat less than 1 per 1,000. In the past, female sex hormones have been used during pregnancy in an attempt to treat threatened or habitual abortion There is considerable evidence that estrogens are ineffective for these indications, and there is no evidence from well-controlled studies that progestogens are effective for these uses. If PREMARIN is used during pregnancy, or if the patient becomes pregnant while taking this drug, she should be apprised of the potential risks to the fetus, and the advisability of pregnancy continuation.

DESCRIPTION: PREMARIN (conjugated estrogens, USP) contains a mixture of estrogens, obtained exclusively from natural sources, blended to represent the average composition of material derived from pregnant mares' urine. It contains estrone, equilin, and 17α-dihydroequilin, together with smaller amounts of 17α-estradiol, equilenin, and 17α-dihydroequilenin as salts of their sulfate esters Tablets are available in 0 3 mg, 0 625 mg, 0 9 mg, 1 25 mg, and 2 5 mg strengths of conjugated estrogens. Cream is available as 0 625 mg conjugated estrogens per gram

INDICATIONS AND USAGE: PREMARIN (conjugated estrogens tablets, USP): Moderate-to-severe *vasomotor* symptoms associated with the menopause. (There is no evidence that estrogens are effective for nervous symptoms or depression without associated vasomotor symptoms and they should not be used to treat such conditions) Osteoporosis (abnormally low bone mass). Atrophic vaginitis. Kraurosis vulvae. Female castration

PREMARIN (conjugated estrogens) Vaginal Cream is indicated in the treatment of atrophic vaginitis and kraurosis vulvae

PREMARIN HAS NOT BEEN SHOWN TO BE EFFECTIVE FOR ANY PURPOSE DURING PREGNANCY AND ITS USE MAY CAUSE SEVERE HARM TO THE FETUS (SEE BOXED WARNING)

Concomitant Progestin Use: The lowest effective dose appropriate for the specific indication should be utilized Studies of the addition of a progestin for 7 or more days of a cycle of estrogen administration have reported a lowered incidence of endometrial hyperplasia Morphological and biochemical studies of the endometrium suggest that 10 to 13 days of progestin are needed to provide maximal maturation of the endometrium and to eliminate any hyperplastic changes Whether this will provide protection from endometrial carcinoma has not been clearly established. There are possible additional risks which may be associated with the inclusion of progestin in estrogen replacement regimens (See PRECAUTIONS.) The choice of progestin and dosage may be important; product labeling should be reviewed to minimize possible adverse effects

CONTRAINDICATIONS: Estrogens should not be used in women (or men) with any of the following conditions. 1 Known or suspected cancer of the breast except in appropriately selected patients being treated for metastatic disease 2 Known or suspected estrogen-dependent neoplasia 3 Known or suspected pregnancy (see Boxed Warning) 4 Undiagnosed abnormal genital bleeding 5. Active thrombophlebitis or thromboembolic disorders. 6. A past history of thrombophlebitis, thrombosis, or thromboembolic disorders associated with previous estrogen use (except when used in treatment of breast or prostatic malignancy).

WARNINGS: Estrogens have been reported to increase the risk of endometrial carcinoma (see Boxed Warning). However, a recent large, case-controlled study indicated no increase in risk of breast cancer in postmenopausal women. A recent study has reported a 2- to 3-fold increase in the risk of surgically confirmed gallbladder disease in women receiving postmenopausal estrogens.

Adverse effects of oral contraceptives may be expected at the larger doses of estrogen used to treat prostatic or breast cancer or postpartum breast engorgement; it has been shown that there is an increased risk of thrombosis in men receiving estrogens for prostatic cancer and women for postpartum breast engorgement Users of oral contraceptives have an increased risk of diseases, such as thrombophlebitis, pulmonary embolism, stroke, and myocardial infarction Cases of retinal thrombosis, mesenteric thrombosis, and optic neuritis have been reported in oral contraceptive users. An increased risk of postsurgery thromboembolic complications has also been reported in users of oral contraceptives If feasible, estrogen should be discontinued at least 4 weeks before surgery of the type associated with an increased risk of thromboembolism, or during periods of prolonged immobilization Estrogens should not be used in persons with active thrombophlebitis, thromboembolic disorders, or in persons with a history of such disorders in association with estrogen use They should be used with caution in patients with cerebral vascular or coronary artery disease. Large doses (5 mg conjugated estrogens per day), comparable to those used to treat cancer of the prostate and breast, have been shown to increase the risk of nonfatal myocardial infarction, pulmonary embolism, and thrombophlebitis When doses of this size are used, any of the thromboembolic and thrombotic adverse effects should be considered a clear risk

Benign hepatic adenomas should be considered in estrogen users having abdominal pain and tenderness, abdominal mass, or hypovolemic shock. Hepatocellular carcinoma has been reported in women taking estrogen-containing oral contraceptives. Increased blood pressure may occur with use of estrogens in the menopause and blood pressure should be monitored with estrogen use A worsening of glucose tolerance has been observed in patients on estrogen-containing oral contraceptives For this reason, diabetic patients should be carefully observed Estrogens may lead to severe hypercalcemia in patients with breast cancer and bone metastases

PRECAUTIONS: Physical examination and a complete medical and family history should be taken prior to the initiation of any estrogen therapy with special reference to blood pressure, breasts, abdomen, and pelvic organs, and should include a Papanicolaou smear. As a general rule, estrogen should not be prescribed for longer than one year without another physical examination being performed. Conditions influenced by fluid retention, such as asthma, epilepsy, migraine, and cardiac or renal dysfunction, require careful observation. Certain patients may develop manifestations of excessive estrogenic stimulation, such as abnormal or excessive uterine bleeding, mastodynia, etc Prolonged administration of unopposed estrogen therapy has been reported to increase the risk of endometrial hyperplasia in some patients Oral contraceptives appear to be associated with an increased incidence of mental depression Patients with a history of depression should be carefully observed Pre-existing uterine leiomyomata may increase in size during estrogen use The pathologist should be advised of estrogen therapy when relevant specimens are submitted If jaundice develops in any patient receiving estrogen, the medication should be discontinued while the cause is investigated. Estrogens should be used with care in patients with impaired liver function, renal insufficiency, metabolic bone diseases associated with hypercalcemia or in young patients in whom bone growth is not yet complete. If concomitant progestin therapy is used, potential risks may include adverse effects on carbohydrate and lipid metabolism.

The following changes may be expected with larger doses of estrogen:

a Increased sulfobromophthalein retention.

b. Increased prothrombin and factors VII, VIII, IX, and X, decreased antithrombin 3, increased norepinephrine-induced platelet aggregability

c Increased thyroid binding globulin (TBG) leading to increased circulating total thyroid hormone, as measured by PBI, T_4 by column, or T_4 by radioimmunoassay. Free T_3 resin uptake is decreased, reflecting the elevated TBG, free T_4 concentration is unaltered.

d. Impaired glucose tolerance.

e. Decreased pregnanediol excretion.

f. Reduced response to metyrapone test

g Reduced serum folate concentration.

h Increased serum triglyceride and phospholipid concentration

As a general principle, the administration of any drug to nursing mothers should be done only when clearly necessary since many drugs are excreted in human milk

Long-term, continuous administration of natural and synthetic estrogens in certain animal species increases the frequency of carcinomas of the breast, cervix, vagina, and liver. However, in a recent, large case-controlled study of postmenopausal women there was no increase in risk of breast cancer with use of conjugated estrogens

ADVERSE REACTIONS: The following have been reported with estrogenic therapy, including oral contraceptives: breakthrough bleeding, spotting, change in menstrual flow, dysmenorrhea, premenstrual-like syndrome; amenorrhea during and after treatment, increase in size of uterine fibromyomata; vaginal candidiasis, change in cervical erosion and in degree of cervical secretion, cystitis-like syndrome, tenderness, enlargement, secretion (of breasts); nausea, vomiting, abdominal cramps, bloating, cholestatic jaundice; chloasma or melasma which may persist when drug is discontinued, erythema multiforme, erythema nodosum; hemorrhagic eruption, loss of scalp hair; hirsutism; steepening of corneal curvature; intolerance to contact lenses; headache, migraine, dizziness, mental depression, chorea, increase or decrease in weight; reduced carbohydrate tolerance, aggravation of porphyria; edema; changes in libido.

ACUTE OVERDOSAGE: May cause nausea, and withdrawal bleeding may occur in females

DOSAGE AND ADMINISTRATION:

PREMARIN® Brand of conjugated estrogens tablets, USP

1 *Given cyclically for short-term use only.* For treatment of moderate-to-severe *vasomotor* symptoms, atrophic vaginitis, or kraurosis vulvae associated with the menopause (0 3 mg to 1 25 mg or more daily). The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible Administration should be cyclic (eg, three weeks on and one week off). Attempts to discontinue or taper medication should be made at three- to six-month intervals

2. *Given cyclically* Osteoporosis. Female castration. Osteoporosis —0 625 mg daily Administration should be cyclic (eg, three weeks on and one week off). Female castration—1.25 mg daily, cyclically. Adjust upward or downward according to response of the patient For maintenance, adjust dosage to lowest level that will provide effective control

Patients with an intact uterus should be monitored for signs of endometrial cancer and appropriate measures taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding

PREMARIN® Brand of conjugated estrogens Vaginal Cream

Given cyclically for short-term use only For treatment of atrophic vaginitis or kraurosis vulvae

The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible.

Administration should be cyclic (eg, three weeks on and one week off)

Attempts to discontinue or taper medication should be made at three- to six-month intervals

Usual dosage range: 2 g to 4 g daily, intravaginally, depending on the severity of the condition

Treated patients with an intact uterus should be monitored closely for signs of endometrial cancer and appropriate diagnostic measures should be taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding

References:

1. Lindsay R, Hart DM, Clark DM. The minimum effective dose of estrogen for prevention of postmenopausal bone loss. *Obstet Gynecol* 1984,63 759-763 **2.** Studd JWW, Thom MH, Paterson MEL, et al: The prevention and treatment of endometrial pathology in postmenopausal women receiving exogenous estrogens, in Pasetto N, Paoletti R, Ambrus JL (eds): *The Menopause and Postmenopause*. Lancaster, England, MTP Press Ltd, 1980, chap 13

© 1987 Ayerst Laboratories
All rights reserved

6437/587

Ayerst®

AYERST LABORATORIES
New York, NY 10017

HIV Positive Confirmed. Then What?

AIDS is tough to treat. Ruthless to an increasing patient population, it challenges physicians and nurses as never before in their commitment to quality care.

The Spellman Center for HIV-Related Disease at St. Clare's Hospital and Health Center in New York City, accepts the challenge posed by AIDS and HIV infection. The first facility in the tri-state region to devote its resources solely to the care of AIDS patients, the Spellman Center designates its highest priority as compassionate patient care. A dedicated staff provides comprehensive medical, nursing, psychosocial, and dental treatment in an atmosphere that is warm and understanding.

The Spellman Center—a 60-bed Acute Care Unit, Emergency Service, Outpatient, Ambulatory Infusion and Dental Clinics—accepts referrals of patients with CDC-confirmed AIDS or ARC (AIDS-related complex), and those who are seropositive for HIV. Spellman also reaches out to offer testing, counseling and treatment to persons at risk for AIDS.

If you need consultation concerning your patients, or more information, please call Dr. Debra Spicehandler, the Medical Director, at (212) 459-8409.

The Spellman Center also maintains an information hotline for patients with questions about AIDS: 1-800-433-AIDS.

The Spellman Center
for HIV-Related Disease
A Designated AIDS Treatment Facility
St. Clare's Hospital and Health Center

415 W 51st St
New York, NY 10019

An Affiliate of New York Medical College

EMERGENCY! MEDICAL CLAIMS OVERLOAD!

The EMC*EXPRESS™ *System*

TO THE RESCUE!

If claims processing paperwork can get your office all backed up, you need to call in GE Information Services' EMC*EXPRESS rescue squad.

The **EMC*EXPRESS System** is a computerized medical claims system which enables you to deliver medical claims to participating carriers within hours of treating a patient. One phone call is all it takes to have your computer speed all the information needed to process your claims to **multiple** carriers, including Medicare, commercial and private.

Working with the software already in your office, the **EMC*EXPRESS** System helps you:

- Reduce your administrative costs for processing claims
- Reduce the number of claims rejected because of incorrect or incomplete information
- Speed up your claims payment—**and that can put new life into your cash flow.**

The EMC*EXPRESS System and Your Office

The EMC*EXPRESS System accommodates a broad range of practice sizes and a wide variety of office computer systems, from easy-to-use claims entry systems to the most sophisticated practice management systems.

So whatever the size of your office, whatever the magnitude of your paperwork problem, we recommend the **EMC*EXPRESS** System as your treatment of choice.

For more than 20 years, GE Information Services has been helping companies around the world do business better. And now GE has teamed up with authorized system vendors to bring rapid, accurate, and cost-effective electronic medical claims communications to the healthcare field.

To have the EMC*EXPRESS rescue squad come to your aid, call an authorized EMC*EXPRESS system vendor or contact us directly at 1-800-433-3683, extension 7321. We'll send help your way before you can say, "Emergency!"

GE INFORMATION SERVICES
USA

The EMC*EXPRESS™ System

PARTICIPATING CARRIERS

Empire Blue Cross and Blue Shield
Medicare B and Private Business Claims
Metropolitan Life Insurance Co.
(Includes Empire NY State Employees)

NEIC ACTIVE PARTICIPANTS

Aetna Life & Casualty
Allstate Life Insurance Co.
Benefit Trust Life
CNA Insurance Companies
Confederation Administration Life Insurance Co.
Confederation Life Insurance Co.
Connecticut Life Insurance Co.
Connecticut General Life Insurance Co. (CIGNA)
Equitable Life Assurance Society of the U.S.
General American Life Insurance Co.
The Great-West Life Assurance Co. of America
Gulf Group Services
The Hartford Insurance Group
John Hancock Mutual Life Insurance Co.
Liberty Life Assurance Co.
Lincoln National Life Insurance Co.
Massachusetts Mutual Life Insurance Co.
Metropolitan Life Insurance Co.
Mutual of Omaha Insurance Co.
New England Mutual Life Insurance Co.
New York Life Insurance Co.
Pacific Mutual Life Insurance Co.
Philadelphia American Life Insurance Co.
Philadelphia Life Insurance Co.
Phoenix Mutual Life Insurance Co.
Pilot Life Insurance Co.
Principal Mutual Financial Group
Provident Life and Accident Insurance Co.
Prudential Insurance Co.
State Mutual Insurance Co.
The Travelers Insurance Co. (includes Railroad Retirement)
Unionmutual Stock Life Insurance Co.

AUTHORIZED SYSTEM VENDORS

Annson Systems	312 564-8310
Articulate Publications, Inc.	800 872-2282
Artificial Intelligence, Inc.	206 271-8633
CALYX	800 558-2208
Colwell Systems, Inc.	800 637-1140
Coopervision	800 772-CVIS
EDP Systems, Inc.	214 881-8454
Effective Solutions and Services	312 635-9911
Health America Systems	312 362-3730
LDS, Inc.	913 648-7111
MOS, Inc.	800 323-6671
Physicians' Micro Systems, Inc.	206 441-8490
Physicians' Office Computer	213 603-0555
Prism Data Systems	800 428-2310
ProSource Systems	800 645-5609
Santiago Data Systems, Inc.	800 652-3500
Unitec, Inc.	800 237-3762
UNIVAIR, Inc.	314 426-1099
Westland Software House, Inc.	818 992-0081

NEW VENDORS AND CARRIERS ADDED REGULARLY. FOR MOST RECENT LISTINGS, CALL 1-800-433-3683.

GE INFORMATION SERVICES
USA

401 N. WASHINGTON ST., ROCKVILLE, MD 20850

NYSJM987

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

OCTOBER 1987

AROUND THE STATE

MANHATTAN

Oct 5–9. **Five Day Emergency Medicine Board Review Course.** 36½ Cat 1 Credits. NYU Medical Center. *Oct 10.* **Imaging Update for Clinicians: Gastrointestinal Radiology.** 6 Cat 1 Credits. NYU Medical Center. *Oct 10–12.* **Cardiopulmonary and Critical Care Medicine: A Specialty Review Course.** 22½ Cat 1 Credits. NYU Medical Center. *Oct 17.* **Imaging Update for Clinicians: Thoracic Radiology.** 6 Cat 1 Credits. NYU Medical Center. *Oct 22–24.* **Comprehensive Seminar on Fine Needle Aspiration: A Review and Update.** 19¾ Cat 1 Credits. NYU Medical Center. Contact: NYU Medical Center Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295/24-hr service.

Oct 8–9. **Tenth Annual Current Concerns in Adolescent Medicine.** 12 Cat 1 Credits. New York Hilton at Rockefeller Center. Contact: Ann J. Boehme, Associate Director for Continuing Education, Schneider Children's Hospital at Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

Oct 9. **AIDS Update—The Dentist and the Dental Patient.** Beth Israel Medical Center. *Oct 11.* **Difficult Behavior in Young Children: The Influence of Temperament.** Beth Israel Medical Center. *Oct 16.* **Effective Clinical Laboratory Utilization in 1987.** Beth Israel Medical Center. *Oct 23.* **Cosmetology Symposium: New Therapy for the Young Skin.** Beth Israel Medical Center. Contact: Beverley R. Baptiste, Medical Education Office, Beth Israel Medical Center, First Ave at 16th Street, New York, NY 10003. Tel: (212) 420-2849.

Oct 9–12. **Psychoanalytic Practice Today: Similarities and Differences.** New York City. Contact: Ann R. Turkel, MD, Secretary-General of the IFPS, 350 Central Park West, New York, NY 10025. Tel: (212) 831-3400.

Oct 16–17. **Functional Endoscopic Sinus Surgery.** 14 Cat 1 Credits. Manhattan Eye, Ear and Throat Hospital. *Oct 30–31.* **Laser Surgery Symposium.** 15 Cat 1 Credits. Manhattan Eye, Ear and Throat Hospital. Contact: Kristen M. Johnson, Administrator, Department of Otolaryngology, 210 East 64th St, New York, NY 10021. Tel: (212) 838-9200, ext 2448.

NASSAU

Oct 8–10. **First International Conference on Drug Research in Immunologic and Infectious Diseases. Antifungal Drugs: Synthesis, Preclinical and Clinical Evaluation.** Garden City Hotel, Garden City. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd Street, New York, NY 10021.

Oct 14. **Ophthalmology for the Pediatrician and General Practice Physician.** 6½ Cat 1 Credits. North Shore University Hospital, Manhasset. Contact: Mary B. Strong, MA, Department of Health Education/Community Affairs, North Shore University Hospital, 300 Community Dr, Manhasset, NY 11030. Tel: (516) 562-3045.

AROUND THE NATION

CALIFORNIA

Oct 10–11. **The Skin from A to Z.** 10 Cat 1 Credits. University of California San Francisco. Contact: Extended Programs in Medical Education, University of California School of Medicine, Room 569-U, San Francisco, CA 94143. Tel: (415) 476-4251.

Oct 18–21. **Preparing for Surgery: Medical Evaluation of the Preoperative Patient.** LaPlaya Hotel, Carmel. *Oct 23–26.* **Present Concepts in Internal Medicine/4th Annual ACP Army Regional Meeting.** Holiday Inn Golden Gateway, San Francisco. Contact: American College of Physicians, Registration Services Div, PO Box 7777-R-0510, Philadelphia, PA 19175.

Oct 19–23. **12th Annual San Diego**

(*continued on p 18A*)

Added Control For America's Coughs

The expectorant action of guaifenesin plus the Advantage of Codeine!

Robitussin and Codeine. Each 5 ml (*1 teaspoonful*) contains: Guaifenesin, USP, 100 mg; Codeine Phosphate, USP, 10 mg (*warning: may be habit forming*); alcohol, 3.5 percent; in a palatable, aromatic syrup

A·H·ROBINS

Pharmaceutical Division Richmond, Virginia 23261-6609

©A.H. Robins Company 1985

Robitussin A-C®

Classified Advertising

PHYSICIANS WANTED

EMERGENCY MEDICINE POSITIONS—Full/part time emergency medicine physicians sought by multi state professional association for openings in metropolitan NY, PA, MD, DC, FL, New England and throughout U.S. Contact or send CV to Liberty Healthcare Corporation, 399 Market Street, Suite 400, Philadelphia, PA 19106, (215) 592-7400 or outside Pa (800) 331-7122.

ARIZONA-BASED PHYSICIAN RECRUITMENT firm has quality opportunities coast to coast. Available positions in most primary care and surgical specialties to include OB/GYN, Orthopedics, ER, and ENT. "Quality Physicians for Quality Clients since 1972." Call 602-990-8080; or send CV to: Mitchell & Associates, Inc., PO Box 1804, Scottsdale, AZ 85252.

NEW YORK, BUFFALO—Seeking full-time and part-time physicians residency trained in emergency medicine or primary specialty for 32,000 annual volume emergency department. Directorship available. Attractive compensation, malpractice insurance & benefit package. Contact: Emergency Consultants, Inc., 2240 S. Airport Rd., Room 42, Traverse City, MI 49684; 1-800-253-1795 or in Michigan 1-800-632-3496.

PHYSICIAN INTERNATIONAL "The Physicians Source"—Our staff of over 40 highly trained, experienced professionals provide our clients with comprehensive, cost-effective services including Physician Search, Locum Tenens, Practice Appraisal and Brokerage, Practice Crisis Assurance Programs, and Medical Management Consulting. Please call us for a free, confidential assessment. Physician International, Four-NYS Vermont Street, Buffalo, NY 14213-2498. (716) 884-3700. Physician International is an Approved Membership Benefit Program of MSSNY.

POSITIONS WANTED

UNIVERSITY-TRAINED, BOARD CERTIFIED INTERNAL MEDICINE, Board-certified Infectious Diseases seeks to return to New York City area. Desire private or group practice or hospital affiliation. Experienced in all aspects of subspecialty; willing to practice Internal Medicine. Available 1988. Reply Dept. 448 c/o NYSJM.

PRACTICES AVAILABLE

FOR SALE: FAMILY PRACTICE. Retiring, immediate occupancy. Professional Building, long standing active practice. In excellent community on main thoroughfare in Nassau County, 200 feet across Queens border. Ideal for new ambitious M.D. or relocation. 1200 square feet modern rooms, fully equipped with x-ray. Low overhead. 10 minutes from 4 major hospitals. Reply Dept. 447 c/o NYSJM.

EQUIPMENT

50% OFF PREVIOUSLY OWNED MEDICAL—Laboratory, x-ray, ultrasound equipment. Buy, Sell, Broker, Repair. Appraisals by certified surgical consultants. Medical Equipment Resale & Repair, 24026 Haggerty Road, Farmington Hills, MI 48018. 1-800-247-5826. (313) 477-6880.

DISCOUNT HOLTZER SCANNING SERVICES starting at $35.00. Spacelabs holter recorders available from $1,275.00. Smallest and lightest holters update. Fast service (24–48 hours) turnover. Hook-up kits starting at $4.95. Special introductory offer of three free tests at the purchase or lease of the holter. Stress test electrodes available at 34 cents. Scanning paper available at $18.95. Cardiologist over-read available at $15.00. If interested call 1-800-248-0153.

ELECTROCARDIOGRAPH (EKG) Hewlett-Packard, 10-11 years old, perfect $399. Phone (516) 789-2891

REAL ESTATE FOR SALE OR RENT

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

MANHATTAN, 220 EAST 69TH STREET. Share expenses. Luxury building. Own 12 × 15 consultation room, garden window; or share two days. Share 3 equipped examination rooms. Laboratory, x-ray services on premises. Suitable for Internist, Surgeon or Psychiatrist. (212) 737-4020.

FOR RENT completely equipped office in upstate New York. Office has large waiting, consultation, treatment, x-ray, lab, physiotherapy, and 2 bathrooms. Utilities included. Attached garage. Only physician here for 54 years. Contact: Francis D. Brown, M.D., Maple-Bank, Hobart, NY 13788.

DUPLEX OFFICE SUITE for rent in Manhattan. Beautiful seven office duplex suite: first and second floor, 2100 sq. ft., two full, two half baths, private secure street entrance, waiting room, kitchen, carpeting, air conditioning. 96th Street near Columbus, long lease available. (212) 678-6000.

PORT WASHINGTON—office space varying in size available in professional office building. One block from LIRR. Call owner at (516) 625-1500.

ESTATE LIKE GARDENS. Corner, 1/2 acre plus, custom designed artist home. 3 bedrooms, 3 baths, a 800 sq. ft. master suite and loft, skylights, cathedral beamed ceiling plus 10ft. hardwood floors. 30 ft. L.R., D.R., Fireplace, French doors onto deck and patio; ground floor suite suitable for Doctor's office. Excellent location, convenient to shopping and parkways, School District #13. $360,000 Principals Only, (516) 427-4170.

MEDICAL OFFICES

NEW YORK CITY:

2nd Ave & 73 St—1350 Sq Ft.
Ground floor in lux apt bldg.
Street & lobby entrances.

HEWLETT, L.I.:

2100 Sq Ft. Top Floor in
well located professional
bldg in heart of Hewlett.

KOEPPEL & KOEPPEL
30 E. 42 St., NY 10017 • 212-687-8980

REAL ESTATE FOR SALE OR RENT—CONT'D

OFFICE SPACE AVAILABLE: Throgs Neck section of Bronx. Modern medical complex, 1500 square feet, part time/full time. Call (212) 409-9494.

EAST SIXTIES, PROFESSIONAL BUILDING. The preferred location for internists, cardiologist, rheumatologist. 7 treatment rooms, 2 reception areas to share with three other internists. Share NYC licensed lab, radiology, thermography. Full or part time. (212) 838-2860.

PRIME MIDTOWN LOCATION. Office space for rent in newly refurbished Madison Medical Building, New York City, Madison Avenue between 32-33rd Streets. 800 usable sq. feet. Long term lease and sublet clause available. Secretarial area, waiting room, two exam rooms. Call Dr. Siegel at (212) 340-6431.

NEW PENTHOUSE MEDICAL OFFICE for lease on Long Island Expressway in Fresh Meadows, Queens, New York. Close proximity to all major highways and transportation. 2,000 sq. ft. available. Will build to suit. Elevators, solarium and on-site parking. Call (718) 463-8800.

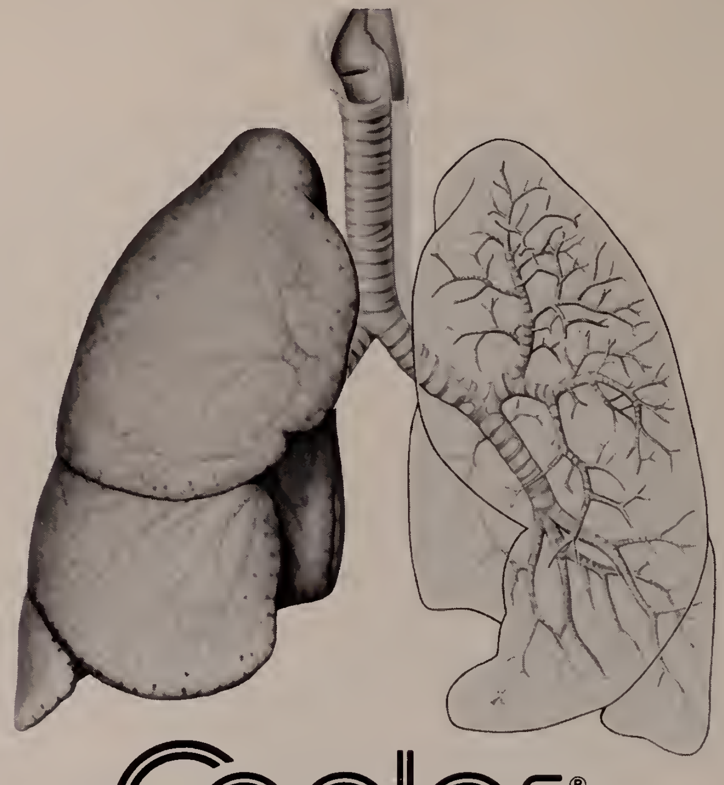
SUITE FULLY EQUIPPED, available for pathologist immediately—Bethpage Medical Building, 4277 Hempstead Tpke., Bethpage, NY 11714. Call Frank at (516) 735-3001.

MISCELLANEOUS

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuarial certification and employee statements of participation—partnership agreements and professionals incorporation—No insurance required—references upon request—Wachstock and Dienstag Attorneys at Law, 122 Cutter Mill Road, Great Neck, NY 11021 (516) 773-3322.

Consider the causative organisms...



Ceclor®
cefaclor

250-mg Pulvules® t.i.d.
offers effectiveness against the major causes of bacterial bronchitis

Haemophilus influenzae, Streptococcus pneumoniae
(ampicillin-susceptible and ampicillin-resistant)

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

Ceclor® (cefaclor)

Summary. Consult the package literature for prescribing information.

Indications: Lower respiratory infections, including pneumonia, caused by susceptible strains of *Streptococcus pneumoniae, Haemophilus influenzae*, and *Streptococcus pyogenes* (group A β-hemolytic streptococci).

Contraindication:
Known allergy to cephalosporins.

Warnings:
CECLOR SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY. POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS.

Administer cautiously to allergic patients.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea. Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.

Precautions:

- Discontinue Ceclor in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of nonsusceptible organisms.
- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- Ceclor should be administered with caution in the presence of markedly impaired renal function. Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk. Exercise caution in prescribing for these patients.

Adverse Reactions: (percentage of patients)

Therapy-related adverse reactions are uncommon. Those reported include:

- Gastrointestinal (mostly diarrhea): 2.5%.
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
- Hypersensitivity reactions (including morbilliform eruptions, pruritus, urticaria, and serum-sickness-like reactions that have included erythema multiforme [rarely, Stevens-Johnson syndrome] or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever): 1.5%; usually subside within a few days after cessation of therapy. Serum-sickness-like reactions have been reported more frequently in children than in adults and have usually occurred during or following a second course of therapy with Ceclor. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.
- Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.
- As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely.
- Rarely, reversible hyperactivity, nervousness, insomnia, confusion, hypertonia, dizziness, and somnolence have been reported.
- Other: eosinophilia, 2%; genital pruritus or vaginitis, less than 1%; and, rarely, thrombocytopenia.

Abnormalities in laboratory results of uncertain etiology

- Slight elevations in hepatic enzymes.
- Transient fluctuations in leukocyte count (especially in infants and children).
- Abnormal urinalysis; elevations in BUN or serum creatinine.
- Positive direct Coombs' test.
- False-positive tests for urinary glucose with Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (glucose enzymatic test strip, Lilly).

[072886R]

PA 8794 AMP

©1987, ELI LILLY AND COMPANY

Lilly

700241

Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285

Eli Lilly Industries, Inc
Carolina, Puerto Rico 00630

MEDICAL SOCIETY OF THE STATE OF NEW YORK ORGANIZED 1807

NEW YORK STATE
JOURNAL OF MEDICINE

September 1987 • Volume 87, Number 9

COMMENTARIES

Fibronectin deficiency following cardiopulmonary bypass

Fibronectin is a glycoprotein of large molecular weight that has both opsonic and adhesive biological functions.[1] The soluble form of fibronectin is found in blood, lymph, and other tissue fluids,[2] while an insoluble form is found in the extracellular matrix of basement membranes, loose connective tissues, and granulation tissue.[3] The paper by Gotta et al[4] in this issue of the *Journal* focuses on the alterations in plasma fibronectin following coronary artery bypass. Comparison to plasma albumin is made in order to evaluate indirectly dilution of the plasma protein pool as a contributing factor to the observed decrease of fibronectin. The authors conclude that while dilution contributes to the hypofibronectinemia, other factors such as opsonic consumption, cold-induced precipitation of fibronectin in association with fibrinogen, and absorption onto foreign surfaces may also contribute to the low fibronectin level. What is it about fibronectin that makes this plasma deficiency potentially important from a clinical standpoint?

Fibronectin is a complex molecule consisting of two similar monomers.[5-7] Its unique domains have a high affinity for collagen, gelatin, fibrin, actin, and heparin. Fibronectin also binds to *Staphylococcus aureus*. Plasma fibronectin is identical to opsonic α_2 surface binding (SB) glycoprotein,[8] which modulates the phagocytic activity of reticuloendothelial cells or the macrophage system with respect to ingestion of nonbacterial particulates.[1] These most likely consist of collagenous debris, fibrin-fibrinogen complexes, connective tissue fragments, intracellular membraneous debris, and immune complexes. Deficiency of plasma fibronectin will limit the phagocytic clearance capacity of the reticuloendothelial system (RES), especially the hepatic Kupffer cells.[1] Such deficiency is observed in patients soon after major surgery, trauma, or burn injury.[2,9] A second period of deficiency is typically observed if such patients become septic in association with multiple organ failure, especially altered lung vascular permeability and adult respiratory distress. Plasma fibronectin deficiency will amplify the increase in lung vascular permeability observed in experimental animals with postoperative sepsis.[10] This association may be mediated by limited reticuloendothelial clearance of blood-borne particulates, which can eventually embolize in the lung and alter endothelial integrity. The ability of plasma fibronectin to serve as a reservoir for fibronectin in tissues[11,12] may also be an important factor in the altered vascular integrity. Acute depletion of plasma fibronectin will limit reticuloendothelial function and decrease resistance to trauma and shock.[1]

Cellular fibronectin is antigenically similar to plasma fibronectin, although differences can be documented with respect to adhesive properties as well as solubility.[5,7] However, both compounds appear to have similar secondary and tertiary polypeptide structures, and both can bind to collagen, a connective tissue matrix protein.[7] Cellular or tissue fibronectin influences cell-cell interaction and cell adhesion to a collagenous substratum. Fibronectin promotes the attachment and spreading of cells in culture as well as cellular differentiation, cell migration, and cell morphology. Fibronectin can be detected under endothelial cells and in liver sinusoids, and sequestered at sites of injury and/or wound repair. It is also found in the provisional matrix of fibrin during the process of wound healing. Endothelial cells, macrophages, fibroblasts, and hepatocytes, as well as other cell types, can synthesize fibronectin in culture, but the hepatocytes in the liver are suspected to be the primary site for the production of the soluble fibronectin found in plasma,[13] which circulates as a 440,000 dalton disulfide-bonded dimer. Patients with fulminant hepatic failure have low plasma fibronectin levels, and the estimated rate of synthesis of fibronectin by the liver[13] is consistent with that required for maintaining the plasma concentration. Plasma fibronectin can be incorporated into the tissue fibronectin pool.[11,12] Its incorporation into the insoluble extracellular matrix, with subsequent formation of fibronectin multimers, may be due to disulfide exchange in the amino terminal domain of fibronectin.[6] The assembly of soluble fibronectin into the matrix may require specific cell receptors, such as have been documented on fibroblasts, which mediate the transfer of soluble fibronectin into the insoluble extracellular matrix fibronectin pool. Thus, in addition to its opsonic role, plasma fibronectin may serve as a reservoir for fibronectin in tissues, where it may influence macrophage clearance of devitalized tissue, local wound repair after injury, as well as structural integrity of vessels and tissue.[1]

Fibronectin monomer consists of at least four globular domains separated by flexible protease-sensitive areas of polypeptide chains.[5,7] The N-terminal domain (25,000-30,000 daltons) binds to fibrin, *S aureus*, and heparin, as well as actin. A second domain (30,000-40,000 daltons) contains the collagen and gelatin binding site. The polypeptide chain between these two domains is sensitive to cleavage by proteases such as thrombin, plasmin, trypsin, and leukocyte elastase. A third domain (140,000-160,000 daltons) contains the fibroblast or cell adhesion site and can also bind heparin. A fourth domain (6,000-10,000 daltons) binds to fibrin and contains the C-terminus, where the two fibronectin monomers are linked by interchain disulfide bonds. Proteases can also cleave this portion of fibronectin. Thus, fibronectin is an adhesive modular glycoprotein[5] with an affinity for many entities; it can be endocytosed by phagocytic cells.[1,8] As such, it may be an important molecule in the blood and tissue fluids required for macrophage clearance of actin-containing cytoskeletal debris and extracellular collagenous debris after injury.

Pulmonary abnormalities and adult respiratory distress are common manifestations of organ failure in patients with sepsis due to surgery or injury. Burn patients with mainly cutaneous injury can also manifest respiratory distress, which may be the result of ventilation-perfusion imbalance and interstitial pulmonary edema. Capillary leaking in the lung is believed to be a major contributing factor to pulmonary edema and post-traumatic pulmonary insufficiency.[1,10] This altered integrity of the vascular bed is accentuated during periods of sepsis and intravascular coagulation after surgery or trauma.[10,14] It is suspected that sequestration of activated phagocytic cells in the vascular and interstitial spaces of the lung, and their release of proteases as well as oxygen metabolites, contribute to this acute lung injury. These events may initiate the fragmentation and/or release of tissue fibronectin found between lung endothelial cells and their basement membranes as well as between lung epithelial cells and their basement membranes. We have observed that plasma fibronectin deficiency in sheep amplifies the increase in lung vascular permeability with sepsis following surgery.[10] Furthermore, infusion of purified plasma fibronectin to maintain a sustained elevated level of circulating fibronectin can limit the increase in capillary leaking observed.[14] The plasma pool is a reservoir for fibronectin in tissues, and its concentration may be critical to vascular integrity.

Our laboratory documented[2] the presence of opsonic glycoprotein or plasma fibronectin deficiency soon after patients experienced surgery, trauma, or injury as well as in association with sepsis.[9] Subsequent reports from other laboratories have confirmed these two phases of plasma fibronectin deficiency. Malnutrition can prolong the hypofibronectinemia. Patients with postsurgical intra-abdominal infections have lower preoperative plasma fibronectin levels than patients without sepsis, and the inability to normalize plasma fibronectin levels after surgery has been correlated with an increased incidence of organ failure.[15] Plasma fibronectin, also known as cold-insoluble globulin, is concentrated in plasma cryoprecipitate, which can be utilized to reverse the immunoreactive fibronectin and bioassayable opsonic deficiency in such patients.[2,9] Cryoprecipitate that has been stored for less than two months is most effective, since prolonged storage (4-12 months) results in a progressive loss of fibronectin's opsonic activity, even though fibronectin antigen concentration is not reduced. Recently, a purified and pasteurized preparation of human fibronectin used in clinical studies has selectively reversed the plasma fibronectin deficiency,[16] but phase III clinical trials—aimed at determining the preparation's therapeutic value and dosage protocol—have yet to be completed. In either case, the potential exists, in the near future, to passively provide purified human plasma fibronectin.

In the report by Gotta et al,[4] the authors clearly demonstrate the profound, acute depletion of plasma fibronectin in association with coronary artery bypass surgery. A similar depletion has been previously documented by Gandhi et al[17] and by Snyder et al[18] in patients undergoing cardiopulmonary bypass. We have also observed a similar acute depletion (Saba, Foster and Phelan, unpublished data, 1987), although the restoration of normal levels in that patient study (n = 9) was more prolonged and typically took several days. Impaired reticuloendothelial function and/or limited opsonic activity have been noted experimentally after extracorporeal circulation,[19,20] but whether fibronectin deficiency is the basis for such macrophage dysfunction remains to be documented in patients. According to Gotta et al[4] as well as others,[17,18] the hypofibronectinemia that occurs in patients who undergo coronary artery bypass cannot be totally explained by hemodilution, since the decline in fibronectin is significantly greater than that detected for albumin, IgG, or C3. Thus, other factors must contribute to the acute plasma fibronectin deficiency.

Plasma contains a protein of large molecular weight that can readily precipitate in the cold.[21] This protein is often referred to as cold-insoluble globulin (CIg), and its cryoprecipitability in conjunction with fibrinogen is enhanced by heparin.[21] Cold-insoluble globulin is identical to plasma fibronectin or opsonic glycoprotein.[1,8] In patients undergoing cardiopulmonary bypass, there is a sufficient amount of heparin and a temperature of 20-22°C or less, both of which are in the range[21] needed to enhance complex formation. Thus, one factor likely to contribute to the deficiency of immunoassayable plasma fibronectin is the cold-induced precipitation of fibronectin with plasma fibrinogen, which is amplified by heparin. Trauma due to surgery and other forms of tissue injury can release gelatin-like or collagen-like debris as well as actin-containing cytoskeletal debris into the circulation.[22] Fibronectin has a high affinity for both collagen and actin, and they can bind fibronectin in the plasma and limit its detection by immunoassay[22] and its opsonic activity. As pointed out by Gotta et al,[4] fibronectin also interacts with foreign surfaces and has been implicated in the thrombogenicity of certain biomaterials. Fibronectin may influence the deposition of fibrin as well as platelets onto foreign surfaces or exposed areas of subendothelium at sites of vascular injury. While absorption of fibronectin onto the oxygenator membrane could have been a contributing factor, it was apparently not a major aspect of the depletion observed in these patients. However, the blood was exposed to other foreign surfaces—ie, pump tubing—and the nonspecific adsorption of this adhesive molecule may additionally deplete fibronectin, although the magnitude of loss by this mechanism remains to be quantified.

As to the restoration of fibronectin levels following the bypass, several factors were considered: release of preformed fibronectin from a storage pool; increased synthesis of fibronectin; solubilization of cold-induced fibronectin complex; and release of fibronectin from the insoluble tissue pool. The data contained in the paper by Gotta et al[4] do not allow for conclusions in this area. Indeed, previous studies on fibronectin levels in patients undergoing cardiopulmonary bypass[17,18] also do not address the mechanism of restoration. Solubilization of precipitated fibronectin that had formed a complex with fibrinogen in the cold may occur after temperature normalization, but this is only speculation. Release of fibronectin from the tissue pool cannot be concluded unless one uses monoclonal antibodies specific for human tissue as opposed to human plasma fibronectin, assuming the tissue pool is antigenically distinct. Increased synthesis would have been a likely factor if the patients had required a longer period for normalization of their fibronectin levels, but its contribution during a six-hour recovery interval can only be speculated. The release of a preformed, intracellular storage pool of soluble, dimeric fibronectin is, in theory, a potential factor, but the existence of such a pool in humans has yet to be documented. Since the amount of soluble fibronectin in the extravascular, interstitial fluid compartment can be significant,[11,14] it may be a source for soluble fibronectin returning to the plasma via the lymphatic system.

The plasma fibronectin level in adults is typically 350 μg/mL ±30 μg/mL. The fibronectin concentration in serum is less than that observed in plasma, due to its binding to the alpha chain of fibrin during clot formation. The fibronectin concentration in lymph is typically 30–40% of the plasma level.[14] Its plasma level in infants is less than that observed in adults. Whether this reflects increased consumption and/or limited synthesis in infants remains to be determined. The fibronectin molecule turns over rapidly, and its plasma concentration in humans can decline quickly with malnutrition.[23] The provision of total parenteral nutrition to malnourished patients with low plasma fibronectin levels can initiate a relatively rapid restoration of normal fibronectin levels,[23] if the patients do not have sepsis. The half-life of plasma fibronectin in adults is 20–25 hours, suggesting a relatively high rate of synthesis of this molecule in the body. Patients studied by Gotta et al[4] demonstrated no postoperative complications or evidence of organ failure. These patients also demonstrated a rapid restoration of fibronectin levels, but a cause-effect relationship cannot be concluded from such data. Other authors have demonstrated that persistent plasma fibronectin deficiency in selected patients with coronary bypass was associated with a higher incidence of postsurgical complications.[18] Richards et al[15] demonstrated that, following surgery, patients with intra-abdominal infections, who manifest sustained plasma fibronectin deficiency, exhibit a higher incidence of multiple organ failure than do those patients with postoperative sepsis who demonstrate rapid recovery of plasma fibronectin levels after surgery.

The magnitude of the plasma fibronectin deficiency, which has now been clearly documented by several investigators in patients undergoing cardiopulmonary bypass, coupled with the number of patients per year that undergo this procedure, suggests that further studies into the clinical significance of transient plasma fibronectin deficiency are warranted. If prolonged fibronectin deficiency in selected patients can be documented to have an adverse effect on nonspecific phagocytic host defense, vascular permeability, or wound healing, then this deficiency can potentially be reversed with intravenous infusion of purified opsonically active human plasma fibronectin.[16] Such therapy must await completion of appropriate clinical trials. The current intense scientific interest in the biologic activity of fibronectin will provide new observations that have both basic medical and clinical significance.

THOMAS M. SABA, PhD
Professor and Chairman
Department of Physiology
Albany Medical College
Albany, NY 12208

1. Saba TM, Jaffe E: Plasma fibronectin (opsonic glycoprotein): Its synthesis by vascular endothelial cells and role in cardiopulmonary integrity after trauma as related to reticuloendothelial function. *Am J Med* 1980; 68:577–594.
2. Saba TM, Blumenstock FA, Scovill WA, et al: Cryoprecipitate reversal of opsonic alpha2-surface binding glycoprotein deficiency in septic surgical and trauma patients. *Science* 1978; 201:622–624.
3. Yamada KM, Olden K: Fibronectins: Adhesive glycoproteins of cell surface and blood. *Nature* 1978; 275:179–184.
4. Gotta AW, Carsons S, Abrams L, et al: Fibronectin levels during cardiopulmonary bypass. *NY State J Med* 1987; 87:493–496.
5. Hynes RO, Yamada KM: Fibronectins: Multifunctional modular glycoproteins. *J Cell Biol* 1982; 95:369–377.
6. McKeown-Longo PJ, Mosher DF: Mechanism of formation of disulfide-bonded multimers of plasma fibronectin in cell layers of cultured human fibroblasts. *J Biol Chem* 1984; 259:12,210–12,215.
7. Rouslahti E, Engvall E, Hayman E: Fibronectin: Current concepts of its structure and function. *Collagen Res* 1981; 1:95–128.
8. Saba TM, Blumenstock FA, Weber P, et al: Physiologic role for cold-insoluble globulin in systemic host defense: Implications of its characterization as the opsonic α_2SB glycoprotein. *Ann NY Acad Sci* 1978; 312:43–55.
9. Scovill WA, Saba TM, Blumenstock FA, et al: Opsonic alpha2 surface binding glycoprotein therapy during sepsis. *Ann Surg* 1978; 188:521–529.
10. Niehaus GD, Schumacker PT, Saba TM: Influence of opsonic fibronectin deficiency on lung fluid balance during bacterial sepsis. *J Appl Physiol* 1980; 49:693–699.
11. Deno DC, Saba TM, Lewis EP: Kinetics of endogenously labelled plasma fibronectin: Incorporation into tissues. *Am J Physiol* 1983; 245:R564–R575.
12. Oh E, Pierschbacher M, Ruoslahti E: Deposition of plasma fibronectin in tissues. *Proc Natl Acad Sci* (*USA*) 1981; 78:3218–3221.
13. Tamkun JW, Hynes RO: Plasma fibronectin is synthesized and secreted by hepatocytes. *J Biol Chem* 1983; 258:4641–4647.
14. Cohler LF, Saba TM, Lewis E, et al: Plasma fibronectin and lung protein clearance with gram-negative bacteremia: Response to fibronectin therapy. *J Appl Physiol* (in press).
15. Richards WO, Scovill WA, Shin B: Opsonic fibronectin deficiency in patients with intra-abdominal infection. *Surgery* 1983; 94:210–217.
16. Saba TM, Blumenstock FA, Shah DM, et al: Reversal of opsonic deficiency in surgical, trauma and burn patients by infusion of purified human plasma fibronectin. Correlation with experimental observations. *Am J Med* 1986; 80:229–240.
17. Gandhi JG, Vander Salm T, Szymanski IO: Effect of cardiopulmonary bypass on plasma fibronectin, IgG and C3. *Transfusion* 1983; 23:476–479.
18. Snyder EL, Barash PG, Mosher DF, et al: Plasma fibronectin levels and clinical status in cardiac surgery patients. *J Lab Clin Med* 1983; 102:881–889.
19. Silva J, Hoeksema H, Fekety FR Jr: Transient defects in phagocytic functions during cardiopulmonary bypass. *J Thorac Cardiovasc Surg* 1974; 67:175–183.
20. Subramanian V, Lowman JT, Gans H: Effect of extracorporeal circulation on reticuloendothelial function. *Arch Surg* 1968; 97:330–335.
21. Mosesson MW: Structure of human plasma cold-insoluble globulin and the mechanism of its precipitation in the cold with heparin or fibrin-fibrinogen com-

plexes. *Ann NY Acad Sci* 1978; 312:11-29.

22. Deno DC, McCafferty MH, Saba TM, et al: Mechanism of acute depletion of plasma fibronectin following thermal injury in rats. Appearance of a gelatinlike ligand in plasma. *J Clin Invest* 1984; 73:20-34.

23. Howard L, Dillon B, Saba TM, et al: Decreased plasma fibronectin during starvation in man. *J Parenter Enteral Nutr* 1984; 8:237-244.

Corticosteroid treatment of sarcoidosis—who needs it?

The finding of Zaki, Lyons, Leilop, and Huang[1] that prolonged corticosteroid therapy failed to influence the eventual outcome of sarcoidosis is an important step toward rational management of this disease. Their prolonged and arduous study corroborates the conclusions reached in five earlier controlled studies dealing with American,[2,3] European,[4,5] and Japanese[6] patients. It is difficult to believe that a significant improvement of treated patients could have been missed in all six trials.

The most important implication of the demonstration that corticosteroid treatment does not avert pulmonary fibrosis and permanent impairment of pulmonary function is that this medication should be restricted to the palliative treatment of disabling symptoms and physiologic derangements. Corticosteroids are effective in reducing ocular inflammation, correcting hypercalcemia, improving pulmonary function, and alleviating symptoms related to hepatic, splenic, articular, myocardial, neural, and cutaneous involvement. The symptomatic use of corticosteroids in this fashion is recommended by all investigators, and is required in a majority of black patients but in only a minority of white patients with sarcoidosis.

Disagreement about the indications for corticosteroid treatment of this disease has been to a considerable extent the result of confusing demonstration of disease activity with need for treatment. Many investigators[7] have assumed that the presence of active granuloma formation is an indication for treatment, and a host of techniques for measuring activity (serum angiotensin converting enzyme assays, gallium-67 scans of the lungs, bronchoalveolar lavage) have been advocated and widely employed as indicators of the need for treatment. These methods are more or less reliable measures of granuloma formation, but none has proven reliable for selecting patients who will have progressive tissue damage. Even the severity of initial roentenographic infiltration has no predictive value as to the eventual outcome.[8]

If corticosteroids do not significantly alter the outcome of sarcoidosis, there is no advantage to early institution of treatment and no point to the search for more sensitive indicators of granuloma activity. Rather, what is needed is a therapeutic agent that will do more than suppress inflammation. The search for a drug that will reduce the development of fibrosis or retard extrathoracic dissemination has thus far been fruitless, but research in this direction must continue.

The recognition that the action of cortiocosteroids is merely palliative should not diminish their clinical application in the treatment of patients with significant disability or suffering. Short three- or four-month courses of therapy are indicated for occasional cases of subacute sarcoidosis with severe extrathoracic manifestations. However, most patients who require corticosteroid therapy for respiratory disease have chronic relapsing disease that requires treatment for one to three years or longer. Since patients with sarcoidosis usually suffer chronic rather than acute illness, there is little point in starting with excessive doses greater than 30 mg daily. Most patients have fewer side effects if treatment is begun with 15 mg daily or 30 mg every other day, tapering the dose as improvement occurs.

Corticosteroids are not uniformly effective, and a dozen times a year we encounter patients unresponsive to moderate tolerable doses of steroids, who are better treated with chlorambucil.[9,10] Whether treated with corticosteroids or chlorambucil, patients frequently relapse after termination of treatment, and close surveillance is necessary for several years afterward.

HAROLD L. ISRAEL, MD, MPH
Emeritus Professor of Medicine
Division of Pulmonary Disease
Department of Medicine
Jefferson Medical College
Thomas Jefferson University
Philadelphia, PA 19107

1. Zaki MH, Lyons HA, Leilop L, et al: Corticosteroids in sarcoidosis: A five-year controlled therapeutic follow-up study. *NY State J Med* 1987; 87:496-499.

2. Israel HL, Fouts DW, Beggs RA: A controlled trial of prednisone treatment of sarcoidosis. *Am Rev Respir Dis* 1973: 107:609-614.

3. Harkleroad LE, Young RL, Savage PJ, et al: Pulmonary sarcoidosis. Long-term follow-up of the effects of steroid therapy. *Chest* 1982; 82:84-87.

4. Selroos O, Sellergren TL: Corticosteroid therapy of pulmonary sarcoidosis. A prospective evaluation of alternate day and daily dosage in stage II disease. *Scand J Respir Dis* 1979; 60:215-221.

5. Eule H, Weinecke A, Roth I: The possible influence of corticosteroid therapy on the natural course of pulmonary sarcoidosis. *Ann NY Acad Sci* 1986; 465:695-701.

6. Yamamoto M, Saito N, Tachibana T, et al: Effects of an 18 month corticosteroid therapy to stage I and stage II sarcoidosis patients (a control trial), in Chretien J, Marsac J, Saltiel JC (eds): *Sarcoidosis and Other Granulomatous Disorders*. Paris, Pergamon Press, 1980, pp 470-474.

7. Keogh BA, Hunninghake GW, Line BR, et al: The alveolitis of pulmonary sarcoidosis. Evaluation of natural history and alveolitis-dependent changes in lung function. *Am Rev Respir Dis* 1983; 128:256-265.

8. Israel HL, Karlin P, Menduke H, et al: Outcome of sarcoidosis related to race, extrathoracic involvement and initial roentgenographic lesions. *Ann NY Acad Sci* 1986; 465:609-618.

9. Israel HL: Effects of chlorambucil and methotrexate in sarcoidosis, in Lavinsky L (ed): *Proceedings of the International Conference on Sarcoidosis*. Prague, University Karlova Press, 1971, pp 632-634.

10. Kataria YP: Chlorambucil in sarcoidosis. *Chest* 1980; 78:36-43.

The role of the physician as the patient's advocate*

Recent surveys have shown that most Americans are eminently satisfied with the medical care they are receiving. New Yorkers generally agree. We have, we are told, the finest institutions and the best doctors, and, as we learn from the media and from television commercials, we have become a mecca for individuals from all over the world who seek our "miracles of modern medical science."

All this is true. But the percentages of survey respondents who are less than pleased with their health care are growing. Complaints from patients about mistreatment, maltreatment, and neglect are becoming more numerous and more strident. And, such complaints are being converted into malpractice claims in numbers and magnitudes that have created a crisis for all parties (except perhaps for the plaintiffs' attorneys).

In my opinion, the problem is that too many physicians have forsaken their traditional role as advocate for their patients. The concept of the physician as the patient's advocate is not new. It was first defined about 2,500 years ago by Hippocrates, who wrote in his first aphorism: "It is necessary for the physician to provide not only the needed treatment, but to provide for the sick man himself, and for those beside him, and to provide for his outside affairs." [1]

This definition has come down through the ages and was reiterated as recently as 1986, in the report of the American Medical Association's Council on Ethical and Judicial Affairs, which said:

> The trust and dependence reposited in the pysician invokes an ethical obligation on the part of the physician far greater than that of the commercial purveyor of services. The obligation of the physician is to be an advocate for the patient. A physician must exercise medical judgment independently of his own or a third party's financial interests. No motive should be allowed to prevail against the physician's fundamental role of alleviating the suffering of his patients.[2]

This was rephrased in the decision by Judge Cooperman of the California appellate court in the case of *Wickline v State of California*.[3] In placing third party payers in legal jeopardy when medically inappropriate decisions result from defects in the design or implementation of cost containment mechanisms, he went on to say:

> The physician who complies without protest with the limitations imposed by the third party payor when his medical judgment dictates otherwise, cannot avoid his ultimate responsibility for his patient's care. He cannot point to the health care payor as the liability scapegoat when the consequences of his own determinative medical decisions go sour.

It must be made clear that the physician's advocacy is quite different from that of an attorney. The attorney's advocacy is narrow: He studies the evidence and presents only that which supports his client's contention and strives mightily to discredit the evidence presented by his adversary, the attorney for the other side. The physician's advocacy is of a different sort: He uses medical art and science to develop a diagnosis and a course of action which he will try to get the patient to accept, whether the patient wants it or not. It does not help the patient to hear only the evidence that pleases him.

Too often, physicians mimic the advocacy of the attorney. One sees this in the professional witnesses who are always ready to testify that a patient is severely disabled (or that he is not really impaired); that the severe back problem was directly caused by lifting something (or that it had nothing to do with it).

One frequently sees less dramatic examples of this type of advocacy in which the physician certifies an inappropriate disability claim; conceals a test result; lies about a blood pressure because the patient asks him to; or proposes a regimen of treatment more likely to enhance the physician's revenues than the patient's well-being. The great significance of these "little lies" is that they undermine the inherent integrity of the physician.

Much of the problem can be attributed to new medical technology and a payment system that rewards procedures. This has led to superspecialization and fragmentation of care. The new instruments and techniques involve arcane expertise that only a few can acquire. As a result, one has the patient under the care of a rheumatologist for arthritis, a hand specialist for a wrist injury, a foot specialist for painful feet, an internist for gastrointestinal symptoms produced by the nonsteroidal anti-inflammatory agents, etc. Medicine has become a profession of specialists who treat particular diseases or organ systems, with the patient often getting lost in the shuffle.

One local hospital has organized a "liaison psychiatric team" comprising psychiatrists, social workers, and others whose involvement has improved patient outcomes and tolerance for treatment in such cases as fractured hips, chemotherapy for cancer, and others. Here is the ultimate: one doctor to treat the disease; another to treat the patient.

Whole generations of medical students and residents have been so imbued with medical science that they can be regarded as mere medical technicians. They have great expertise and do wondrous things but it cannot be said that they really "take care of patients." Fortunately, medical education is changing, and once again students and residents are being exposed to humanistic concepts and being trained in communication skills so that they can really relate to their patients as people. It needs to be emphasized that it is not a case of "either/or"—the humane physician must still have scientific knowledge and technical skill.

---

* Presented at the Annual CME Assembly of the Medical Society of the State of New York, March 13, 1987.

Economic pressures are often cited as a basis for physician advocacy. Physicians must stand up for their patients against the payors whose cost containment initiatives would block the availability of services they really need. One hears of patients being denied hospital admission and being discharged "quicker and sicker." But one wonders how much of the physicians' advocacy is really motivated by their drive to maintain their revenues, by their resistance to changing profligate practice patterns, and, most of all, by their objection to intrusion on the physician's autonomy by third party payors and their representatives. That they appear this way suggests the need for change in the way physicians present their arguments.

As we all know, it is a changed world out there. Patients are learning to be more assertive and aggressive and are being coached in the questions to ask. A whole new industry of second surgical opinions, preadmission certification, and utilization review has grown out of the inference that physicians' recommendations are not always the most appropriate for their patients.

There are now highly sophisticated data systems that monitor the behavior of physicians, hospitals, and other providers, as well as individual patients, to detect aberrations that may suggest at the least bad habits that can be corrected and at the worst, gouging and outright venal behavior.

Where do we go from here? I contend that the physician should resume the role of the case manager, not only in the sense of the "gate keeper" who bars expenditures for needless or inappropriate medical services, but as one who takes charge and truly manages the care of the patient, and by extension manages also the response of the patient's family or significant others. The physician/advocate should be responsible for all referrals, not only for making them, but for evaluating and integrating the recommendations that evolve.

The physician should act as the patient's advocate in confronting hospital administrators and others who sometimes compromise the well-being if not the health of the patient. The quality of caring in many of our most prestigious hospitals is abominable. Economics and staff shortages may be valid excuses but the physician should not yield to them. "VIP suites" are not the answer. Yes, many patients are overdemanding and unrealistic in their expectations, but surely, at the least, they can expect to be treated with dignity and respect.

The physician who truly cares about the patient will get to know the person as well as the diseased organ, and by acting as an advocate on that patient's behalf will help to restore the respect and admiration that the medical profession is so rapidly losing.

LEON J. WARSHAW, MD
Executive Director
The New York Business Group
on Health, Inc
New York, NY 10017

1. Richards DM Jr: The first aphorism of Hippocrates. *Perspect Biol Med* 1961; 5:61.

2. Dickey NW: Report of the Council on Ethical and Judicial Affairs, Rept No. A(1-86). American Medical Association, 1986.

3. *Wickline v State of California*, Case No. NWC 60672, 1986.

Fibronectin levels during cardiopulmonary bypass

ALEXANDER W. GOTTA, MD; STEVEN CARSONS, MD; LAWRENCE ABRAMS, MD; ANN E. KEANY, CRNA

ABSTRACT. The concentration of plasma fibronectin was determined in nine patients undergoing coronary artery bypass grafting using cardiopulmonary bypass. Fibronectin levels were determined by electrophoresis using monospecific serum and were contrasted with alterations in plasma albumin level, used as an index of dilution during bypass. Fibronectin levels declined sharply (59.3%) immediately after bypass was started, and this decline was significantly greater ($p < 0.05$) than the decline in albumin (36%) at the same time. Fibronectin levels subsequently rose, but never reached pre-bypass levels. The decrease in fibronectin was not due solely to dilution by the pump prime, but was probably due in part to opsonization of cellular debris generated by surgery and bypass and also to cold precipitation of the heparin-fibrinogen-fibronectin complex. Adsorption onto the tubing of the bypass apparatus may also have contributed to the decline. The previously described decrease in opsonic activity after cardiopulmonary bypass may be due to alterations in the fibronectin level, as fibronectin is a major opsonin-marking particulate matter for ingestion by the reticuloendothelial system.

(NY State J Med 1987; 87:493–496)

Circulating blood is cleansed of foreign particulate matter by the macrophage cells of the reticuloendothelial system (RES)[1] in the liver, spleen, lungs, and bone marrow. Particles are engulfed, withdrawn from circulation, metabolized, detoxified, or stored and released later. Prior to phagocytosis, however, foreign matter must first be identified and marked by an opsonin, and only then can the cleansing process be carried out. Failure of opsonization and phagocytosis to occur leads to foreign particulate microembolization and, if it is of sufficient magnitude, can lead to multiorgan micro- and macroinfarction, and consequent organ failure, with the kidneys and lungs being at particular risk.[2,3] Trauma to the cellular components of the blood as they pass through the cardiopulmonary bypass apparatus, combined with surgical manipulation, generates foreign matter that requires removal by the RES.[4-6] The rate-limiting factor in RES function is not cellular activity but, rather, opsonization; and decreased levels of circulating opsonin lead to apparent RES failure.[1] Opsonic activity is decreased during cardiopulmonary bypass,[7-9] and this decrease may play a role in the increased risk of postoperative infection after cardiopulmonary bypass.[8,10,11] Plasma fibronectin is a major nonimmunoglobulin, noncomplement opsonin, and estimation of circulating levels of this substance serves as an index of opsonic activity.[2,12,13] We have determined plasma fibronectin levels during extracorporeal circulation in otherwise healthy patients undergoing coronary artery bypass grafting, in order to assess whether alterations in opsonic activity might be due to changes in fibronectin concentrations.

PATIENTS AND METHODS

The study was approved by the Committee on Investigations Involving Human Subjects of the State University of New York Health Science Center at Brooklyn. After providing their informed consent, nine patients scheduled for coronary artery bypass were anesthetized with fentanyl, 20 μg/kg intravenously, pancuronium, 0.08 mg/kg intravenously, and isoflurane, 1% ± 0.5% inhaled in oxygen. The pump prime was composed of 1,000 mL normal saline, 1,000 mL 5% dextrose in water, 50 mL sodium bicarbonate solution (44.6 meq), 3 mL heparin solution (3,000 units), and 250 mL albumin solution (12.5 g), to total 2,303 mL. Five hundred mL was withdrawn for possible later use, leaving 1,803 mL for the prime. This solution was cooled to 5–10°C before bypass, and the patient was cooled to 20–22°C after bypass was started. The cardiopulmonary bypass apparatus was a Sarns bubble oxygenator, using disposable Bentley tubing. Heparin, 3 mg/kg, was administered intravenously prior to bypass. Ten mL of whole blood was drawn via a central venous line at these points:

after the administration of heparin, but before bypass;

five minutes after bypass started;

mid-bypass, ie, at the point where rewarming was begun (mean bypass time was 120 min, and mid-bypass averaged 60 min);

immediately prior to coming off bypass;

off bypass, but before administration of protamine.

Blood samples were centrifuged at 400 × g, and supernatant plasma was decanted, added to tubes containing the proteinase inhibitors trasylol (500 U/mL) and phenylmethylsulfonyl fluoride (1 mM), and then stored at 20°C until assayed. Plasma fibronectin levels were determined by rocket electrophoresis.

Antifibronectin antiserum was prepared in rabbits. Whole rabbit antiserum was absorbed with fibronectin-depleted human plasma. Monospecificity was assured by immunoelectrophoresis. Monospecific antihuman fibronectin serum was prepared in rab-

---

From the Departments of Anesthesiology (Drs Gotta and Abrams, and Ms Keany) and Medicine (Dr Carsons), State University of New York Health Science Center at Brooklyn, NY.

Address correspondence to Dr Gotta, Professor, Department of Anesthesiology, State University of New York Health Science Center at Brooklyn, 450 Clarkson Ave, Box 6, Brooklyn, NY 11203.

bits and absorbed with fibronectin-depleted human plasma as previously described.[14] Fibronectin was isolated from normal human plasma by gelatin-Sepharose affinity chromatography.[14] The antiserum was incorporated into 1% molten agarose (1:125 v/v) and then poured on a glass slide. Ten wells were punched in the gel; serial dilutions of a fibronectin standard plasma were placed into four of the wells. The remaining six wells received 1.5 μL of the sample. Electrophoreses were carried out for 16 hours at 60V. The height of the rocket precipitation arc was proportional to the fibronectin concentration as calculated from the standard curve.

Since fibronectin levels would obviously be decreased by mixing with the non-opsonin-containing fluids in the pump prime, plasma albumin levels were determined by radial diffusion at the same time as the fibronectin levels were measured, and these were used as an index of dilution.

The fibronectin levels for all patients were averaged, and the level determined from the sample taken prior to bypass was considered baseline. Deviations from baseline were tested for significance using Student's test, with $p < 0.05$ considered significant.

RESULTS

After correcting for the albumin added in the prime, mean plasma albumin levels decreased 36.7% immediately after bypass was started ($p < 0.05$). Estimating the average patient's blood volume at 5,000 mL, the pump prime of 1,803 mL should produce a dilution of 36.1%. Thus the use of albumin as an index of dilution seems valid. Plasma fibronectin levels decreased significantly immediately after beginning cardiopulmonary bypass, declining from a mean of 435 μg/mL to 177 μg/mL immediately after bypass was started, and then rose to a plateau of approximately 300 μg/mL after the mid-bypass point (Table I). The fibronectin levels during bypass differed significantly from baseline, with a decline of 59.3% immediately after bypass was started ($p < 0.001$), 41.4% at mid-bypass ($p < 0.01$), 28.0% just prior to ending bypass ($p < 0.0025$), and 30.1% ($p < 0.05$) at the completion of bypass. The decline in fibronectin at five minutes (59.3%) was significantly greater ($p < 0.05$) than the decline in albumin (36.7%) at the same time. Albumin levels varied slightly during bypass but did not differ significantly from those obtained 15 minutes after bypass was started.

DISCUSSION

Fibronectin is a major circulating plasma glycoprotein and is also found on the cell surface and in extracellular matrix.[15] It is similar to cold-insoluble globulin (CIg). The protein is composed of two polypeptide chains of approximately 220,000 daltons linked together by disulfide bridges, with a total molecular weight of 450,000 daltons. The bulk of the circulating protein is probably derived from synthesis by hepatocytes and vascular endothelial cells. Normal plasma levels are approximately 350 μg/mL, are somewhat higher in men, and tend to increase with age.

Fibronectin possesses opsonic properties, marking particulate matter for ingestion by the reticuloendothelial system. Fibronectin may function as the major opsonin for particulate debris, since opsonic activity for these substances as determined by bioassay appears to parallel circulating plasma fibronectin levels. Other opsonins, such as properdin, factor B, and C3, may share the ability to opsonize particulate matter, but they specialize in antibacterial activity.[16] Multiple studies have also demonstrated the importance of heparin as a cofactor in fibronectin-mediated opsonization.[17-19] Decreased RES activity after high particulate load may be a reflection of depleted opsonic capacity rather than impaired cellular function.[20] Extensive surgery, trauma, burns, and bacterial septicemia cause tissue destruction and generate particulate matter and decreased fibronectin levels.[13,21] In these conditions, hypoopsonemia ordinarily lasts 72 hours and is followed by hyperopsonemia, which reaches a peak in 11–30 days.[21] Failure of hyperopsonemia to develop is associated with significant morbidity and mortality, which can be improved, however, by the administration of cryoprecipitate that contains plasma fibronectin.[13,22]

Fibronectin has also been implicated in the thrombogenicity of biomaterials,[23] and decreased levels of the opsonin may alter fibrin and platelet deposition on plastic surfaces, such as the cardiopulmonary bypass tubing. Plasma fibronectin maintains solubility of fibrin monomers and can resolubilize fibrin in insoluble fibrin and fibrinogen complexes, thus suggesting an antithrombotic mechanism for fibronectin.[24] Decreased fibronectin levels may lead to fibrin polymerization and/or fibrin-collagen binding and precipitation.

Previous studies have demonstrated decreases in fibronectin levels during or after cardiopulmonary bypass but have left unanswered the following question: Does this decrease occur solely because of dilution of non-fibronectin-containing fluids, or is active consumption of the opsonin of primary importance?[25-27] There is evidence that car-

TABLE I. Fibronectin (F) and Albumin (A) Levels

Patient	Age (yr)	Sex	A1* g/dL	A2† g/dL	F1 μg/mL	F2 μg/mL	F3 μg/mL	F4 μg/mL	F5 μg/mL
1	55	M	2.20	2.06	710	260	302	350	418
2	51	M	2.50	2.36	300	111	192	246	282
3	63	F	1.60	0.56	438	170	243	370	226
4	60	M	2.10	1.26	546	225	255	498	384
5	71	M	2.30	1.26	279	21	339	186	324
6	61	F	2.20	1.50	548	290	356	290	276
7	57	M	2.10	0.66	261	207	84	282	228
8	46	M	2.90	1.76	514	214	332	360	364
9	52	M	2.70	1.56	322	98	190	238	230
Mean	57		2.29	1.45	435	177	255	313	304
(±SEM)‡	(0.8)		(0.13)	(0.17)	(51.7)	(28.6)	(29.6)	(30.9)	(24.1)

* A1 is albumin level immediately before bypass; A2 is albumin level 15 minutes after bypass; F1 is fibronectin level immediately before bypass; F2 is fibronectin level 15 minutes after bypass; F3 is fibronectin level at mid-bypass; F4 is fibronectin level just prior to ending bypass; F5 is fibronectin level immediately after ending bypass.

† Albumin levels remained unchanged after 15 minutes after bypass.

‡ SEM = standard error of the mean.

diopulmonary bypass causes complement activation, with a decrease of C3 and C4 which is not attributed to hemodilution or infusion of non-complement-containing fluids.[27] In one study, fibronectin levels decreased by 34.5% during bypass and then returned to normal within four to five hours after surgery.[25] Subsequent decreases in opsonin levels were associated with adverse outcomes, suggesting a relation between fibronectin levels and postoperative morbidity and mortality. In another study, fibronectin levels fell to 32% of preoperative concentration; this was attributed to consumption, because the fibronectin level decreased more than the levels of IgG and C3.[26]

The patients in the study reported here were clinically stable and would not be expected to have decreased plasma fibronectin levels. The initial, profound decrease in fibronectin observed during cardiopulmonary bypass is not entirely explained by dilution, since albumin, used as the index of dilution, is decreased to a lesser extent. In these patients, the immediate decrease may have been due to utilization in the clearance of circulating debris generated from the formed elements of the blood. Alternatively, reversible adsorption of fibronectin to the bypass apparatus may have occurred. Fibronectin adsorption to Silastic or polyvinylchloride from the blood of heparinized animals is maximal at 15–30 minutes and is subsequently released from the biomaterial over the ensuing 60–90 minutes.[23] After eluting a used oxygenator membrane with Tris buffer and 0.15M saline, we were unable to demonstrate fibronectin by polyacrylamide gel electrophoresis in the presence of sodium dodecyl sulfate (SDS-PAGE) in the solution subsequently eluted by 8M urea. This suggests that little fibronectin was adsorbed to the oxygenator apparatus at the end of the procedure, consistent with the animal studies cited above. Fibronectin isolated from the whole plasma of a patient at the 15-min point (corresponding to the lowest level) revealed the presence of a band at 220 kD on SDS-PAGE, suggesting that the reduced levels were not secondary to extensive fragmentation of the protein during bypass.

Heparin forms a cold-precipitable complex with CIg, or with mixtures of CIg and fibrinogen, but not with fibrinogen alone.[18,19] While fibrinogen is not essential for heparin-induced precipitation, the presence of fibrinogen reduces the threshold for heparin-induced CIg cryoprecipitation and, by precipitating with the heparin CIg complex, increases the total amount of precipitate. The optimal level of heparin for CIg precipitation in normal plasma is 0.15–0.25 mg/mL at 2°C, and at this concentration, 88% or more of the plasma CIg will precipitate.[18] Allowing for full dilution of administered heparin in the estimated blood volume, this heparin concentration appears attainable. Thus, with the addition of the cold pump prime, a heparin-fibronectin precipitate may have formed; this would explain, at least in part, the decreased fibronectin levels. Increasing the concentration of heparin will decrease heparin-CIg complex formation and precipitation, but this effect becomes important only at heparin concentrations of 1–2 mg/mL,[18] which were not obtainable in our patients.

After one hour, plasma fibronectin levels began to increase. This increase may have been due to rapid mobilization of secreted fibronectin to the plasma space or to transformation of cell surface fibronectin into plasma fibronectin. Release of precipitated fibronectin may also have been facilitated by the increase in temperature.

The clinical courses of these nine patients were uneventful, with no evidence of organ failure or coagulopathy. This may have been due to rapid restoration of fibronectin levels, although fibronectin concentrations per mL of plasma remained somewhat below normal. Each patient in this series received two units of fresh frozen plasma after bypass was completed, in order to abort the development of coagulopathy, and this may have contributed to the restoration of normal opsonic activity in the postoperative period. Although we have demonstrated decreased fibronectin levels and thus decreased opsonic activity, the role of plasma product administration in the restoration of opsonic activity in patients undergoing cardiopulmonary bypass remains speculative.

REFERENCES

1. Saba TM: Physiology and physiopathology of the reticuloendothelial system. *Arch Intern Med* 1970; 120:1031–1052.

2. Scovill WA, Saba TM, Blumenstock FA, et al: Opsonic alpha2 surface binding glycoprotein therapy during sepsis. *Ann Surg* 1978; 188:521–529.

3. Pardy BJ, Dudley HAF: Post-traumatic pulmonary insufficiency. *Surg Gynecol Obstet* 1977; 144:259–269.

4. Ashmore PG, Svitek V, Ambrose P: The incidence and effects of particulate aggregation and microembolism in pump-oxygenator systems. *J Thorac Cardiovasc Surg* 1968; 55:691–697.

5. Kvarstin B, Cappelen C, Osterud A: Blood platelets and leucocytes during cardiopulmonary bypass. *Scand J Thorac Cardiovasc Surg* 1974; 8:142–145.

6. Mortensen JD: Evaluation of tests for blood damage produced by oxygenators. *Trans Am Soc Art Intern Organs* 1977; 23:747–755.

7. Subramanian V, Lowman JT, Gans H: Effect of extracorporeal circulation on reticuloendothelial function. *Arch Surg* 1968; 97:330–335.

8. Hairston P, Manos JP, Graber CD, et al: Depression of immunologic surveillance by pump-oxygenator perfusion. *J Surg Res* 1969; 9:587–593.

9. Silva J, Hoeksema H, Fekety FR Jr: Transient defects in phagocytic functions during cardiopulmonary bypass. *J Thorac Cardiovasc Surg* 1974; 67:175–183.

10. Iribarren COS, Ekestrom S: The causes of death after open-heart surgery. *J Thorac Cardiovasc Surg* 1964; 47:725–739.

11. Goodman JS, Schaffner W, Collins HA, et al: Infection after cardiovascular surgery. Clinical study including examination of antimicrobial prophylaxis. *N Engl J Med* 1968; 278:117–123.

12. Saba TM, Blumenstock FA, Weber P, et al: Physiologic role of cold-insoluble globulin in systemic host defense. Implications of its characterization as the opsonic alpha2 surface-binding glycoprotein. *Ann NY Acad Sci* 1978; 312:43–55.

13. Scovill WA, Saba TM, Blumenstock FA, et al: Opsonic alpha2 surface binding glycoprotein therapy during sepsis. *Ann Surg* 1978; 188:521–529.

14. Carsons S, Lavietes B, Diamond HS, et al: The immunoreactivity, ligand, and cell binding characteristics of rheumatoid synovial fluid fibronectin. *Arthritis Rheum* 1985; 28:601–612.

15. Saba TM, Jaffe E: Plasma fibronectin (opsonic glycoprotein): Its synthesis by vascular endothelial cells and role in cardiopulmonary integrity after trauma as related to reticuloendothelial function. *Am J Med* 1980; 68:577–594.

16. Alexander JW, McClellan MA, Ogle CK, et al: Consumptive opsoninopathy. Possible pathogenesis in lethal and opportunistic infection. *Ann Surg* 1976; 184:672–678.

17. Saba TM, Filkins JP, DiLuzio NR: Properties of the "opsonic system" regulating in vitro hepatic phagocytosis. *J Reticuloendothelial Soc* 1966; 3:398–414.

18. Stathakos NE, Mosesson M: Interactions among heparin, cold-insoluble globulin, and fibrinogen in formation of the heparin-precipitable function of plasma. *J Clin Invest* 1977; 60:855–865.

19. Mosesson MW: Structure of human plasma cold-insoluble globulin and the mechanism of its precipitation in the cold with heparin or fibrin-fibrinogen complexes. *Ann NY Acad Sci* 1978; 312:11–29.

20. Jenkin CR, Rowley D: The role of opsonins in the clearance of living and inert particles by cells of the reticuloendothelial system. *J Exp Med* 1961; 114:363–374.

21. Scovill WA, Saba TM, Kaplan JE, et al: Deficits in reticuloendothelial humoral control mechanisms in patients after trauma. *J Trauma* 1976; 16:898–904.

22. Saba TM: Prevention of liver reticuloendothelial systemic host defense failure after surgery by intravenous opsonic glycoprotein therapy. *Ann Surg* 1978; 188:142–152.

23. Young BR, Lambrecht LK, Cooper SL: Plasma proteins: Their role in initiating platelet and fibrinogen deposition on biomaterials. *Adv Chem* 1982; 199:317–350.

24. Kaplan JE, Snediker PW: Maintenance of fibrin solubility by plasma fi-

bronectin. *J Lab Clin Med* 1980; 96:1054–1061.

25. Snyder EL, Barash PG, Mosher DF, et al: Plasma fibronectin levels and clinical status in cardiac surgery patients. *J Lab Clin Med* 1983; 102:881–889.

26. Gandhi JG, Salm TV, Szymanski IO: Effect of cardiopulmonary bypass on plasma fibronectin, IgG, and C3. *Transfusion* 1983; 23:476–479.

27. Chiu RC, Samson R: Complement (C3,C4) consumption in cardiopulmonary bypass, cardioplegia, and protamine administration. *Ann Thorac Surg* 1984; 37:229–232.

See also pp 487–490.

Corticosteroid therapy in sarcoidosis

A five-year, controlled follow-up study

MAHFOUZ H. ZAKI, MD, DrPH; HAROLD A. LYONS, MD[†]; LINDA LEILOP, MD;
CHIN TANG HUANG, MD

ABSTRACT. One hundred eighty-three patients suffering from sarcoidosis were included in a controlled, double-blind therapeutic trial to test the efficacy of corticosteroids in the treatment of the disease.

Rigid criteria were utilized for diagnosis and evaluation. Patients were divided into two groups: one group was treated with prednisone for at least a two-year period, and the other was given a placebo. A randomized block design was used to ensure comparability and randomization among the various subgroups. The experimental and control groups compared relatively well regarding the stage of the disease, the duration of sickness, age, and sex. All but four patients were black. Twenty-four patients were excluded for various reasons not directly related to therapy or sarcoidosis.

Patients were followed for at least a two-year period, the majority for more than three years. The efficacy of therapy was determined through roentgenographic and pulmonary function testing. Slightly greater improvement was noted in the chest radiographs of patients receiving prednisone than patients on placebos. These differences were insignificant among all subgroups except one.

Pulmonary function tests did not show a specific trend among the groups. An apparent but insignificant improvement in forced vital capacity, forced expiratory volume, and diffusing capacity for carbon monoxide was observed in patients treated with prednisone compared to patients receiving placebo.

The results of this study indicate that among this group of sarcoidosis patients, prolonged therapy with prednisone did not significantly affect the prognosis, as determined by chest radiographs and pulmonary function tests.

(NY State J Med 1987; 87:496–499)

The effectiveness of steroid therapy in the treatment of pulmonary sarcoidosis has been controversial, despite the use of steroids for more than 30 years.

Stone and coworkers[1] studied 22 male patients who were treated with cortisone for a six-year period. The authors noted variations in pulmonary function that were attributed to differences in preexisting pathologic lesions. The majority of these cases worsened after treatment, and the authors concluded that cortisone probably accelerated the development of pulmonary fibrosis.

Israel and coworkers[2] treated 36 patients with cortisone and noted symptomatic improvement among those patients whose illnesses were of short duration. The long-term effects, however, were less impressive.

Israel and coworkers[3] later studied 90 patients who were treated for three months and followed for one year in a randomized, controlled double-blind trial. The authors observed significant palliative effect; however, the steroid therapy was unable to avert pulmonary fibrosis or extrathoracic manifestations.

Colp and coworkers[4] studied pulmonary function in 120 patients with sarcoidosis. Some of these patients were treated with steroids, others were not. In the authors' opinion, steroid therapy hastens the granulomatous resolution and thus should be recommended for patients with functional impairment. Among the untreated group, there was a tendency for the diffusing capacity to decrease slightly and for the vital capacity to remain the same.

Johns and coworkers[5] noted during a five-year follow-up of 192 patients that clinical improvement was dependent on the administration of steroids, and that continued treatment was required in 48% of patients because of recurrent clinical relapses when treatment was withdrawn. In the authors' opinion, long-term maintenance treatment with low doses of prednisone (10–15 mg daily) may be indicated when relapses occur with marked frequency.

DeRemee[6] found steroid therapy to be effective in suppressing the granulomatous reaction. In his opinion, patients with Stage I sarcoidosis should be followed with frequent chest radiographs, since resolutions often occur as the disease progresses. Patients in Stages II and III should be treated with prednisone on alternate days, and the dose should be adjusted according to the clinical progress.

Harkleroad and coworkers[7] conducted a 15-year pro-

From the Departments of Medicine and Preventive Medicine and Community Health, State University of New York Health Science Center at Brooklyn, NY.

Address correspondence to Dr Zaki, Director, Division of Public Health, Suffolk County Department of Health Services, 225 Rabro Dr E, Hauppauge, NY 11788.

This work was supported by USPHS Grant No. RO1 HL 16651.

[†] Deceased.

TABLE I. Patients by Age and Therapeutic Regimen

Age (yr)	Drug	Placebo
<25	18	15
25–34	42	30
35–44	19	10
≥45	14	9
Unspecified	1	1
Total	94	65

spective study of two groups of patients with sarcoidosis. One group of 25 was treated with prednisone, the other was not and was used as a control. Both groups were matched for age, sex, ethnic background, degree of pulmonary dysfunction, and duration of disease. This small sample did not show any beneficial effects resulting from early steroid therapy.

Eule and coworkers[8] reported a nine-year follow-up study of 172 patients with sarcoidosis. Two thirds of the patients were treated with corticosteroids and one third were not. In this study, corticosteroid therapy had an immediate suppressive effect on the granulomatous process but no real effect on the long-term course of sarcoidosis Stages I, II, and III.

The questionable efficacy of steroid therapy in sarcoidosis during the past two decades prompted the initiation of the present study, the main objective of which was to evaluate the efficacy of corticosteroids in the treatment of sarcoidosis under controlled conditions.

PATIENTS AND METHODS

In view of the difficulties often encountered in the diagnosis of sarcoidosis, the following rigid criteria were utilized prior to admission to the study:

Clinical manifestations compatible with sarcoidosis;

No prior therapy with antituberculosis drugs, cortisone, anti-inflammatory drugs, or immunosuppressive agents;

Skin anergy to tuberculin, blastomycin, coccidioidin and histoplasmin;

No exposure to silica, beryllium, or other granuloma-inducing agents;

No prior evidence of tuberculosis, fungal, or other granulomatous diseases;

Tissue biopsy compatible with sarcoidosis;

Negative cultures for *Mycobacterium tuberculosis* and fungi

When the diagnosis of sarcoidosis was confirmed, patients were classified according to two variables:

Stage of disease based on chest film findings:
Nonpulmonary abnormalities (Stage 0)
Bilateral hilar lymphadenopathy (Stage I)
Bilateral hilar lymphadenopathy with parenchymal lung involvement (Stage II)
Parenchymal lung involvement alone (Stage III)

Duration of disease:
Less than six months
Six months to less than 12 months
More than 12 months

A randomized block design was utilized. Within each block, patients were matched in pairs according to age, sex, ethnic background, and duration of disease. One member of each pair was assigned at random to the study group, the other to the control group. A double-blind technique was used in order to eliminate both experimenter and subject bias. All patients gave informed consent, and institutional approval was procured.

Patients on drug therapy were given 40 mg of prednisone daily for three months and then maintained on 20 mg daily for at least two years. Patients in the control group received the equivalent number of similar, but inert, tablets. In most instances, the originally prescribed course of therapy, namely a prednisone regimen or a placebo, was followed during the therapeutic period. On a few occasions, the code had to be broken because of serious side reactions or a concomitant clinical condition that required the administration of appropriate medication. These patients were excluded from the final analysis.

On admission to the study, patients were given a steroid therapy sheet, which outlined the possible side reactions. Patients were asked to visit the clinic on a monthly basis to provide a complete history and undergo a physical examination. At bimonthly intervals, a standard chest film (posteroanterior and lateral view) was obtained, and at six-month intervals, physiologic studies were performed. The latter included forced vital capacity (FVC), forced expiratory volume in 1 second (FEV_1), maximal midexpiratory flow rate (MMFR), diffusing capacity for carbon monoxide (DLCO), and arterial oxygen tension (PO_2).

RESULTS

A total of 183 patients with sarcoidosis (Stages 0, I, II, and III) who satisfied the aforementioned criteria were admitted to the study. Of these, 24 had to be excluded because of lack of cooperation, relocation, concomitant medical problems (pregnancy, hypertension, cancer, etc), and death. The exclusion of these 24 patients does not introduce any bias in the final analysis, since the reasons for exclusion were not directly related to sarcoidosis or to the therapeutic regimen.

Table I shows the distribution of patients by age and therapeutic regimen. As is noted, more than two thirds of the patients were below 35 years of age. Seventy-five percent of patients were women, and all but four patients were black. Table II shows the distribution of patients by pathologic lesions, period of disease, and therapeutic regimen. Bilateral hilar lymphadenopathy was the most prevalent pathologic lesion, whether seen alone (Stage I) or in addition to parenchymal infiltrates (Stage II). Only 19 patients presented with parenchymal lesions alone (Stage III), and 17 with nonpulmonary sarcoidosis (Stage 0). The study and control groups compared well in relation to these variables. More than 70% of patients were followed for periods in excess of three years, and 35% were followed for a four-year period.

TABLE II. Patients by Pathologic Condition, Period of Disease, and Therapeutic Regimen

Chest Film Findings	Drug			Placebo			Total	
	<6 mo	≥6 mo	≥12 mo	<6 mo	≥6 mo	≥12 mo	Drug	Placebo
Hilar adenopathy	25	6	7	15	5	6	38	26
Both hilar adenopathy and parenchymal lesions	21	6	7	18	3	4	34	25
Parenchymal lesions	4	4	5	3	1	2	13	6
Others*	—	9	—	—	8	—	9	8
Total	50	25	19	36	17	12	94	65

* Nonpulmonary sarcoidosis.

TABLE III. Radiologic Findings Following Therapy by Type of Therapy

	Drug*	Placebo*
Complete improvement	21 (27.3%)	13 (22.8%)
Partial improvement	30 (39.0%)	20 (35.1%)
No change	21 (27.3%)	17 (29.8%)
Worsening	4 (5.2%)	6 (10.5%)
Normal at initial and final examinations	1 (1.3%)	1 (1.8%)
Total	77	57

* Figures in parentheses indicate percentages of column totals.

Radiologic Findings. Evaluation of the radiologic findings was performed only on patients who had been followed for at least a two-year period (most of them were followed for more than four years) and who had had at least four chest films (most had had in excess of ten). Patients were considered to have improved completely if the preexisting lesions completely regressed. Partial improvement was marked by reduction of the hilar lesion or parenchymal infiltrate. The patient's condition was said to have worsened with accentuation of the original lesions, or to have showed no change when neither the type nor the extent of lesions showed any appreciable change. Serial radiologic examinations for 77 patients in the experimental group and for 55 patients in the control group were evaluated by at least two physicians.

Among the patients on prednisone, 21 (27.3%) showed complete improvement, as compared with 13 (22.8%) of patients on placebo. Partial improvement was noted in 39% of patients in the experimental group and in 35.1% of patients in the control group. These differences were found to be insignificant at the 0.05 level (Table III). The disease got worse in four patients (5.2%) in the prednisone group, as compared with six patients (10.5%) in the control group. No appreciable change was noted in 27.3% and 29.8% of the two groups, respectively.

When the radiologic findings were compared according to the existing pathologic lesions, a similar pattern was noted—a slightly higher but nonsignificant rate of complete improvement among patients on prednisone compared to patients taking placebo. The only exception was in the case of patients with hilar lymphadenopathy: 11 of 25 patients (44%) on prednisone showed partial improvement, as compared with five out of 20 patients (25%) on placebo (Tables IV, V). This difference was found to be significant at the 0.05 level. In the case of patients with parenchymal infiltrates only, the numbers were too small to allow for a meaningful comparison.

Pulmonary Functions. Pulmonary function studies consisted of the dynamic ventilatory function using the Steadwell spirometer and the normal standards of Morris. Diffusion capacity of the lung was determined by the single-breath method using carbon monoxide and the normal values of Cotes.

Table VI shows the percentages of predicted values on initial testing for FVC, FEV_1, MMFR, and DLCO. The experimental and control groups compared relatively well, with a few exceptions.

TABLE IV. Radiologic Findings Following Therapy in Patients with Hilar Adenopathy by Type of Therapy

	Drug*	Placebo*
Complete improvement	7 (28.0%)	5 (25.0%)
Partial improvement	11 (44.0%)	5 (25.0%)
No change	6 (24.0%)	9 (45.0%)
Worsening	1 (4.0%)	1 (5.0%)
Total	25	20

* Figures in parentheses indicate percentages of column totals.

TABLE V. Radiologic Findings Following Therapy in Patients with Hilar Adenopathy and Parenchymal Lesions by Type of Therapy

	Drug*	Placebo*
Complete improvement	13 (28.9%)	8 (26.6%)
Partial improvement	18 (40.0%)	12 (40.0%)
No change	11 (24.4%)	5 (16.7%)
Worsening	3 (6.7%)	5 (16.7%)
Total	45	30

* Figures in parentheses indicate percentages of column totals.

Pulmonary function was assessed only for those patients who took their drug regularly, who underwent frequent testing for most of the tests, and who were followed for at least two years. Post-treatment pulmonary function evaluations were made following completion of therapy in more than 90% of patients. Of the remaining patients, evaluations were made a few weeks later. Patients were classified as unchanged (change within ±10%), improved (change over +10%), or worsened (change in excess of −10%).

Among patients with Stage I disease who were treated with prednisone, there was a slight apparent, but not statistically significant improvement in the FVC, FEV_1, and DLCO, compared to patients who received placebo (Table VII). The same trend was observed among patients with Stage II disease (Table VIII). In the case of patients who presented with parenchymal lesions, namely, Stage III disease, the maximal midexpiratory flow rate improved in six out of ten patients treated with prednisone, as compared with one out of five patients who received placebo.

The diffusing capacity of the lung for carbon monoxide showed a similar trend. Five out of eight patients on prednisone improved, as compared with one out of four patients on placebo. The small numbers, however, do not justify the use of significance tests.

DISCUSSION

The questionable etiology of sarcoidosis, the often encountered difficulties in reaching a definitive diagnosis, the pathologic nature of the disease, and the characteristic remissions make the evaluation of therapeutic regimens a difficult task. Many drugs have been used in the treatment of sarcoidosis, including corticosteroids, chloroquine, methotrexate, oxyphenbutazone, and allopurinal. In the experience of most investigators, these drugs were found to be effective in suppressing the granulomatous reaction and had practically no effect on already established fibrosis.

During the past three decades, corticosteroids have been by far the most widely used therapeutic agents, especially in Stages II and III of the disease. In some instances, only symptomatic patients were treated with corticosteroids. Many of the previous therapeutic trials included only a few patients and did not include controls or utilize a double-blind approach. Compounding these deficiencies were the lack of agreement on the optimum dosage of corticosteroids, differences in the period of therapy or follow-up, and the criteria utilized for diagnosis of sarcoidosis or evaluation of therapeutic effects of corticosteroids.

The present investigation was initiated to evaluate the effects of long-term, adequate prednisone therapy on patients with sarcoidosis, and at the same time to assess some of the findings that have been reported in previous studies. As has been stated previously, all but four of the patients in the experimental and control groups were black. As is known, blacks tend to experience more severe disease than whites.

Evaluation of the therapeutic effect of corticosteroids in

TABLE VI. Percentage of Predicted Vital Capacity and Diffusion for Patients on Initial Testing by Therapeutic Regimen

	Drug				Placebo			
	<65	65–79	80–99	≥100	<65	65–79	80–99	≥100
FVC	16	22	30	20	16	20	21	13
FEV_1	10	30	45	1	3	22	44	1
MMFR	44	14	16	11	38	12	10	9
DLCO	41	18	19	6	35	15	12	3

TABLE VII. Pulmonary Functions in Patients with Hilar Adenopathy

	FVC		FEV_1		MMFR		DLCO		Po_2	
Change	Drug	Placebo	Drug	Placebo	Drug	Placebo	Drug	Placebo	Drug	Placebo
Within ± 10%	13 (42%)	6 (30%)	24 (77%)	17 (85%)	6 (19%)	4 (20%)	14 (50%)	10 (55%)	13 (47%)	11 (69%)
Improved	12 (39%)	6 (30%)	7 (23%)	3 (15%)	18 (58%)	11 (55%)	10 (36%)	5 (28%)	6 (21%)	2 (12%)
Worsened	6 (19%)	8 (40%)	0	0	7 (23%)	5 (25%)	4 (14%)	3 (17%)	9 (32%)	3 (19%)
Total	31	20	31	20	31	20	28	18	28	16

TABLE VIII. Pulmonary Functions in Patients with Hilar and Parenchymal Lesions

	FVC		FEV_1		MMFR		DLCO		Po_2	
Change	Drug	Placebo	Drug	Placebo	Drug	Placebo	Drug	Placebo	Drug	Placebo
Within ± 10%	11 (37%)	8 (40%)	18 (60%)	14 (70%)	7 (23%)	1 (5%)	9 (36%)	6 (37%)	18 (72%)	5 (36%)
Improved	13 (43%)	6 (30%)	4 (13%)	2 (10%)	8 (27%)	12 (60%)	13 (52%)	7 (44%)	6 (24%)	5 (36%)
Worsened	6 (20%)	6 (30%)	8 (27%)	4 (20%)	15 (50%)	7 (35%)	3 (12%)	3 (19%)	1 (4%)	4 (28%)
Total	30	20	30	20	30	20	25	16	25	14

pulmonary sarcoidosis was made both roentgenographically and functionally under controlled double-blind conditions. Marked improvements in chest radiographs were noted among patients on prednisone as well as among those on placebo. Pulmonary function studies did not show a specific trend among either treated patients or controls. Although subjective symptoms were not among the criteria utilized in evaluation, it is our impression that patients on prednisone might have had more symptomatic improvement than patients on placebo.

The study indicates that prolonged therapy for sarcoidosis with adequate doses of prednisone exerted minimal beneficial effects, whether the patients had localized lesions in the mediastinum or presented with pulmonary involvement demonstrated radiologically.

Future therapeutic trials may take advantage of the recent achievements in the assessment of pulmonary disease through studies of bronchoalveolar lavage or angiotension converting enzyme.[9-11] Whether or not these new, sophisticated evaluative tools will show that corticosteroids exert specific beneficial effects on patients with sarcoidosis can only be determined through long-term controlled studies.

REFERENCES

1. Stone DJ, Schwartz A, Feltman JA, et al: Pulmonary function in sarcoidosis: Results with cortisone therapy. *Amer J Med* 1953; 15:468–476.
2. Israel HL, Sones M, Harrell D: Cortisone treatment of sarcoidosis; experience with 36 cases. *JAMA* 1954; 156:461–466.
3. Israel HL, Fouts DW, Beggs RA: A controlled trial of prednisone treatment of sarcoidosis. *Am Rev Respir Dis* 1973; 107:609–614.
4. Colp C, Park SS, Williams MH Jr: Pulmonary function follow-up to 120 patients with sarcoidosis. *Ann NY Acad Sci* 1976; 278:301–307.
5. Johns CJ, MacGregor MI, Zachary JB, et al: Extended experience in the long-term corticosteroid treatment of pulmonary sarcoidosis. *Ann NY Acad Sci* 1976; 278:722–731.
6. DeRemee RA: The present status of treatment of pulmonary sarcoidosis: A house divided. *Chest* 1977; 71:388–393.
7. Harkleroad LE, Young RL, Savage PJ, et al: Pulmonary sarcoidosis. Long-term follow-up of the effects of steroid therapy. *Chest* 1982; 82:84–87.
8. Eule H, Weinecke A, Roth I: The possible influence of corticosteroid therapy on the natural course of pulmonary sarcoidosis. *Ann NY Acad Sci* 1986; 465:695–701.
9. Lieberman J: *Sarcoidosis*. Orlando, Fla, Grune and Stratton, Inc, 1985.
10. Hunninghake GW, Crystal RG: Pulmonary sarcoidosis. A disorder mediated by excess helper T-lymphocytic activity at sites of disease activity. *N Engl J Med* 1981; 305:429–434.
11. Crystal RG, Roberts WC, Hunninghake GW, et al: Pulmonary sarcoidosis: A disease characterized and perpetuated by activated lung T-lymphocytes. *Ann Intern Med* 1981; 94:73–94.

See also p 490.

The discovery of the first drug effective in treating acute lymphoid leukemia in children

CARL POCHEDLY, MD

Today, the bright outlook for children with acute lymphoid leukemia (ALL) is well known. More than 50% of these children appear to be cured by modern therapy.[1] This situation is in stark contrast to the gloomy prognosis for children with ALL a few decades ago.

To appreciate more fully the progress that has been made during the past 40 years in the treatment of leukemia in children, one needs to consider what it was like for a child to have leukemia in the prechemotherapy era, that is, before 1948. At that time, the likelihood of prolonged survival was nil; the available treatment was almost useless. As a standard pediatric textbook of 1933[2] stated, "Acute leukemias run their course uninfluenced by treatment. Blood transfusion may be of benefit, but improvement is very transitory. Most cases succumb in the course of a few weeks . . ." It is not surprising that leukemia was then referred to as "a cataclysmic disaster of childhood."[2] To establish the diagnosis of acute leukemia was to pass a death sentence, since the average survival from the time of diagnosis was only 3–5 months, with many children dying in a few weeks.[3-5] The best that could be offered was limited palliation and sympathetic terminal care. The same outlook applied to nearly all other cancers as well.

It is not surprising that in the 1940s, the attitude of physicians toward the treatment of cancer was one of extreme pessimism. It was generally believed that the administration of drugs other than to relieve pain was quackery. Radiation therapy, which was widely used in both the leukemias and other forms of cancer, gave temporary relief of certain symptoms, but offered little chance of cure or significant prolongation of life.

In this gloomy setting, the announcement in 1948 of the first drug effective in the treatment of acute leukemia in children created a sensation. However, the important studies that led up to this breakthrough had been carried out during the preceding 30 years.

"YELLOW CROSS," SEPSIS, AND LEUKOPENIA

The military use of mustard gas, so-called "yellow cross," was one of the barbaric innovations introduced during World War I. Gas warfare was first used by the Germans in Belgium, in the fields of Ypres. But the other side also made use of this weapon, with devastating results. The gas used in chemical warfare was sulfur mustard or dichlorethyl sulfide. Exposure to sulfur mustard caused severe burns to the skin as well as to the membranes lining the respiratory tract. Among those victims who did not die immediately of shock and pulmonary edema, but who died several days or weeks later of pneumonia and generalized septicemia, it was noted that the most frequent cause of death was overwhelming infection. But accompanying sepsis in these dying soldiers was a low white blood cell count, instead of the expected leukocytosis that usually accompanies any severe infection. In addition, postmortem examinations of victims of mustard gas showed atrophy of the lymph nodes and spleen which, if anything, should have been enlarged in people who died of overwhelming infections.[6,7]

These astute observations of the blood counts, lymph nodes, and spleens of soldiers dying after exposure to mustard gas are very impressive. They were made by extremely busy physicians working amid the agony and chaos of the battlefield. Shells were exploding and soldiers were dying all around them, yet these battle-weary physicians remained alert enough to notice some peculiar blood counts, a finding then of seemingly little importance.

The possible clinical application of these findings to the treatment of diseases of the blood and lymph nodes was not recognized until the completion of research done early in World War II. These studies were part of the secret program of chemical warfare research carried out under the sponsorship of the United States Army. Pharmacologic studies of the toxic effects of sulfur mustard in experimental animals were undertaken in 1941. The aims of these studies were to determine the mode of action of sulfur mustard, to study the systemic effects of the mustards, and to look for possible antidotes to the toxic action of the mustards. It was soon found that the general or systemic effects of the mustards were far more interesting than the blisters they produced on the skin.[8]

An analog of sulfur mustard, nitrogen mustard, was found to be more convenient for study in animals, because it could be given by intravenous injection. When injected into rabbits in small doses, nitrogen mustard caused nau-

---

From the Department of Pediatrics, Health Science Center, University of Texas, San Antonio, TX.

Address correspondence to Dr Pochedly, Santa Rosa Children's Hospital, 519 W Houston St, San Antonio, TX 78285.

sea and vomiting. When injected in large doses, it caused destruction of the bone marrow and a severe decrease in the white blood cell counts, similar to the effect observed in soldiers following exposure to sulfur mustard gas. This observation was the clue to the possible usefulness of nitrogen mustard in the treatment of diseases of the blood, lymph nodes, and spleen.

The idea of using nitrogen mustard in the treatment of lymphoid cancers and leukemia was recognized as early as 1942. First, nitrogen mustard was given to mice in whom lymphoid tumors had been artificially implanted. Researchers noted dramatic regression of the lymphoid tumors. These impressive studies in mice quickly led to clinical trials of nitrogen mustard against lymphomas and leukemias in human patients. Since these studies were done under military secrecy, the first report of a clinical trial of nitrogen mustard in the treatment of human diseases was not published until after the war, in 1946.[9] Nitrogen mustard was found to be most effective in causing dramatic but temporary improvement in patients with Hodgkin disease and lymphosarcoma.

As predicted from the laboratory experiments, nitrogen mustard caused profound depression of the leukocyte count in human patients. But, surprisingly, the most beneficial clinical effect occurred in cancers of the lymph nodes and spleen, rather than in the leukemias. Many patients with chronic leukemia improved while receiving nitrogen mustard, but patients with acute leukemia were entirely resistant to treatment with this drug.[10]

In spite of this success, the vast majority of early efforts in cancer chemotherapy were fruitless; this led, inevitably, to disillusionment and scorn for chemotherapy. The implication was that attempts at chemotherapy of cancer were outside the mainstream of medicine and were at best sincere but misguided efforts.[11] The subsequent discovery in 1948 of drugs effective in the treatment of acute leukemia did much to alleviate this scornful attitude toward chemotherapy, and helped remove the disdainful regard of those physicians who persisted in using drugs for treatment of cancer.

TRIAL OF ANTIFOLATES

Before 1948, there was no known way to destroy leukemic cells and thus produce a remission of any significant duration. In that year, however, a quantum leap in therapy was achieved. Led by Sidney Farber (Fig 1), a group of investigators at the Boston Children's Hospital tested a drug called aminopterin, and found that it could induce a complete remission in about one third (ten out of 16) of children with acute leukemia.[12]

However, Farber's clinical trials were impeded by the stigma still clinging to any use of drugs in the treatment of cancer. When the trials were begun, the pediatric residents at Boston Children's Hospital were strongly opposed to the studies. They believed the treatments were futile and only caused increased suffering among the children. Only through strenuous persuasion by Chief of Pediatrics Dr Charles Janeway was it possible to calm the fears of the young staff and to persuade them to put aside their doubts about the scientific merits of the trial of aminopterin. Of course, these objections disappeared completely when the first apparent "cures" were observed (R. Mercer, personal communication, 1985).

FIGURE 1. Dr Sidney Farber (photograph taken at about the time of the epochal trials of aminopterin).

Farber's Golden Hunch. What was the rational basis for trying aminopterin in the treatment of leukemia? Allegedly, aminopterin was tried because it was known to antagonize or neutralize the effects of a vitamin, folic acid. But why did Farber think that giving this antifolic acid drug would help children with leukemia?

Folic acid was known to be important for the formation of blood cells. Lack of folic acid in the diet of experimental animals causes anemia and a decrease in the number of white blood cells. Also, observations in leukemic mice seemed to show that folic acid might actually stimulate the growth of leukemic cells.[13] In attempting to confirm these observations, Farber gave large doses of folic acid to some children with leukemia who were terminally ill. He felt that the course of leukemia in these children was accelerated.

Thus, giving large doses of folic acid to children with leukemia appeared to increase the rate of leukemic cell growth.[14] Doing this, Farber thought, made the leukemia worse and caused the children to die more quickly. However, this alleged leukemia-accelerating effect of folic acid was vague and unconvincing. It was only a hunch, based more on intuition than on tangible facts. But it turned out to be a golden hunch. Nonetheless, Farber never published his preliminary observations; in fact, he did not even mention them in the articles in which he reported his trial of aminopterin in the treatment of leukemia. His idea could not be supported by any experimental studies. Today, as research on humans is scrutinized more closely, it is doubtful that a modern hospital research committee, institutional review board, or human research panel would give Farber permission to try antifolate drugs in children with leukemia.

The Trial of Antifolates. Meanwhile, at Lederle Laboratories, Dr Yoshida Subbarow was studying various folic acid compounds. Dr Farber asked Dr Subbarow to synthesize some compounds that would antagonize the action of folic acid. Farber's concept was that, if leukemia is accentuated by folic acid, then the disease might be inhibited by giving compounds antagonistic to folic acid. Although the validity of this concept was never proven, it stimulated Farber to try a number of folic acid antagonists for treatment of children with leukemia. Early in 1947, diopterin was tried; it produced results suggesting a slight antileukemic effect. Then, late in 1947, another folic acid antagonist, aminopterin, was used. This drug turned out to be a winner in the treatment of childhood acute leukemia. It induced remissions of six weeks to five months in ten out of 16 children. The report of Farber and his colleagues, published as the lead article in the *New England Journal of Medicine*, June 3, 1948, proved to herald a new era. Later, aminopterin was replaced by another antifolate, methotrexate, which was less toxic and easier to control.

Methotrexate exerts its cytotoxic effect by competing with folate derivatives for binding sites on the enzyme dehydrofolate reductase. Methotrexate blocks production of tetrahydroxofolate and causes impaired DNA synthesis and death of rapidly growing cells. It is ironic that the proposed basis for the trial of antifolate drugs in the treatment of children with acute leukemia was false, but the results of the trial itself proved to be a monumental step forward for the field of cancer chemotherapy. Thus, one must conclude that the studies that led to the first effective antileukemic drug were based on the investigator's intuition, rather than a rational hypothesis. Many critical experiments that led to important scientific discoveries were motivated by a personal bias or whim of the investigator, as in this case.

In 1948, Farber's study resulted in publication of an article that reported the occurrence of temporary remissions in one third of children with acute leukemia following treatment with aminopterin.[12] This study is now regarded as a landmark both in childhood leukemia and in cancer chemotherapy in general.

When the first patients with leukemia went into remission following treatment with aminopterin, the investigators had a few ecstatic moments, believing they had found the "magic bullet" that would specifically destroy leukemic cells and be the absolute cure of leukemia. This hope was soon dashed as the "cured" patients began to relapse.

Additionally, the frequent infections that occurred in these patients were difficult to manage, because at the time the only antimicrobial drugs available were the sulfonamides and limited quantities of penicillin. Streptoymcin was restricted to use among children with tuberculosis. Thus, in the late 1940s, treatment of infections associated with leukemia was ineffective. This decreased the number of children who could achieve remission with aminopterin, since many died of infection soon after chemotherapy was started. When children developed high fevers, despair also developed. Nothing could be done for patients who were septic. "When leukemic children got high fevers, we didn't do much about it. There wasn't much to do. When they got septic, they died" (J. A. Wolff, personal communication, 1987).

These observations emphasize the important role that antibiotics still play today in the successful treatment of cancer. Patients given chemotherapy become highly susceptible to serious infections. Without antibiotics to combat these infections, children with leukemia frequently die before they can benefit from the chemotherapy.

Following the development of a successful treatment for leukemia, Farber was able to establish a small tumor clinic at the Boston Children's Hospital. It consisted of an entrance hall in the pathology department plus a room leading to the men's toilet. Soon afterwards, Dr Farber obtained money to establish the Jimmy Fund, named in honor of a child who died of leukemia. This enabled Farber to establish a separate ward for children being treated for cancer. The Jimmy Fund ward grew into the establishment of the Jimmy Fund Building, which evolved into the present-day, world famous Dana-Farber Cancer Institute. Today, this institute stands as a symbol of the monumental scientific discovery that was made by Farber and his colleagues in 1948.

AFTERMATH AND FOLLOW-UP

Farber demonstrated for the first time that it was possible to produce temporary remissions in a significant proportion of children with acute leukemia. His findings, which were rapidly confirmed, created a sensation both in the scientific community and among the lay public. The cover of *Time* magazine of July 11, 1948, was emblazoned with a photograph of Dr Farber and carried the bold caption, "Leukemia is cured." The disappearance of leukemic cells from the blood and bone marrow and the clinical improvement of these children was so dramatic that it appeared at first as if a cure had been produced, and that the long sought anticancer "magic bullet" had been found. But, unfortunately, relapses of leukemia ensued.

These findings provided a powerful stimulus for research on the development of new anticancer drugs. The effort grew enormously, as did the subsequent expenditure of money. The ultimate result was a revolutionary improvement in the outlook for children with acute leukemia and the establishment of cancer chemotherapy as a legitimate area for serious medical research.

Although the lay press extolled the finding as the cure for leukemia, colleagues in cancer therapy were skeptical. In fact, when Farber presented his first report in May 1948, most of his colleagues were so incredulous they were not even prepared to discuss it with him. When the results of Farber's studies were confirmed, however, physicians everywhere were eager to learn more. In order to accommodate this clamor for information, Farber held large seminars, to which people came from all over the country and from abroad to learn about the new treatment.

At that time, cytologic differences between the acute myeloid leukemias and the acute lymphoid leukemias were not considered in determining the treatment to be given (J. A. Wolff, personal communication, 1987). All patients with acute leukemia were treated the same. Presumably, a larger proportion of Farber's patients would have entered remission following aminopterin therapy if only children with acute lymphoid leukemia were treated. It was observed that aminopterin did not work nearly as well in adults, in whom there is a large preponderance of myeloid leukemias, as it did in children. Thus, many hematologists who treated only adults with leukemia were skeptical of Farber's claims. Prior to about 1960, all cases of acute leukemia were referred to as stem cell leukemia as far as treatment was concerned. Thereafter, the differences in response to chemotherapy of lymphoid and myeloid leukemias were noted, and differing treatments were tried.

FURTHER IMPROVEMENTS IN CHEMOTHERAPY

The demonstration by Farber and colleagues of the powers of aminopterin, and its analog methotrexate, was followed in a few years by reports of remissions induced with adrenocorticotropic hormone (ACTH) and the corticosteroids.[14] In 1953, Burchenal et al demonstrated 6-mercaptopurine to be clinically active.[15] With use of these three drugs, it became possible to palliate the disease in a high percentage of patients and to increase the median survival time to about 12 months.[16,17]

There followed many more useful drugs, and the new science of cancer chemotherapy evolved rapidly. The new drugs most useful for treatment of acute leukemia were vincristine, daunorubicin, and 1-asparaginase. After several useful antileukemic drugs were found, physicans had to learn the best way of giving the drugs: the dosage, frequency, and combinations of several drugs that would produce the best results. This took many years of trial and error on large numbers of children with leukemia.[14-17]

In the early 1970s, it was noted that although children with leukemia could readily be put into remission, before too long leukemia would appear in the brain, followed by a general relapse of the disease. The explanation for this was that antileukemic drugs, when given by mouth or by intravenous injection, do not penetrate into the brain and spinal cord, and so cannot destroy leukemic cells sequestered in the brain. The leukemic cells hiding there later grow and multiply, and cause relapse of leukemia. It was shown by Pinkel and colleagues[1] that one must treat the brain and spinal cord directly by radiographic therapy and by intrathecal injection of drugs. This treatment is given when the general leukemia therapy program is started, to prevent the later development of leukemia in the brain. It greatly increased the number of children with acute lymphoid leukemia who survived for five years or longer.

Now, in 1987, one can say that the most common type of leukemia in children, acute lymphoid leukemia, is potentially curable in the majority of cases. This means that out of every 100 children who get acute lymphoid leukemia, more than 50 are still alive, and healthy, with no evidence of this disease five years later.

There is vastly more hope today than there was 40 years ago. And there is every reason to believe that the favorable influence of chemotherapeutic treatment of leukemia will continue to increase in the years ahead.

Acknowledgments. Drs James Wolff and Robert Mercer were fellows at the Boston Children's Hospital in the late 1940s, during the time that the trials of aminopterin were done. Material obtained from the author's conversations with Drs Wolff and Mercer is included in this article. In addition, Drs Wolff and Mercer read the manuscript and offered comments and suggestions.

REFERENCES

1. Pinkel D, Simone J, Hustu HO, et al: Nine years' experience with "total therapy" of childhood acute lymphocytic leukemia. *Pediatrics* 1972; 50:246-251.
2. Holt LE, McIntosh R: *Diseases of Infancy and Childhood.* New York, Appleton-Century Co, 1933, p 564.
3. Tivey H: Prognosis for survival in leukemias of childhood: Review of literature and proposal of simple method of reporting survival data for these diseases. *Pediatrics* 1952; 10:48-51.
4. Bierman HR, Cohen P, McClelland JN, et al: The effect of transfusions and antibodies upon the duration of life in children with lymphogenous leukemia. *J Pediatr* 1950; 37:455-461.
5. Diamond LK, Luhby AL: Pattern of "spontaneous" remissions in leukemia of childhood observed in 26 of 200 cases. *Am J Med* 1963; 10:238-241.
6. Krumbhaar E, Krumbhaar H: The blood and bone marrow in yellow cross gas (mustard gas) poisoning. *J Med Res* 1919; 40:497-506.
7. Alexander SF: Medical report of the Bari Harbor mustard casualties. *Milit Surg* 1947: 101:1-17.
8. Gilman A: The initial clinical trial of nitrogen mustard. *Am J Surg* 1963; 105:574-578.
9. Goodman LS, Wintrobe MM, Dameshek W, et al: Nitrogen mustard therapy. *JAMA* 1946; 132:126-132.
10. Gunz FW: The dread leukemias and the lymphomas: Their nature and their prospects, in Wintrobe MM (ed): *Blood, Pure and Eloquent.* New York, McGraw-Hill Book Co, 1980, pp 511-545.
11. Krakoff IH: Cancer chemotherapy, in Horton J, Hill GJ (eds): *Clinical Oncology.* Philadelphia, WB Saunders Co, 1977, pp 157-181.
12. Farber S, Diamond LK, Mercer RD, et al: Temporary remissions in acute leukemia in children prolonged by folic acid antagonist, 4-aminopteroylglutamic acid (aminopterin). *N Engl J Med* 1948; 238:787-793.
13. Heinle RW, Welch AD: Experiments with pteroylglutamic acid and pteroylglutamic acid deficiency in human leukemia. *J Clin Invest* 1948; 27:539-540.
14. Pearson OH, Eliel LP, Rawson RW: Regression of lymphoid tumors in man induced by ACTH and cortisone, in Mote JR (ed): *Proceedings of the First Clinical ACTH Conference*, Philadelphia, Blakiston, 1950.
15. Burchenal JH, Murphy ML, Ellison RR, et al: Clinical evaluation of a new antimetabolite, 6-mercaptopurine, in the treatment of leukemia and allied diseases. *Blood* 1953; 8:965-969.
16. Burchenal JH: Hematologic neoplasms, in Holland J, Frei E (eds): *Cancer Medicine.* Philadelphia, Lea and Febiger, 1973, pp 1167-1173.
17. Zubrod CG: Historic milestones in curative chemotherapy. *Semin Oncol* 1979; 6:490-505.

Physician framers of the Constitution

FLORENCE KAVALER, MD, MPH; DAVID METZ, MPA; ANN B. COHEN, MSL

The men who were the architects of the founding principles of the United States included farmers, lawyers, ministers, and physicians. As we celebrate the bicentennial of the United States Constitution, it seems an appropriate time to reflect on the lives and contributions of the three physicians who were active participants in the formation of our nation. They were Hugh Williamson of North Carolina, James McHenry of Maryland, and James McClurg of Virginia. The three were among the 55 delegates representing their states at the constitutional convention, and Williamson and McHenry were among the 39 signers of the Constitution in Philadelphia on September 17, 1787. They were all highly educated, multitalented men whose patriotism led them to devote years of their lives to the service of their emerging nation. Williamson and McClurg were able to combine their medical careers with their other interests, but McHenry chose to leave medicine early in his career in order to pursue a life in politics.

HUGH WILLIAMSON (1735–1819)

Hugh Williamson (Fig 1) could almost match his close friend Benjamin Franklin in range of interests and talents. Educated as a physician, he was also trained (but not ordained) as a minister, became a successful merchant, taught as a professor of mathematics, and won recognition for his scientific work on climate. Born in West Nottingham, Pennsylvania, he was a member of the first class of the College of Philadelphia, which later became part of the University of Pennsylvania. After receiving his degree in 1757, he administered his father's estate and studied for the Presbyterian ministry. Two years after his graduation, he returned to the College of Philadelphia as a mathematics professor.[1(p298)]

Disillusioned by factional disputes among Presbyterians and tired of teaching after a few years, Williamson moved to Europe and studied medicine at Edinburgh, London, and Utrecht, Holland. He received his medical degree from the University of Utrecht in 1764 and returned to Philadelphia to practice. However, being physically and emotionally frail, he found medical practice too taxing. Whenever a patient was in danger, Williamson developed a high fever. Not surprisingly, he was soon spending most of his time working in his family's business and pursuing interests in science and mathematics.[1]

In 1768, Williamson was elected to the American Philosophical Society and the next year was appointed to a commission that observed the transits of Venus and Mercury. He presented a number of original ideas in "An Essay on Comets" in 1771, and as a result was awarded an honorary degree from the University of Leyden.[2(p208)]

The well-traveled Williamson journeyed to the West Indies and to Europe in 1773, primarily to raise money for an academy in Delaware. While waiting for his ship to sail from Boston, he witnessed the Boston Tea Party, and brought the first news of the event to England. There the British Privy Council called on him to testify about what he had observed, and he used the opportunity to warn the councilors that the colonies would rebel unless the British

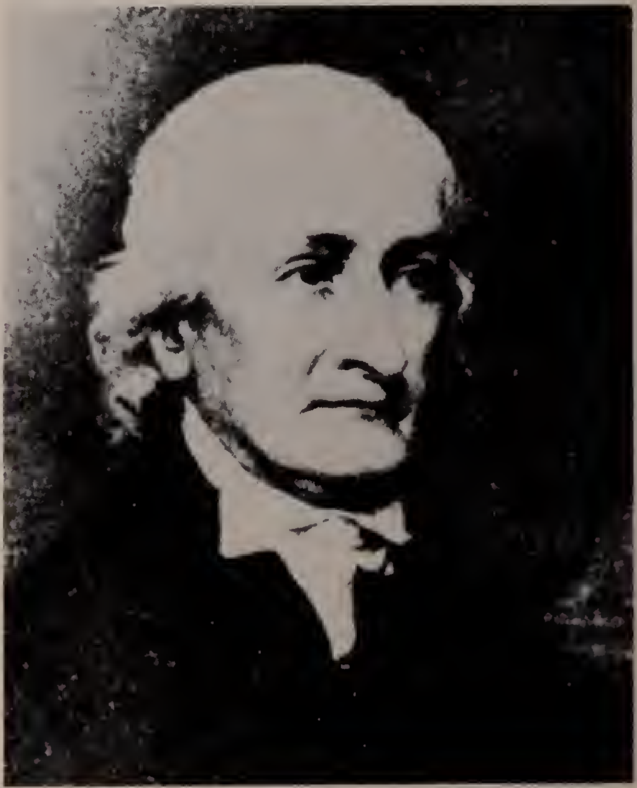


FIGURE 1. Hugh Williamson, MD (1735–1819).

Dr Kavaler is Professor of Preventive Medicine and Community Health, SUNY Health Science Center at Brooklyn, Brooklyn, NY; Mr Metz is President of Capital Care (subsidiary of Blue Cross/Blue Shield of National Capital Area), Arlington, Va; and Ms Cohen is a research associate, Staten Island, NY.

Address correspondence to Dr Kavaler, Department of Preventive Medicine and Community Health, SUNY Health Science Center at Brooklyn, Box 43, 450 Clarkson Ave, Brooklyn, NY 11203-2098.

changed their policies. While in England, he met Benjamin Franklin, and the two developed a close friendship while collaborating on electrical experiments. Williamson also passed along to Franklin a copy of a letter from Massachusetts Royal Governor Thomas Hutchinson to his lieutenant governor, in which Hutchinson urged "abridgement of . . . English liberties" in the colonies. The Hutchinson-Oliver letters deepened the divisions between the British and the colonists and created a sensation back in America.[2(p209)]

Williamson's increasing commitment to the American colonists' cause was further evident in a pamphlet he wrote (anonymously) in England in 1775 called *The Plea of the Colonies*, which solicited the support of English Whigs for the Americans. When the colonists declared their independence the next year, Williamson quickly sailed home from the Netherlands.[1(p299)] He lived for a short time in Charleston, SC, before settling in Edenton, NC, where he started a mercantile business trading with the French West Indies. He also returned to the practice of medicine and applied for a medical post with the colonial forces, but all positions were filled. After Williamson helped inoculate soldiers against smallpox, however, the governor of North Carolina named him surgeon-general of the state's troops. Following the Battle of Camden, South Carolina, Williamson frequently crossed British lines to tend to wounded American prisoners and British troops as well.[2(p209)]

Williamson is also credited with instituting health and sanitation practices which dramatically lowered the rates of disease and noncombat deaths among colonial forces. He advocated inoculation as a prerequisite for military service, and experimented with hygiene, clothing, diet, shelter, and drainage methods which resulted in significantly reduced rates of illness. Under Williamson's supervision, only two men, out of a force numbering as many as 1,200, died in a six-month period, an unparalleled achievement for the times.[1(p299)]

After the war, Williamson's political career began with his election in 1782 to the House of Commons in North Carolina and to the Continental Congress, where he served until 1785 and then served again in 1787. In 1786, he wrote *Letters of Sylvius*, published anonymously, to demonstrate the evils of paper money and to advocate an excise rather than a land or poll tax.[1(p299)] He was chosen as a delegate to the constitutional convention, where he served on five committees and played a significant part in the proceedings, particularly the major compromise on representation. The most active member of the North Carolina delegation, he attended sessions faithfully and spoke frequently.[2(p209)]

Thomas Jefferson described Williamson as "a very useful member [of the Congress], of an acute mind, attentive to business, and of an high degree of erudition." [1(p299)] He was considered pleasant and genial and was widely popular, though he could be intolerant of those he considered irreligious and was capable of rudeness toward those he disliked. Major William Pierce, a Georgia delegate to the convention, wrote short sketches describing the other representatives. Pierce said Williamson "is a Gentleman of education and talents. He enters freely into public debate from his close attention to most subjects, but he is no Orator. There is a great degree of good humor and pleasantry in his character; and in his manners there is a strong trait of the Gentleman." [3(p44)] However, the French Minister, Otto, who reported developments from the convention to Foreign Minister Montmorin in Paris, had a harsher assessment of Williamson's contributions: "Physician and former professor of astronomy. Excessively bizarre, loving to hold forth, but speaking with spirit. It is difficult to know his character well; it is even possible that he does not have any; but his activity has given him much influence in Congress for some time." [3(p50)]

On July 18, 1787, Williamson wrote North Carolina lawyer (and later US Supreme Court Justice) James Iredell that, "I think it is more than likely that we shall not leave this place [Philadelphia] before the middle of August. The diverse and almost opposite interests that are to be reconciled occasion us to progress very slowly." [4(v3,p55)] Madison reported that Williamson "did not think a better plan was to be expected and had no scruples against putting his name to it [the Constitution]" on September 17, 1787.[4(v2,p645)]

"It seems to be generally admitted that the system of government which has been proposed by the late convention is well calculated to relieve us from many of the grievances under which we have been laboring," Williamson said in 1788, when he campaigned for ratification of the Constitution at home in North Carolina. "If I might express my particular sentiments on this subject, I should describe it as more free and more perfect than any form of government that has ever been adopted by any nation; but I would not say it has no faults . . . When you refer the proposed system to the particular circumstances of North Carolina, and consider how she is to be affected by this plan, you must find the utmost reason to rejoice in the prospect of better times," Williamson said.[4(v3,p238)] After the convention, Williamson was chosen to settle outstanding accounts between the state and the federal government, and the next year he was elected to the first US House of Representatives, where he served two terms.

Williamson married Maria Apthorpe in 1789, and they had two sons. Then, in 1793, the family moved to New York City to better accommodate Williamson's wide-ranging interests.[2(p209)] Over the years, he published many political, educational, economic, historical, and scientific works, but he received the most recognition for his scientific papers, and in particular his essays on climate, including "Observations on the Climate in Different Parts of America," in 1811.[1(p300)]

Williamson was an original trustee of the University of North Carolina and later held trusteeships at the College of Physicians and Surgeons and the University of the State of New York. He was also a founder of the Literary and Philosophical Society of New York and a prominent member of the New York Historical Society.[1(p300)]

Williamson died in New York in 1819 at the age of 83 and was buried at Trinity Church.[2(p210)]

JAMES McHENRY (1755–1845)

Irish-born James McHenry (Fig 2) immigrated to Philadelphia in 1771, and studied medicine there for two years under the famous Dr Benjamin Rush, who was considered the most influential (and controversial) surgeon of the time.[2(p171)] McHenry, who was extremely hostile to England, volunteered for military service in 1775. He served as a military surgeon for the Fifth Pennsylvania Battalion during the war, and was captured by the British in 1776 at Fort Washington, NY. He was released in a prisoner exchange in March 1778, and was immediately assigned to Valley Forge, where he became secretary to General George Washington.[1(p62)] His association with Washington marked the end of his medical career, and he spent the rest of his life in politics and administration.

In 1780, McHenry joined the staff of the Marquis de Lafayette and the next year entered the Maryland senate. (McHenry's ties to Maryland were through his father and brother, who had established a profitable importing business in Baltimore. McHenry inherited the concern in 1790.) His senate service lasted from 1781 to 1786, and from 1783 to 1786 he also served in the Continental Congress.[2(p171)] In 1784, he married Margaret Allison Caldwell. They had three children.[2(p172)]

McHenry did not play a major role in the Philadelphia convention, missing many sessions because of the illness of his brother, and he made no substantial contributions to the debate. However, he did keep detailed notes of the proceedings of the conventions, which are considered an important part of the historical record.[5(v3,p1238)] Historian Max Farrand said that after his brother's illness, McHenry "returned to the Convention and to his note-taking with all the enthusiasm of the beginner. The records became more and more brief as time passed, but they are valuable because they are, for the latter part of the Convention's work, almost the only material we have besides the Journal [compiled by John Quincy Adams] and Madison's Debates." [4(v1,pxx)]

FIGURE 2. James McHenry, MD (1755–1845).

In his notes on a conference of the Maryland delegates on August 6, McHenry wrote that "finding that we could come to no conclusions, I recommended meeting again tomorrow, for unless we could appear in the convention with some degree of unanimity it would be unnecessary to remain in it, sacrificing time and money without being able to render any service." [4(v2,p191)]

Delegate Pierce of Georgia described McHenry as "a man of specious talents, with nothing of genious [sic] to improve them. As a politician there is nothing remarkable in him, nor has he any of the graces of the Orator. He is however, a very respectable young Gentleman, and deserves the honor which his Country has bestowed on him." [3(p40)] McHenry apparently realized his own limitations as well. Describing his decision to sign the Constitution, he wrote on September 17:

> Being opposed to many parts of the system, I make a remark why I signed it and mean to support it.
>
> Firstly, I distrust my own judgment, especially as it is opposite to the opinion of a majority of gentlemen whose abilities and patriotism are of the first cast; and as I have had already frequent occasions to be convinced that I have not always judged right.
>
> Secondly, Alterations may be obtained, it being provided that the concurrence of ⅔ of the Congress may at any time introduce them.
>
> Thirdly, Comparing the inconveniences and the evils which we labor under and may experience from the present confederation, and the little good we can expect from it—with the possible evils and probable benefits and advantages promised by the new system, I am clear that I ought to give it all the support in my power." [4(v2,p649)]

McHenry continued his political work after the conventions, campaigning for ratification of the Constitution in Maryland. Then, because of his close association with General Washington, he was named to the Maryland commission which formally welcomed the general on his journey to New York for his inauguration in April 1789.[1(p62)] Six years later, Washington named McHenry Secretary of War, succeeding Timothy Pickering, who had become Secretary of State. Washington held McHenry in high regard, and McHenry wielded considerable influence in the Cabinet and in the distribution of patronage. He retained his position as Secretary of War into the administration of John Adams, who succeeded Washington. However, McHenry, a staunch Federalist, was politically aligned with Alexander Hamilton, who opposed Adam's policies regarding the threat of war with France. Adams came to believe that McHenry, Hamilton, and others were attempting to undermine his political base and destroy his chances for reelection, and in May 1800, Adams demanded McHenry's resignation from his Cabinet.[1(p63)]

Even after his resignation, McHenry faced criticism from congressional Democratic-Republicans for mismanagement of the war department, but a legislative committee vindicated him. His detailed defense of his actions was read from the House floor, and after the inquiry was dropped he lived in retirement on an estate outside of Baltimore.[1(p63)] Still an avid Federalist, he opposed the War of 1812,[2(p172)] even though his son John volunteered to defend Fort McHenry (named after the elder McHenry while he was Secretary of War). But McHenry had no further involvement in politics, and was taken up with few activities other than a Bible society in Baltimore.[1(p63)] He died in 1816 at the age of 62 and was buried in Baltimore's Westminster Presbyterian Cemetery.[2(p172)]

JAMES McCLURG (1746–1823)

James McClurg (Fig 3) was born near Hampton, Virginia, and graduated from the College of William and Mary in Williamsburg,[2(p170)] where Thomas Jefferson was also a student. McClurg acquired a reputation there as a gifted scholar, particularly in classical disciplines.[6(p328)] At 17, his parents sent him on a trip to Europe with his sister. They hoped the young man's feeble constitution would be bolstered by the sea voyage, and believed exposure to foreign culture would broaden his education.[6(p328)] Soon after his arrival in Europe, he decided to pursue a career in medicine, following the path of his father, Dr Walter McClurg, who was superintendent of the Hampton Small Pox Hospital, one of the first hospitals of its kind in America.[1(p596)]

McClurg enrolled in a medical course at the University of Edinburgh, where his scholarly achievements continued to bring him recognition in the academic and scientific communities. He graduated in 1770 after completing a thesis entitled "De Calore," which was regarded as an important and original work, then did postgraduate work in medicine in Paris and London.[6(p329)] While living in London in 1772, he wrote his celebrated essay, "Experiments upon the Human Bile and Reflections on the Biliary Secretions." Blanton wrote that McClurg began his paper with an essay on medical reasoning, in which he asserted that medical science had always been subject to change, and that progress had resulted from the traditional arguments between dogmatists and empiricists. McClurg then proceeded to outline his own new theory, based on experiments illustrating the effect of heat and acid upon the bile, and in turn, of the bile upon muscle and fat.[6(p329)] McClurg concluded that bile was formed from blood in the liver, blood which was advancing towards putrefaction; that its functions were antiputrefactive, antifermentative, and neutralizing to gastric acidity. He also noted a sweet taste in bile which he believed was due to a sugar similar to milk sugar.

FIGURE 3. James McClurg, MD (1746–1823).

Blanton continued:

> From this point on theory ran away with fact. He argued that if bile is formed from putrefactive blood and if heat putrefies blood, hot countries should be conducive to liver disease and that malaria, marshes, badly ventilated ships and rooms, which hasten degeneration of the blood, should also be conducive to liver disease. The treatment of patients subject to such unfortunate hematogenous disturbances was a natural corollary of this theory. He recommended the removal of the patient from an environment that would promote putrefactive changes in the blood, free purgation to assist nature in throwing off "this enlarged secretion" of bile, the wearing around the body of camphor and other antiseptics to ward off the noxious influence of the atmosphere, and finally the use of quinine because of its tonic effect.

McClurg's theory was well-regarded in international scientific and medical circles, and Blanton observed that

> . . . it is not difficult to understand how such a theory at such a time carried the day. Couched in fine English, ornamented by frequent classical quotations and conveying all the persuasion of a syllogistic argument, it was irresistible. Looking upon it with modern eyes one wishes that the experimental method which had been used so successfully by Harvey had not been thus prostituted by a theorist who thought more of proving his point than of establishing facts beyond peradventure of a doubt.

McClurg returned to Virginia in 1773, and established a successful medical practice in Williamsburg. He joined the Revolutionary Army and wrote his friend Thomas Jefferson, requesting an appointment as physician for the Continental troops stationed in Virginia:

> It is believed here, that a Physician will be appointed to the Continental Troops in this colony; an office I desire exceedingly, as it wd. gratify at the same time my passion for improvement in the profession I am destined to, & my zeal to do my country some service. In this time of general activity, I do not like to be an idle spectator; & I know not any post wch. wd. suit me so well.

McClurg did not get the position, but did serve as a surgeon in the Virginia state militia during the war. When the war ended, he was named superintendent and inspector of hospitals in Virginia.[6(pp329-332)]

In 1779, McClurg married Elizabeth Selden,[2(p170)] and that same year was named professor of anatomy and medicine when the chair was first created at the College of William and Mary. (The only other chair in the field in America at the time had been established in Philadelphia.) McClurg was appointed after Jefferson became a member of the school's Board of Visitors, and he served with faculty members who included James Madison, who was president of the college and professor of natural philosophy and mathematics.

McClurg was one of the most eminent physicians in Virginia, despite the fact that he did not conduct his practice in the typical style of the times. He did not use surgical instruments, nor did he own an apothecary shop, although most of his professional colleagues did both. In commenting on this McClurg wrote:

> With respect to myself, I can assure you that the profits of my profession do not support my family, and that I must, if I had no other dependence, live very indifferently. This however is partly owing to my not uniting the apothecary's and the surgeon's business with the physician's as is common in this country; and to my not being adept in another necessary qualification of a doctor, viz., dunning.[6(pp330-332)]

While McClurg was well known and widely respected as a physician, he also established a reputation in politics. James Madison unsuccessfully lobbied for the appointment that McClurg received as US Secretary of Foreign Affairs in 1782. In 1787, McClurg agreed to join the Virginia delegation to the constitutional convention, after Richard Henry Lee and Patrick Henry declined the position.[2(p170)] At the convention, he advocated life tenure for the president, a proposal which received the votes of four of the ten states then represented. He also argued for the power of the federal government to override state laws, and was a strong supporter of greater independence of the executive from the legislative branch.[1(p597)]

Madison, in his notes from the convention on July 20, 1787, wrote that McClurg "asked whether it would not be necessary, before a committee for detailing the Constitution should be appointed, to determine on the means by which the Executive is to carry the laws into effect, and to resist combinations against them. Is he to have a military force for the purpose, or to have the command of the Militia, the only existing force that can be applied to that use? As the Resolutions now stand, the committee will have no determinate directions on that great point." [4(v2,p69)]

In his brief sketch of McClurg, Pierce of Georgia said McClurg was "a learned physician, but having never appeared before in public life his character as a politician is not sufficiently known. He attempted once or twice to speak, but with no great success. It is certain that he has a foundation of learning, on which, if he pleases, he may erect a character of high renown." [3(p40)]

McClurg left the convention in early August to return to his wife, to whom he was extremely devoted. Rumors at the time were that his devotion to his spouse bordered on the obsessive, and when he originally left home for Philadelphia, friends speculated that he would not be able to endure a separation from his wife for long.[7] Madison wrote McClurg, urging his return to Philadelphia, where the Virginia delegation was becoming increasingly factionalized, but was unable to persuade him to come back. McClurg responded on August 5, 1787:

> I am much obliged to you for your communication of the proceedings of ye Convention since I left them, for I feel that anxiety about ye result, which its Importance must give to every honest citizen. If I thought that my return could contribute in the smallest degree to its Improvement, nothing should keep me away. But as I know that the talents, knowledge and well-establish'd character of our present delegates have justly inspired this country with ye most entire confidence in their determinations; and that my vote could only operate to produce a division and so destroy ye vote of ye State, I think that my attendance now would be certainly useless, perhaps injurious.[4(v3,p67)]

Even after leaving the delegation, McClurg remained interested in the proceedings of the convention, particularly regarding strengthening of the federal government's powers. "I have still some hope that I shall hear from you of ye reinstatement of ye Negative [federal veto], as it is certainly ye only mean by which the several Legislatures can be restrain'd from disturbing ye order and harmony of ye whole, and ye Governmt render'd properly national, and one," he said in a letter to Madison on August 22.[4(v3,p73)]

McClurg continued to be involved with politics at home after leaving Philadelphia, and served on Virginia's executive council during George Washington's administration.[1(p597)] He was elected mayor of Richmond in 1797, 1800, and again in 1803.[6(p333)] He served as the first president of the Medical Society of Virginia in 1820 and again in 1821, and in 1820 the second edition of the *Philadelphia Journal of the Medical and Physical Sciences* was dedicated to him by its editor. The inscription read:

> To James Maclurg [sic], M.D., of Richmond, Virginia, who, to the widest range of professional knowledge, unites in an eminent degree, the accomplishments of a polite scholar, and eloquent writer, this attempt to promote the cultivation and diffusion of medical literature in the United States is inscribed, as well on account of the profound veneration which I entertain for his character and attainments, as from the ambition, that my undertaking should appear under the auspices of an illustrious citizen of my native state.[6(p334)]

McClurg died in 1823 after a carriage accident caused by runaway horses. He was 77, and was buried in old St John's Courtyard in Richmond.[6(p333)]

The lives of these three physicians, Williamson, McHenry, and McClurg, stand as examples of professional excellence as well as of commitment to public service. While celebrating the bicentennial of our nation's Constitution, we recognize the patriotism of these three men and honor their contributions to the founding of our nation.

REFERENCES

1. American Council of Learned Societies: *Dictionary of American Biography*. New York, Charles Scribner's Sons, 1981, vol 19.
2. Charleton JH, Ferris RG, Ryan MC: *Framers of the Constitution*. National Archives and Records Administration, National Archives Trust Fund Board, Washington, DC, 1986.
3. Padover SK: *The Living U.S. Constitution*, ed 2. New York, New American Library, 1983.
4. Farrand M: *The Records of the Federal Convention of 1787*, rev ed. New Haven, Conn, Yale University Press, 1937.
5. Levy LW, Karst KL, Mahoney DJ: *Encyclopedia of the American Constitution*. New York, Macmillan Publishing Co, 1986.
6. Blanton WB: *Medicine in Virginia in the Eighteenth Century*. Richmond, Va, Garrett & Massie, Inc, 1981.
7. Brant I: *James Madison, Father of the Constitution, 1787-1800*. New York, Bobbs-Merrill Co, Inc, 1950, p 122.

Mr Keane's tree

Pascal James Imperato, MD

He told me that he had planted it when he was a young man and it a sapling. He had found it standing on the edge of a wood in Hempstead, Long Island. Now, decades later, it stood over 30 feet tall in front of his house, casting a dense, cool shadow on hot summer afternoons across the small lawn that spread out in front of his house. He thought that for a maple it had a peculiar shape, and laughed about it. But I thought its round form distinctive and different, setting it apart from others of its kind. He, too, was unique, with his sparkling Irish wit, articulated through a brogue, his sharp insights, and his genuine human warmth.

The tree shielded his lawn from the afternoon sun and its foliage filtered and cooled drafts of hot summer air, making the dark oval below a splendid place in which to rest. He used to sit there on a lawn chair next to his wife, Julia, whom he always called Mama in the presence of others. And together they would look out over a large field which once stretched to woods and more fields beyond when the tree was a sapling. The field was still there, fenced in and marked off for baseball by the Parks Department. But the woods were gone and the other fields too, replaced by a highway, paved streets, and rows of regimented houses. There were always extra chairs on the lawn, an added enticement for passersby to sit and cool themselves in the company of good and witty conversation. He sat in that pool of shade like a human magnet and was never without the company of neighbors and occasional strangers who happened by. He once told me that thanks to the tree his old age was devoid of loneliness. All he had to do was sit in its shade and visitors came. I didn't agree, because even without the tree and the shade and coolness it gave, he had what it took to sustain human companionship.

He and Mama had moved into their house in December of 1928, two months before my parents moved into theirs. They had raised seven children, my parents six. Despite all the years I lived near him, I never really got to know him until I was a grown man and returned for a time to live in my parents' home. By that time all of his children were gone—to California, Connecticut, Pennsylvania, New Jersey, Long Island, and Manhattan—and he and Mama were alone. He was in his late seventies then and worried a lot about illness and dying but did little to prevent either, relying instead on God's will and a pinch of fate. His own father, who had married a young girl when he was in his fifties and had nine children, outlived his wife and lived into his nineties. I used to reassure him by citing his paternal genetic endowment as a portent of longevity. I also induced him to get regular medical care and saw that he took his medicines every day instead of once in a while, which was his preference. He was a tall, thin man with a full head of white hair, and he walked as erect as a palace guardsman. He didn't like the analogy, though, because he had fled Ireland as a young man, leaving the pursuing British military with nothing more than his bicycle. He stowed away on a ship bound for New York, and when he got there with a few coins in his pocket he simply left the pier and started walking, hoping to find a place to stay and a job. That took more courage than facing death, I thought, but he didn't agree.

He cherished his independence and feared losing it. He drove his own car every day, took Mama shopping, mowed the lawn beneath the tree, shoveled snow, went to church every Sunday, and planted a backyard garden full of tulips, zinnias, rose bushes, and tomato plants. A plumber all his life, he did many of his own home repairs as well and was always ready with a wrench, hammer, paint brush, or other tools to help neighbors with theirs. By contrast, his neighbor and crony Henry Gruber was helpless when it came to home repairs. But Mr Gruber, like Mr Keane, had retained his independence as well, despite being a widower and nearing 90 years of age. He traveled once a week to Manhattan on the subway to work as a volunteer at the Veterans Administration Clinic, kept a beautiful garden, was a natty dresser, and led an active social life. Mr Gruber went to the temple every weekday morning and on Saturdays, and sometimes Mr Keane drove him there and back. Mr Keane was relentless in his teasing Mr Gruber about his tailor-made suits, telling him that they were a waste of money because they would never get much use. Mr Gruber took it all in good humor and once confided to me that it was Mr Keane's way of dealing with his own fears of death.

The first break in the perimeter of independence that surrounded Mr Keane's life came on a warm June day when, sitting in the shade of his tree, he learned that Mr Gruber had been sent to a nursing home. Mr Gruber was well, active, and as independent as he and there seemed no

---

Address correspondence to Dr Imperato, Editor, *New York State Journal of Medicine*, 420 Lakeville Rd, Lake Success, NY 11042.

reason for him to leave home. Although he didn't tell me, I knew what he was thinking. If it happened to Mr Gruber, it could happen to him. He dreaded the idea of being institutionalized more than death.

In the spring of 1980 Mr Keane's tree burst forth with a vigorous crop of pale green buds and pods, followed in quick succession by a profusion of leaves. I had never seen his tree so full of life, and no doubt the pruning it had received the previous fall from the Parks Department had much to do with it. By June the pool of shade across the lawn was an unbroken oval of almost pure black, with scarcely a speck of light on it. While the tree was budding the New York City Department of Transportation sent notices to home owners that the streets and sidewalks in this area of Queens County were to be ripped up and replaced, and that new trees would be planted to compensate for any uprooted in the process. Before long, bulldozers plowed up the street. Mr Keane spoke to the workmen about his tree, telling them to take care not to damage the roots. He stood near it several times a day as the forms for the new curb and sidewalk were laid. The supervisors favored removing the tree because a large number of roots had to be cut to accommodate the new curb and sidewalk. Mr Keane protested and I intervened, explaining that the tree meant a great deal to him. But deep down I knew that the tree was really a mirror of his own life and the prospect of its death filled me with a foreboding I couldn't dismiss. The supervisors finally relented, and modified the curb and the sidewalk so that today the sidewalk around the tree takes on a jigsaw shape that accommodates the root system. Despite their care, roots were cut and damaged and some branches withered and soon died. Yet the bulk of the tree was there; only part of it had died.

Soon after Mrs Keane suffered a mild stroke. I first learned of it when he telephoned me one night and told me that he hated to bother me but that Mama had had a headache for two days. She had localized right frontal pain and no distinctive neurological signs. Because she had glaucoma and a cataract in the right eye, the differential diagnosis was somewhat difficult. I suggested we take her to New York Hospital, where the diagnosis of a stroke was confirmed and where she rapidly recovered. However, she was never really the same after that, and he who had relied on her for so much now had to assume more of the burden for their joint independence.

That September my wife was due to give birth to our second child. Our first, Alison, was then two years old and in many ways saw him and Mama as grandparents. She regularly went to Grandma Keane's house for afternoon tea (mostly milk with a dash of tea), which she drank from a porcelain tea cup Mrs Keane had given her. She took her "tea" sitting at the top of a kitchen stool at the head of the table. My wife gave birth to our first son, Gavin, on September 15 and Mr and Mrs Keane were overjoyed. Mrs Keane could hardly wait to see the baby. She wanted a daily report on him and my wife, and looked forward to their coming home. The other event in her life then was a clothes dryer that Mr Keane had purchased for her, but which had not yet arrived. She had never had one and looked forward to the end of using clotheslines.

Two days after Gavin was born, Mrs Keane felt unwell while eating lunch. She told her husband she was going to the basement bathroom, and when she did not return or answer his calls he went down himself. I could barely understand him when he spoke, but I knew that something terrible had happened. When Gavin arrived home the following day, only he was there to see him.

The good intentions of his seven children quickly faded before his resolute determination to go it alone. He loved all of them and was extremely proud of them and of his many grandchildren. His memory about the details of their doings was prodigious, and not a time went by when we met that he did not speak of them. Yet I sensed that somehow they were unaware of how he really felt about them. Whenever I told him this he would say, "Oh, go away with you now." The pride that sustained his independence as an old man also stood in the way of his releasing his true feelings. This troubled me, because I also sensed that perhaps his children carried childhood memories of him similar to my own, of a stern man who could discipline and disapprove with but a glance. Raising children and paying a mortgage through the Great Depression took its toll, he once told me. On the brink of survival there was little room for indulging children. He had survived a near foreclosure in the early 1930s when he was unable to meet the payments on a mortgage held by a German grocer in Ridgewood, Queens. He got no pity from the finance company and as a last resort asked to speak to the mortgage holder. The grocer stood firm, but he persisted and through the intervention of the grocer's wife got a reprieve. She told her husband that he couldn't throw a man and his wife and several children out on the street with nowhere to go. Experiences of this kind left their marks on him for many years, marks which had now softened and even faded away.

His children arranged for a homemaker to come each day, but he would have none of it and threw her out. Mama had done the shopping and the cooking, the cleaning and the wash, tasks which he tried to do, but with little success. The new clothes dryer arrived but stood in the basement unused. As a part of the tree was gone, so too was a vital part of him no longer there. Children, friends, and neighbors, armed with concern and love, met defeat before a fierce independence that only calamity could overcome. While the tree struggled to repair its life, so too did he, alone and unaided.

Mr Keane's age and physical state should have precluded his driving a car. But his car symbolized his mobility; it was part of the centerpiece of his independence. I once asked him to drive me to the repair shop where I had to pick up my own car. I will never forget the trip. We arrived in record time, moving with greater than prudent speed, through red lights and the wrong way down one-way streets. In the process we demonstrated that most drivers had either taken defensive driving lessons or else showed good judgment in keeping clear of us. I asked him to follow me on the return, which he did, and that evening spoke to him about his driving. He patiently heard me out and then began his reply with, "Now doctor, I'm but a simple plumber." This opener, in which he formally addressed me and simultaneously deprecated himself, always told me that he had something very important to say about which he had invariably made up his mind. This time it was no different. He stood firm and there was no convincing him otherwise. The following spring he was backing his car out of the long driveway that led to his garage. He must have hit the accelerator instead of the brake, sending the car out into the street at enormous speed. It crashed against the chain link fence that enclosed the field, bounded back again, careening across lawns until it crashed into the fence again. He emerged from the wrecked car, shaken but unhurt, never to drive again. When I spoke with him about it that night he had little recollection of what happened, leading me to suspect that he may have had a transient stroke. He was glad no one else was injured, but angry with himself that the accident had been witnessed by some neighbors he did not particularly like. "Ye know who I mean," he said, using the archaic form of the pronoun that was his habit whenever he got excited. As he saw it, he had shown them that he was incapable of going on alone. But there was more to it than that. The accident galvanized whatever doubts he had about carrying on by himself and brought him to the inescapable conclusion that he could not. He accepted this with an amazing degree of equanimity. It was as if he knew that he was entering a new phase of his life and was prepared for it. Letting go of his home of 53 years and the secure world he had known there was easier now because his wife was no longer a part of it. He went into this new horizon with an open mind, much as he had crossed the ocean when he was a youth. He was in search of a final niche and was prepared to find it through trial and error. He lived with some of his children for a while and finally, after a trip to Ireland where he fell ill, came to the decision to live in St Patrick's Home in the Bronx, where his wife's nephew was the chaplain. He who had scorned nursing homes and feared being institutionalized entered not with fear, but with the joy of having found a safe haven where he could establish a new self.

I had by this time moved to a new home on Long Island and our relationship now depended on the telephone. He told me that we were well out of the old neighborhood, but he really didn't believe his own words, nor did I. He avidly kept abreast of people's doings there, and whenever we spoke he already knew most of the news I had to give him. He generally asked me about the tree, his tree. Was it still there? How did it look? As the highway leading to my office passed within view of it I could always answer his queries. What I didn't tell him was that the tree did not seem the same to me anymore. It has lost its connection to the people who had given it meaning and was more pleasant in memory than in real life. It had no place now in the forward march of either of our lives.

He never wanted me to visit him in the home, for in a way I was not a part of his new world. We communicated with one another over a bridge of happy memories and events that happened to those whom we knew from the old neighborhood. I never told him about those who had died because I didn't want to upset him, but invariably he already knew. He grew to enjoy each and every day, telling me once that he was grateful to God every time he saw the sun rise. He had come to measure his future longevity not in decades and years, but progressively in months, weeks, and days, his independence in terms of feeding himself and walking to the phone.

He died in his eighty-ninth year, on a peaceful spring day, defying the Irish legend he believed in that if any old Irishman makes it to March 21st he will live out the year. I attended his burial as I had his wife's in the small, quiet cemetery north of New York City that he had come to know of through necessity. It was here that during the Great Depression he had buried two of their newborns. The cost was nominal, he once told me, well below that of other cemeteries which he could not then afford. He often spoke to me about this place, and arriving there early before the funeral cortege I found that it was as he had said down to every detail. A small stream flows below the gentle slope on which he and his wife are buried, and beyond it the land rises into a wooded hillside. He had chosen this spot himself, and in 1973 had had a tombstone erected with both their names and their dates of birth on it. When I asked him why he had done this he replied that once he was dead no one would remember to erect a stone, and that if one were already up they would probably forget to inscribe his name on it or else get his birth date wrong. He had left nothing to chance on this account, for at the top of the stone it read

Martin Keane
1898–

I stood beneath a locust tree waiting for the cortege to arrive. As I did I thought of another tree and of other times, of warm summer days, and of two gentle people whose lives had enriched my own.

Diaphragmatic rupture due to blunt trauma

The case for timely intervention

Latham B. Murray, MD; Roger W. MacMillan, MD; Turner Osler, MD;
John E. Olson, MD

Traumatic rupture of the diaphragm occurs in approximately 5% of patients who require laparotomy for penetrating or blunt trauma of the chest or abdomen.[1-3] Though this diagnosis may be readily apparent in the acute setting, occult diaphragmatic rupture may not become apparent for years or even decades. The patients described in this report were admitted to a rural hospital within 48 hours of each other. The report illustrates the varied spectrum of presentation of traumatic rupture of the diaphragm and underscores the need for prompt diagnosis and timely treatment of this injury in the acute setting.

Case Reports

Case 1. A 66-year-old farmer was repairing his idling tractor when the machine slipped into gear and began moving. The rear wheels of the tractor rolled over his pelvis, and the wheel of the attached loaded manure spreader ran over his chest. He was able to walk 30 feet to the tractor, turn off the ignition, and summon help.

In the past, the patient had undergone repair of an epigastric hernia, bilateral inguinal herniorrhaphies, and had had three laparotomies. Two of these were for complications of diverticulitis, and a third, two years prior to this injury, was to repair small bowel obstruction secondary to adhesions.

On arrival at the hospital, the patient was fully conscious and alert, complaining of pain in the pelvis and chest. His blood pressure was 120/86 mm Hg; pulse, 72 beats/min; and respiratory rate, 18/min, unlabored with normal chest wall excursion and breath sounds bilaterally. Sternal compression elicited lateral chest wall tenderness on both sides, with the lower ribs being tender to direct palpation bilaterally. A diffuse superficial abrasion was present over the lower chest anteriorly, with mild tenderness to deep palpation in the left upper and lower quadrants, but no signs of peritoneal irritation.

The hematocrit was 39.5% and remained stable during the evaluation. The initial chest radiograph showed elevation of the left hemidiaphragm, several left rib fractures, and a small right pneumothorax. A pelvic film showed bilateral avulsion fractures of the iliac wings. Since the patient was stable and to further delineate the contents of the left hemithorax, a computerized axial tomographic (CAT) scan of the chest and upper abdomen was obtained. This demonstrated abdominal viscera in the left hemithorax and an enlarging right pneumothorax (Fig 1). The liver, spleen, and pancreas were intact on CAT scan, and there was no evidence of any free intraperitoneal air.

A right thoracostomy tube was inserted, and an abdominal exploration was performed through a left subcostal incision. The stomach, spleen, and splenic flexure with adjacent transverse colon were displaced into the left chest cavity through a 14-cm laceration in the posterolateral aspect of the left hemidiaphragm. The viscera were returned to the peritoneal cavity, and the diaphragmatic wound was repaired with interrupted sutures of 2-0 Ticron®.

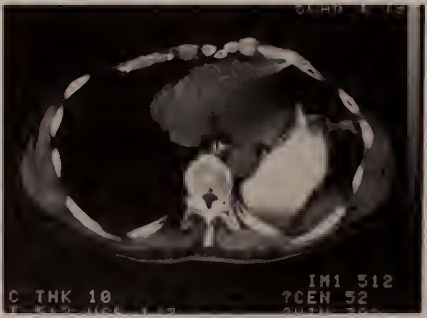


FIGURE 1. Case 1: CAT scan showing contrast-filled stomach, spleen tip, and air-filled loops of colon in left hemithorax.

The postoperative course was uneventful, and the patient was discharged after 11 days with a full subsequent recovery.

Case 2. A 32-year-old man was admitted to the hospital with gastric outlet obstruction manifested by five days of progressive bloating, pain in the right upper quadrant, nausea, and vomiting.

For three years prior to this presentation, the patient had noted bloating and borborygmi whenever he drank carbonated beverages, and for the past year he had experienced postprandial vomiting after large meals. Because of these symptoms, an upper gastrointestinal endoscopy had been performed nine months previously at a local hospital and demonstrated only mild esophagitis. Trials of cimetidine, metaclopramide, and antacids resulted in no sustained relief. In the two months prior to admission he had lost 25 pounds.

Fourteen years previously, he had been in a major car accident, sustaining massive trauma to the right side of his body, including rib fractures, a pelvic fracture, and an open humerus fracture, as well as a concussion. He required multiple blood transfusions to combat hemorrhagic shock, although apparently significant abdominal trauma was unrecognized at that time. He ultimately recovered after an 11-week hospital stay and remained asymptomatic for the next 11 years.

On admission, the patient was afebrile, and the vital signs were normal. His upper abdomen was mildly tender, distended, and tympanitic with a succussion splash. A chest radiograph demonstrated an air-fluid level in the right hemithorax (Fig 2). While in the emergency department, the patient vomited 2,200 mL of liquid and undigested food. A nasogastric tube was then inserted, with the immediate drainage of an additional 1,100 mL of guaiac-positive material.

An upper gastrointestinal series (Fig 3) demonstrated an inverted stomach in the

---

From the Department of Surgery, The Mary Imogene Bassett Hospital, Cooperstown, NY.

Address correspondence to Dr MacMillan, Attending Surgeon, Department of Surgery, The Mary Imogene Bassett Hospital, One Atwell Rd, Cooperstown, NY 13326.

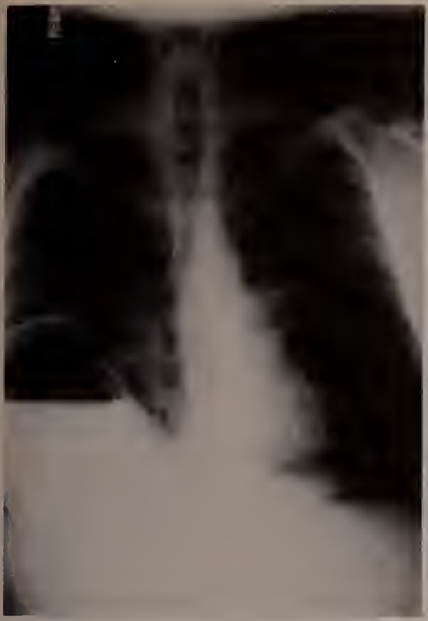


FIGURE 2. Case 2: Chest film taken on admission showing an air-fluid level in the right chest cavity. Note the position of the gastric bubble below left hemidiaphragm.

right hemithorax with a high-grade partial obstruction of the gastric outlet.

At laparotomy, a 12 × 12-cm oval defect was found in the right hemidiaphragm, with a near-complete transection of the liver at the falciform ligament. The inverted stomach, transverse colon, right lobe of the liver and gall bladder, and the medial portion of the left lobe of the liver were found in the right chest cavity. The abdominal viscera were reduced by gentle traction, and the right hemidiaphragm was repaired with a two-layer imbricated closure of interrupted 2-0 Ticron®. The postoperative course was uncomplicated, and the patient returned to work, asymptomatic, eight weeks after surgery.

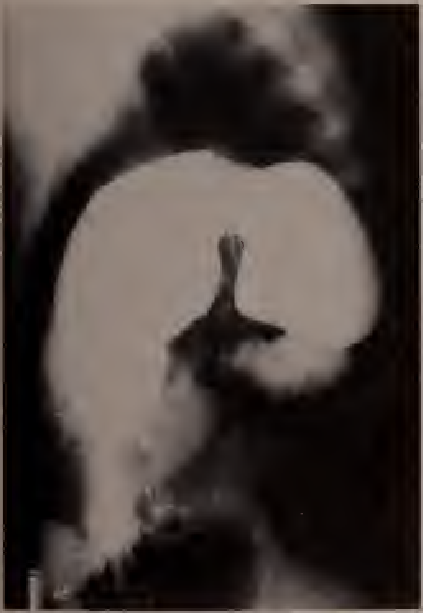


FIGURE 3. Case 2: Upper gastrointestinal series, left lateral upright position. Antrum and duodenal bulb are dilated, with a trickle of barium passing into the second and third portions of the duodenum.

DISCUSSION

In 1579, Ambroise Pare described two cases of traumatic diaphragmatic hernia resulting in death,[4] one secondary to strangulation of the colon in an artillery captain wounded by a gun shot eight months previously, and the other due to strangulation of the stomach in a patient having sustained blunt trauma three days prior to death. The first antemortem diagnosis of traumatic diaphragmatic hernia was reported by Henry I. Bowditch[5] in 1853. In this case, a young man whose thoracic spine had been fractured by falling timber sustained a rupture of the left diaphragm in the accident. In 1900, Walker[6] reported the first successful surgical repair of a diaphragmatic hernia caused by blunt trauma.

Physical findings in blunt diaphragmatic rupture are at best nonspecific. However, the triad of mediastinal shift, dullness or tympany over the lower thorax, and bowel sounds heard in the chest further suggest the diagnosis on a clinical basis.[7]

An upright chest film is a valuable tool for making the diagnosis of an acute diaphragmatic rupture.[8-10] Roentgenographic findings of an elevated or blurred hemidiaphragm, lower rib fractures, gas bubbles within the thorax, pleural fluid, or mediastinal shift suggest the injury.[11] The diagnosis can be confirmed by radiography after the passage of a nasogastric tube, provided the stomach has herniated into the chest.[12] In subacute or delayed cases of diaphragmatic rupture, an upper gastrointestinal series, barium enema, or other contrast studies may be required to make the diagnosis.[13] Peritoneal lavage is useful in detecting associated intra-abdominal injuries but not for the diagnosis of diaphragmatic rupture, unless lavage fluid is found to egress from a previously placed chest tube.[1,10] Diagnostic pneumoperitoneum,[14] arteriography,[7] liver/spleen scan,[9] and CAT scan[15] may all be useful in the stable patient.

The majority of traumatic diaphragmatic ruptures occur on the left side. In most instances, the presence of the liver protects the right hemidiaphragm from injury.[2,7] Acute herniation of abdominal viscera through the disrupted left hemidiaphragm results in a mediastinal shift, which may cause respiratory symptoms that are out of proportion to the otherwise apparent thoracic trauma.[17] Blunt trauma resulting in a diaphragmatic rupture is almost always of sufficient magnitude to cause serious associated injuries. This is particularly true in right-sided cases, virtually all of which are associated with significant injuries to the other organ systems; these cases result in a mortality rate of nearly 50%.[18] Concomitant orthopedic injuries are common, and in patients sustaining blunt abdominal trauma, the presence of a pelvic fracture should raise the index of suspicion that a diaphragmatic rupture may have occurred.[5]

Once the diagnosis of traumatic rupture of the diaphragm has been established, treatment is by immediate surgical repair. The choice of incision for the repair has been a point of controversy over the years, with early authors favoring the thoracic or thoraco-abdominal approach.[11,18,19] Even some recent authors have advocated thoracotomy for diaphragmatic repair,[2] but most now agree that the abdominal incision is the preferred approach in the acute situation, because of the high incidence of associated intra-abdominal injuries and the ease with which the repair can be accomplished.[20] Regardless of the placement of the initial incision, it should be stressed that both the abdomen and chest are to be prepared and draped in the sterile field should a second incision be necessary. In cases that present later, adhesions in the chest can complicate a reduction of the herniated viscera, and thoracotomy may be the preferred approach. Repair can be performed with heavy, nonabsorbable, interrupted sutures in either a simple one-layer closure or a two-layered imbricated technique. Prosthetic materials are rarely required, but may be necessary if there has been a long interval between injury and repair, making approximation of the defect difficult without excessive tension. However, in the patient described here who was treated 14 years after injury, there was no difficulty closing the 12 × 12-cm defect in a linear manner without tension.

Since traumatic rupture of the diaphragm is rarely an isolated injury, mortality figures from this entity reflect the extent of multiple trauma. Published series of blunt diaphragmatic rupture report mortality rates of 4–50%, with nearly all of the deaths occurring as a consequence of associated injuries. An exception is the report of Beauchamp et al,[9] in which the majority of deaths occurred as a result of delayed diagnosis and treatment of the diaphragmatic injuries. Complication rates following successful repair are high, mostly involving pulmonary insufficiency from atelectasis or contusion of lung parenchyma, lung abscess, or persistent pneumothorax.[2,3] In cases of strangulation or perforation of a herniated viscus, serious sequelae, including empyema, can prolong the hospital stay and contribute to morbidity.[14]

In case 1, the absence of any associated intra-abdominal injuries and prompt diagnosis contributed to a quick recovery and short hospital stay. In the second case, the patient experienced serious and chronically debilitating symptoms at a time remote from his injury. He was fortunate not only to have survived the nonoperative treatment of his acute injury, with an associated severe liver laceration, but also the symptoms of gastric outlet obstruction. Strangulation and infarction of the stomach could have occurred and led to a very different outcome.

REFERENCES

1. Freeman T, Fischer RP: The inadequacy of peritoneal lavage in diagnosing acute diaphragmatic rupture. *J Trauma* 1976; 16:538–542.
2. Nano M, Dei Poli M, Mosetti C, et al: Traumatic diaphragmatic hernias. *Surg Gynecol Obstet* 1980; 151:191–192.
3. Dajee A, Schepps D, Hurley EJ: Diaphragmatic injuries. *Surg Gynecol Obstet* 1981; 153:31–32.
4. Sabiston DC, Spencer FC: *Gibbon's Surgery of the Chest*, ed 4. Philadelphia, WB Saunders Co, 1983, pp 833–848.
5. Bowditch HI: Diaphragmatic hernia. *Buffalo Med J* 1853; 9:1–39, 65–94.
6. Walker EW: Diaphragmatic hernia, with report of a case. *Int J Surg* 1900; 13:257–261.
7. Pomerantz M, Rodgers BM, Sabiston DC: Traumatic diaphragmatic hernia. *Surgery* 1968; 64:529–534.
8. Strug B, Noon GP, Beall AC Jr: Traumatic diaphragmatic hernia. *Ann Thorac Surg* 1974; 17:444–449.
9. Beauchamp G, Khalfallah A, Girard R, et al: Blunt diaphragmatic rupture. *Am J Surg* 1984; 148:292–295.
10. Brown GL, Richardson JD: Traumatic diaphragmatic hernia: A continuing challenge. *Ann Thorac Surg* 1985; 39:170–173.
11. Hood RM: Traumatic diaphragmatic hernia. *Ann Thorac Surg* 1971; 12:311–324.
12. Iuchtman M, Freire EC, Jacob ET: Acute diaphragmatic hernia caused by blunt trauma. *Am Surg* 1977; 43:460–463.
13. Carter R, Brewer LA: Strangulating diaphragmatic hernia. *Ann Thorac Surg* 1971; 12:281–290.
14. Ward RE, Flynn TC, Clark WP: Diaphragmatic disruption secondary to blunt abdominal trauma. *J Trauma* 1981; 21:35–38.
15. Heiberg E, Wolverson MK, Hurd RN, et al: CT recognition of traumatic rupture of the diaphragm. *Am J Roentgenol* 1980; 135:369–372.
16. McCune RP, Roda CP, Eckert C: Rupture of the diaphragm caused by blunt trauma. *J Trauma* 1976; 16:531–537.
17. Flancbaum L, Morgan AS, Esposito T, et al: Non-left sided diaphragmatic rupture due to blunt trauma. *Surg Gynecol Obstet* 1985; 161:266–270.
18. Carter BN, Giuseffi J: Strangulated diaphragmatic hernia. *Ann Surg* 1948; 128:210–225.
19. Carter BN, Giuseffi J, Felson B: Traumatic diaphragmatic hernia. *Am J Roentgenol* 1951; 65:56–72.
20. De la Roche AG, Creel RJ, Mulligan GWN, et al: Diaphragmatic rupture due to blunt abdominal trauma. *Surg Gynecol Obstet* 1982; 154:175–180.

Bilateral diaphragm paralysis associated with topical cardiac hypothermia

MANUEL R. CABRERA, MD, JOHN R. EDSALL, MD

Current techniques of cardioplegia and myocardial protection during cardiopulmonary bypass usually involve systemic body cooling, instillation of cold electrolyte solution through the aortic root, and the application of topical pericardial hypothermia. The benefits of this technique have been demonstrated experimentally and clinically. Myocardial hypothermia reduces myocardial oxygen consumption[1] and is associated with an increased rate of spontaneous defibrillation in the post-ischemic period.[2] Topical cardiac hypothermia provides more uniform myocardial cooling.[3] This technique involves repeated cold saline irrigation of the pericardial sac or instillation of an iced saline slush with or without use of a cooling jacket. Unilateral diaphragmatic paralysis is well recognized following topical hypothermia and is of little consequence. In contrast, bilateral paralysis due to cold injury is an unusual complication that results in prolonged intubation and hospitalization. We report a case of bilateral diaphragmatic paralysis following cardiac hypothermia that was managed with intermittent mechanical ventilation.

CASE REPORT

A 61-year-old man with a history of a heart murmur presented to a community hospital with the acute onset of shortness of breath, fever, and cough productive of blood-streaked sputum. Physical examination revealed bilateral coarse rales, and a chest radiograph showed bilateral infiltrates in the mid-lung field. Progressive pulmonary edema developed, and the patient was transferred to Columbia Presbyterian Medical Center.

Cardiac catheterization showed severe mitral regurgitation, and mitral valve replacement was performed through a right atriotomy via the interatrial septum. At operation, there was myxomatous degeneration of the mitral valve with rupture of the chordae tendineae. The aortic cross clamp time, during which topical hypothermia was performed with continuous cold saline lavage at 0° to 4 °C, was 87 minutes. Total bypass time was 143 minutes.

Following hemodynamic stabilization, the patient's endotracheal tube was removed on the fourth hospital day. He required reintubation after 40 minutes, because of respiratory distress attributed to atelectasis in the left lower lobe and increased bronchial secretions. Two further attempts at extubation were unsuccessful, and a tracheostomy was performed on the 14th hospital day. During spontaneous breathing, the patient complained of orthopnea and difficulty sleeping. He was noted to have paradoxical inward motion of the abdominal wall during inspiration.

Pulmonary function tests showed a vital capacity of 1.6 L in the sitting position and 1.2 L in the supine position. The maximal inspiratory and expiratory muscle pressures at the mouth were −35 cm H_2O and +38 cm

---

From the Department of Medicine, College of Physicians and Surgeons, Columbia University, New York, NY.

Address correspondence to Dr Cabrera, Assistant Professor, Department of Medicine, College of Physicians and Surgeons, Columbia University, 630 West 168th St, New York, NY 10032.

H_2O, respectively. Echosonography of the thorax in the supine position demonstrated absence of diaphragm motion during spontaneous respiration. Phrenic nerve conduction studies revealed a slightly prolonged latency of 9.2 msec bilaterally and reduced amplitudes of 175 μV on the right and 100 μV on the left. Measurement of transdiaphragmatic pressure in standard fashion using latex esophageal and gastric balloons and done on the 30th hospital day revealed a pressure of 1 cm H_2O during tidal breathing (normal, 5–8 cm H_2O) and 2 cm H_2O with maximal inspiration (normal, >25 cm H_2O).[4]

The patient was placed on a portable volume ventilator (Thompson M-25B: Thompson Respiration Products, Inc, Boulder, Colo) and maintained good gas exchange without respiratory acidosis while receiving assisted ventilation six hours a day. The patient was discharged home on the 35th hospital day with a tracheostomy and positive pressure ventilatory support averaging six hours a day, seven days a week. Comparison of inspiratory and expiratory radiographs at discharge demonstrated a 0.5-cm difference in diaphragm height on the left and a 2.1-cm difference on the right. Repeat inspiratory and expiratory radiographs six weeks later demonstrated a 1.5-cm difference in diaphragm excursion on the left and no change from the previous study on the right. He continued to have orthopnea and exertional dyspnea, but was increasingly able to perform his usual activities at home. Four months after surgery, his vital capacity was 3.4 L, and the maximal inspiratory and expiratory muscle pressures at the mouth were −60 cm H_2O and +150 cm H_2O, respectively. The tracheostomy tube was removed, and he continued to do well.

DISCUSSION

In 1963, Scannell[5] reported that four of 25 cardiac surgical procedures using topical hypothermic cardioplegia were complicated by diaphragm paralysis. Two of the patients were reported to demonstrate bilateral paralysis. In one patient, pathologic evaluation of the phrenic nerve yielded changes consistent with cold injury. With the increased application of hypothermic cardioplegia in the early 1970s there has been an increased recognition of atelectasis and hemidiaphragm elevation, more commonly occurring on the left. Rousou et al[6] reported elevation of the left hemidiaphragm in two of 16 patients (12.5%) with the use of a cooling jacket, 12 of 50 patients (24%) with iced saline slush, and nine of 35 patients (23%) with both. In a prospective study of 40 patients after surgery, Benjamin et al[7] found that 26 (83%) showed atelectasis and infiltrates on chest radiographs; 18 of the 26 had radiographic evidence of diaphragm paralysis. Markland et al[8] studied transcutaneous nerve stimulation in 44 open heart procedures and found decreased conduction in five patients (11%) compared to the preoperative values. None of these findings were bilateral abnormalities. In an animal model, Marco and coworkers[9] showed that direct or indirect exposure of the phrenic nerve to crushed ice for 30 to 60 minutes resulted in unresponsiveness of the nerve to conduction which lasted from seven to 62 days, and showed a spectrum of nerve injury from mild demyelination to severe demyelination with evidence of axon fragmentation. While unilateral phrenic nerve injury and associated elevated hemidiaphragm can be found in a significant number of patients who have undergone open heart surgery, these changes appear to be of little clinical significance. In the reports cited above, no differences in postoperative course or length of hospital stay were noted.

Bilateral diaphragm paralysis, however, is unusual and is associated with significant morbidity. Brown et al[10] reported on a patient with bilateral paralysis which was diagnosed with an esophageal and gastric balloon study. The patient required continuous ventilatory support in the intensive care unit for eight weeks. A follow-up study eight months later demonstrated the patient's recovery. Kohorst and coworkers[11] reported on two patients with bilateral paralysis. One had recovered on follow-up after ten months. The other remained symptomatic up to the time of cardiopulmonary arrest six months after surgery. Chandler et al[12] described five patients with 50% decreases in vital capacity from the upright to the supine position who had clinical evidence of bilateral diaphragm paralysis. Of the above eight reported cases of bilateral diaphragm paralysis, six followed triple bypass surgery, and two occurred after valve replacement.

The recognition of diaphragm paralysis was usually delayed and was suspected only on further evaluation of unexplained orthopnea, dyspnea on exertion, or difficulty weaning. Confirmation of bilateral paralysis may be difficult. A decrease in vital capacity from the upright to the supine position is nonspecific. The classic "sniff test" is often misleading, because the diaphragm may descend passively in the upright position.[4] In this patient, ultrasonography resolved those requisite difficulties. It was easily obtainable and was performed at the bedside with the patient in the supine position following surgery. The absence of diaphragm motion on inspiration, demonstrated by the ultrasound study, prompted further diagnostic evaluation.

Ultrasonography has been used in the evaluation of diaphragm motion in healthy persons[13] and in patients with subdiaphragmatic disease.[14] More recently, it has been recommended as the primary imaging modality in children with suspected abnormalities of diaphragm motion or position, because of the procedure's easy availability—an important feature in treating the critically ill patient—the absence of radiation, and its ability to also detect paradiaphragmatic masses and fluid collections.[15] Documentation of bilateral paralysis can best be achieved by phrenic nerve conduction studies or measurement of transdiaphragmatic pressure, as was done in this case.

Persistence of paralysis may contribute to later morbidity and mortality, as suggested by one of Kohorst's patients. In the patient described here, continued ventilatory support with the use of a portable volume ventilator allowed earlier discontinuation of intensive care requirements and earlier hospital discharge. Our objective was to provide continued ventilatory support during recovery of the phrenic nerve. Marino and Braun,[16] in a study of 35 patients with hypercapnia and recurrent respiratory decompensation, showed that the use of intermittent negative or positive pressure ventilation resulted in complete or partial normalization of arterial P_{CO_2}. The patients also demonstrated an increase in daily activities, improved respiratory muscle strength, and maximum voluntary ventilation after a mean of five months of therapy. Hypoventilation can often result from bilateral diaphragm paralysis, particularly in the supine position, which may worsen during REM sleep. This patient used the ventilator for decreasing periods during the day, with improvement in symptoms and increased activity, presumably due to rest of the accessory muscles of respiration during the recovery period.

REFERENCES

1. Buckberg GD, Brazier JR, Nelson RL, et al: Studies of the effects of hypothermia on regional myocardial blood flow and metabolism during cardiopulmonary bypass. I. The adequately perfused, beating, fibrillating, and arrested heart. *J Thorac Cardiovasc Surg* 1977; 73:87–94.

2. Roe BB, Hutchinson JC, Fishman NH, et al: Myocardial protection with cold, ischemic, potassi-

um-induced cardioplegia. *J Thorac Cardiovasc Surg* 1977; 73:366-374.

3. Landymore RW, Tice D, Trehan N, et al: Importance of topical hypothermia to ensure uniform myocardial cooling during coronary artery bypass. *J Thorac Cardiovasc Surg* 1981; 82:832-836.

4. Loh L, Goldman M, David JN: The assessment of diaphragm function. *Medicine* 1977; 56:165-169.

5. Scannell JC, discussion of McGoon DC, Mankin HT, Kirklin JW: Results of open-heart operation for acquired aortic valve disease. *J Thorac Cardiovasc Surg* 1963; 45:47-66.

6. Rousou JA, Parker T, Englemen RM, et al: Phrenic nerve paresis associated with the use of iced slush and the cooling jacket for topical hypothermia. *J Thorac Cardiovasc Surg* 1985; 89:921-925.

7. Benjamin JJ, Cascade PN, Rubenfire M, et al: Left lower lobe atelectasis and consolidation following cardiac surgery: The effect of topical cooling of the phrenic nerve. *Radiology* 1982; 142:11-14.

8. Markland ON, Moorthy SS, Mahomed Y, et al: Postoperative phrenic nerve palsy in patients with open heart surgery. *Ann Thorac Surg* 1985; 39:68-73.

9. Marco JD, Hahn JW, Barner HB: Topical cardiac hypothermia and phrenic nerve injury. *Ann Thorac Surg* 1977; 23:235-237.

10. Brown KA, Hoffstein V, Byrick RJ: Bedside diagnosis of bilateral diaphragmatic paralysis in a ventilator-dependent patient after open-heart surgery. *Anesth Analg* 1985; 64:1208-1210.

11. Kohorst WR, Schonfeld SA, Altman M: Bilateral diaphragmatic paralysis following topical cardiac hypothermia. *Chest* 1984; 85:65-68.

12. Chandler KW, Rozas CJ, Kory RC, et al: Bilateral diaphragmatic paralysis complicating local cardiac hypothermia during open heart surgery. *Am J Med* 1984; 77:243-249.

13. Harris RS, Giovannetti M, Kim BK: Normal ventilatory movement of the right hemidiaphragm studied by ultrasonography and pneumotachography. *Radiology* 1983; 146:141-144.

14. Haber K, Asher WM, Freimanis AK: Echographic evaluation of diaphragmatic motion in intra-abdominal diseases. *Radiology* 1975; 114:141-144.

15. Diament MJ, Boechat MI, Kangarloo H: Real-time sector ultrasound in the evaluation of suspected abnormalities of diaphragmatic motion. *JCU* 1985; 13:539-543.

16. Marino W, Braun NMT: Reversal of the sequelae of respiratory muscle fatigue by intermittent mechanical ventilation (abstract). *Am Rev Resp Dis* 1982; 125(pt 2):85.

Bochdalek hernia in adulthood

RICHARD FINE, MD; EDGAR BORRERO, MD; ALEX STONE, MD

Congenital diaphragmatic hernia of the foramen of Bochdalek (pleuroperitoneal canal) usually presents in the first few days of life. Abdominal viscera herniate into the chest due to incomplete or absent fusion of the pleuroperitoneal folds. Compression of the developing lung leads to pulmonary hypoplasia, which is the single most important prognostic indicator in the neonatal period.[1] Infants with Bochdalek hernias will be in acute respiratory distress. Despite extensive research, this congenital anomaly is still associated with a high mortality rate.[2,3]

When a Bochdalek hernia is diagnosed in childhood or adulthood, the clinical presentation differs greatly from that seen in the neonate. Most reports of noninfantile Bochdalek hernias (delayed presentation of congenital diaphragmatic hernias) describe gastrointestinal tract symptoms and some pleuritic chest pain without evidence of pulmonary disorders.

The case report presented here and the accompanying literature review will help to elucidate some of the theories that previous authors have proposed to explain late-onset or delayed presentation of diaphragmatic herniation.

CASE REPORT

A 44-year-old man was admitted to the Long Island Jewish Medical Center with a two-day history of increasingly severe abdominal pain in the left upper quadrant, nausea, and vomiting, accompanied by left pleuritic chest pain. Ten months prior to this admission, he underwent elective repair of a left sliding inguinal hernia that contained part of the sigmoid colon. A preoperative chest film taken at that time revealed an air-fluid level at the base of the left lung (Fig 1). Subsequently, an upper gastrointestinal (GI) series and a barium enema revealed herniation of the splenic flexure of the colon through the left hemidiaphragm, a small hiatus hernia, and a moderate sized diverticulum in the descending portion of the duodenum (Fig 2). Yearly physical examinations, including chest films, failed to reveal any abnormalities prior to this admission. The patient denied any history of abdominal or chest trauma.

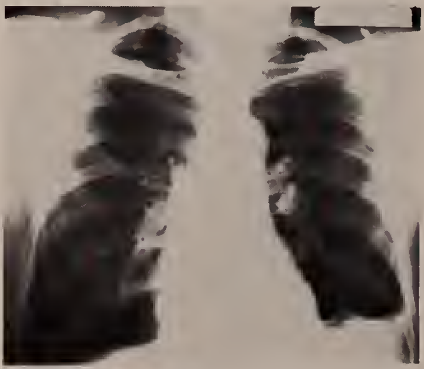


FIGURE 1. Preoperative chest film revealing air-fluid level at the left base.

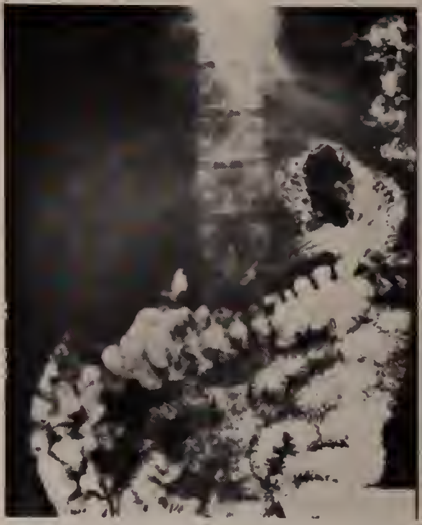


FIGURE 2. Barium enema followed by an upper GI series shows herniation of the splenic flexure through the left hemidiaphragm.

Despite the presence of the diaphragmatic hernia, the patient underwent successful repair of a large sliding inguinal hernia. He remained asymptomatic for approximately five to six weeks, when he had two separate episodes of abdominal pain in the left upper quadrant, which resolved spontaneously. Ten days later, he began to experience the symptoms that led to this hospital visit. He denied any recent change in bowel movements, melena, or hematochezia, but did complain of intermittent mild shortness of breath during the week prior to admission.

Physical examination revealed an apparently healthy man in acute distress. His blood pressure was 140/100 mm Hg, his

---

From the Department of Surgery, Long Island Jewish Medical Center, New Hyde Park, NY, and the State University of New York (Stony Brook), Stony Brook, NY.

Address correspondence to Dr Stone, Department of Surgery, Long Island Jewish Medical Center, New Hyde Park, NY 11042.

pulse rate was 100/min, and his respiratory rate was 26/min. There were no breath sounds in the left lower mid-axillary intercostal spaces. The abdomen was flat and soft with hypoactive bowel sounds. Tenderness and guarding were present in both the epigastrium and the left upper quadrant. No rebound tenderness was elicited. The liver and spleen were nonpalpable. A stool sample was negative for occult blood.

Laboratory values included a hematocrit of 50%, hemoglobin, 17.1 g/dL; and white blood cell count, 16,900/cc, with a normal differential. The serum electrolyte levels were all within normal limits. The roentgenogram of the chest and abdomen revealed hyperlucent areas in the lower left hemithorax with no definable left hemidiaphragm. A large air-fluid level in the region of the left upper abdomen was also noted, which was interpreted as obstructed large bowel (Fig 3).

The patient underwent an exploratory laparotomy on the night of his admission. A large, incarcerated posterolateral left diaphragmatic hernia was found. The defect in the diaphragm measured approximately 16 cm × 4 cm. Using blunt traction on the stomach and the colon, and lysing chronic adhesions in the area of the left lower lung, the stomach, left colon, omentum, and spleen were reduced from the lower left chest cavity. There was no malrotation of the bowel, and no hernia sac was present. The diaphragmatic defect was closed primarily without tension using interrupted O silk sutures.

The patient's postoperative course was uneventful, except for a small pleural effusion in the left costophrenic angle. He was discharged from the hospital on the sixth day after surgery and has done well.

DISCUSSION

The occurrence of a diaphragmatic hernia was cited in the literature as early as 1690, but it was not until 1834 that Laennac suggested the possibility of laparotomy to repair these defects.[4] The posterior lateral foramen was described in 1848 by Bochdalek, a professor of anatomy, and it has since been named after him.[5]

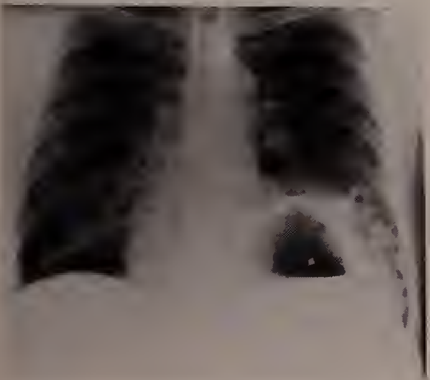


FIGURE 3. Chest film taken prior to emergency laparotomy reveals large air-fluid level in the lower left hemithorax.

A congenital diaphragmatic hernia results from the failure of fusion of the pleuroperitoneal folds. During the third week of gestation, the diaphragm originates as the septum transversum. As the septum transversum migrates posteriorly, it fuses with the dorsal mesentery of the foregut, leaving a defect referred to as a pleuroperitoneal hiatus posteriorly on each side of the spine. For a short period of time, the abdominal and thoracic contents may freely communicate through these defects, until the pleuroperitoneal folds fully develop. If these folds fail to develop completely, a Bochdalek hernia results. The process is usually completed by the ninth week of gestation, with the left side being the last to close. No sac will be present if failure of fusion occurs early during gestation. If development stops sometime later, a sac may be present; it is comprised of peritoneum and parietal pleura.[4,6]

The incidence of Bochdalek hernias in the neonate has been reported as ranging from one in 3,600[7] to one in 7,000,[8] with late presentations being even less frequent. Males predominate by a 2:1 ratio in the neonatal period and by 3:1 in the delayed presentation, with 70–90% of the defects being found on the left side.[7]

The mortality rate in the neonate approaches 100% without surgery and with surgery it has remained between 25% and 50%.[8] In contrast, surgical cure of noninfantile Bochdalek hernia is reportedly 80–100%.[4,8] Although most delayed presentations of this defect occur in the first and second decades of life, there have been multiple reports of patients presenting in the third to eighth decades.[10–13,15–25]

In 1959, Kirkland[12] reviewed 39 cases of noninfantile Bochdalek hernias and found that a few patients remained asymptomatic, while the majority presented with vague, recurrent respiratory and abdominal complaints. Acute intestinal obstruction developed in only five patients. Several other authors have reported similar findings related to mechanical obstruction of the bowel within the hernia.[10,13,14]

In 1971, Fromm and Lucas[15] stated that the presence of a Bochdalek hernia may be excluded by a previous normal chest roentgenogram. However, more recent case reports have documented that, in spite of earlier normal chest roentgenograms, congenital diaphragmatic hernias may present later in life.[13,16–18] When reviewing the chest roentgenogram, one must have a high index of suspicion, because the preliminary radiologic interpretation is frequently a left lower lobe pneumonia, pleural effusion, or eventration of the diaphragm.[10,13,17]

The noninfantile Bochdalek hernia is analogous to the delayed presentation of inguinal hernias commonly found in infants and children.[19] Many authors have reconfirmed the statement by Hight et al[19] that "alterations of growth, intra-abdominal pressure, and size of the defect will determine the age of onset of clinical symptomatology."[10,17,20,21] During surgery, in several cases, the spleen is found to barely fit through the defect when being reduced, and therefore it is postulated that the spleen has a plugging effect and protects against herniation for a period of time.[10,18] Temporal relationships have been demonstrated between pressure changes in the thoracoabdominal cavities and the onset of clinical symptoms. These changes have occurred after imbibing copious quantities of fluid, after ingesting a large meal,[22] during sexual intercourse,[23] and as a result of sneezing or coughing.[11] Development and aggravation of incarcerated hernias has also been described during pregnancy and labor.[24] Other conditions that can lead to a secondary diagnosis of a diaphragmatic hernia include peritonitis, paralytic ileus, trauma, and operative manipulation.[7,13,14]

In the case presented here, the spleen may in fact have plugged the defect for many years, thus preventing herniation and delaying the onset of symptoms and the subsequent diagnosis. Sequential changes in thoracoabdominal pressures secondary to the barium enema, reduction of a large sliding hernia followed by slow postoperative rehabilitation requiring coughing and deep breathing exercises may have permitted the abdominal contents to herniate into the left thorax through the small diaphragmatic defect.

Surgical repair of a diaphragmatic hernia should be attempted as soon as the diagnosis is confirmed. Most surgeons prefer to repair the defect via the abdomen, although it may be accomplished by a technically more difficult approach through the chest. Incarceration is a potentially life threatening situation that depends on a number of factors including the patient's pulmonary function and dependence on the one lung that is being collapsed. Other serious complications can occur, including small and large bowel ischemic necrosis with or without perforation, necrosis of the stomach, splenic infarction, necrosis of the liver, and acute respiratory failure with hypoxemia and shock.[14,16,20]

In retrospect, the patient presented here would have been better served had elective surgical repair of his congenital diaphragmatic hernia been done when the diagnosis was confirmed with the colon visible in the left chest on barium enema. This would have avoided the subsequent incarceration of the abdominal viscera in the hernia. As previously stated, the results of elective repair of delayed Bochdalek hernias are usually excellent.

REFERENCES

1. Lores ME, Delgado R, Marquez E: Delayed presentation of a congenital diaphragmatic hernia resulting in stomach incarceration. *Bol Asoc Med PR* 1982; 74:136–137.
2. Ruff SJ, Campbell JR, Harrison MW, et al: Pediatric diaphragmatic hernias. An 11 year experience. *Am J Surg* 1980; 139:641–645.
3. Ein SH, Barker G, Olley P, et al: The pharmacologic treatment of newborn diaphragmatic hernia—a 2-year evaluation. *J Pediatr Surg* 1980; 15:384–394.
4. Ravitch MM: Congenital diaphragmatic hernia, in Nyhus LM, Condon RE (eds): *Hernia*, ed 2. Philadelphia, JB Lippincott Co, 1978, pp 602–619.
5. Bochdalek: Einige Bemerkungen uber die Entstehung des angeborenen Zwerchfellbruches. Als beitrag zur pathologishen anatomic der hernien. *Vrtlischr fd Prakt Heilk Prag* 1848; 3:89–87.
6. Snyder WH Jr, Greany EM Jr: Congenital diaphragmatic hernia. Seventy-seven consecutive cases. *Surgery* 1965; 57:576–588.
7. Booker PD, Meerstadt PW, Bush GH: Congenital diaphragmatic hernia in the older child. *Arch Dis Child* 1981; 56:253–257.
8. Gravier L, Dorman GW, Votteler TP: Congenital diaphragmatic hernia in children. *Surg Gynecol Obstet* 1971; 132:408–410.
9. Dibbins AW, Wiener ES: Mortality from neonatal diaphragmatic hernia. *J Pediatr Surg* 1974; 9:653–662.
10. Wiseman NE, MacPherson RI: 'Acquired' congenital diaphragmatic hernia. *J Pediatr Surg* 1977; 12:657–665.
11. Lynch JM, Adkins JC, Wiener ES: Incarcerated congenital diaphragmatic hernias with bowel obstruction (Bochdalek). *J Pediatr Surg* 1982; 17:537–540.
12. Kirkland JA: Congenital posterolateral diaphragmatic hernia in the adult. *Brit J Surg* 1959; 47:16–22.
13. Day B: Late appearance of Bochdalek hernia. *Br Med J* 1972; 1:786.
14. Wooley MM: Delayed appearance of a left posterolateral diaphragmatic hernia resulting in significant small bowel necrosis. *J Pediatr Surg* 1977; 12:673–674.
15. Fromm SH, Lucas CE: An unusual complication of chronic diaphragmatic hernia in an adult patient. *J Thorac Cardiovasc Surg* 1971; 61:654–656.
16. Kirchner SG, Burko H, O'Neill JA, et al: Delayed radiographic presentation of congenital right diaphragmatic hernia. *Radiology* 1975; 115:155–156.
17. Glasson MJ, Barter W, Cohen DH, et al: Congenital late posterolateral diaphragmatic hernia with previously normal chest x-ray. *Pediatr Radiol* 1975; 3:201–205.
18. Ro JS, Refsum S Jr, Nordshus T: Late presentation of left-sided congenital diaphragmatic (Bochdalek) hernia. *Z Kinderchir* 1981; 34:279–283.
19. Hight DW, Hixoh SD, Reed JO, et al: Intermittent diaphragmatic hernia of Bochdalek: Report of a case and literature review. *Pediatrics* 1982; 69:601–604.
20. Osebold WR, Soper RT: Congenital posterolateral diaphragmatic hernia past infancy. *Am J Surg* 1976; 131:748–754.
21. Sutton PP, Longrigg N: Strangulated Bochdalek hernia in an adult. *J R Coll Surg Edinb* 1982; 27:58–59.
22. Hoffman E: Strangulated diaphragmatic hernia. *Thorax* 1968; 23:541–549.
23. Berkowitz RE: Foramen of Bochdalek hernia in an adult: Case report. *Milit Med* 1981; 146:356–357.
24. Gimovsky ML, Schifrin BS: Incarcerated foramen of Bochdalek hernia during pregnancy. A case report. *J Reprod Med* 1983; 28:156–158.
25. Weinshelbaum AM, Weinshelbaum EI: Incarcerated adult Bochdalek hernia with splenic infarction. *Gastrointest Radiol* 1982; 7:287–289.

Radionuclide imaging in the diagnosis of diaphragmatic hernia associated with neonatal group B streptococcal sepsis

ROBERT CHECOLA, MD; STEPHEN R. KANDALL, MD; FUKIAT ONGSENG, MD;
SYLVAIN M. WEINBERGER, MD; RICHARD GOLDFARB, MD

Group B streptococcal infection is now the leading cause of neonatal septicemia, with a reported incidence of 1.3–3 per 1,000 live births.[1] In an increasing percentage of such cases that occur within the first few days of life, delayed-onset symptomatic diaphragmatic herniation has been documented.[2–5] Despite suspicion of abdominal-thoracic herniation, precise diagnostic confirmation is necessary before surgical correction of the diaphragmatic defect can be undertaken. We describe a neonate in whom liver-spleen radionuclide imaging was used to confirm the diagnosis of diaphragmatic herniation when other diagnostic procedures yielded equivocal findings.

CASE REPORT

A 2,500-gm male infant was born after a 34-week gestation following rupture of membranes. The infant's Apgar scores were 5 at one minute and 8 at five minutes. Tachypnea, retractions, and nasal flaring were evident from birth. An initial chest film taken at two hours of age showed hyperinflation of the lungs with slight, bilateral haziness. A blood culture drawn at one hour of age, prior to antibiotic treatment, grew group B beta-hemolytic streptococcus; cerebrospinal fluid (CSF) culture was negative.

During the first day, the infant's condition rapidly deteriorated; he required endotracheal intubation and mechanical ventilation. Repeat blood cultures drawn on day three were sterile. Positive pressure ventilation was discontinued after nine days, but the infant required reintubation, mechanical ventilation, and increased ambient oxygen. A chest film taken at that time revealed increased density in the lower lobe of the right lung consistent with an effusion; thoracentesis yielded 8 mL of a yellow transudate which contained no bacteria. On day 11, the right diaphragm could no longer be defined on radiologic examination (Fig 1). An ultrasound study suggested a mass in the right side of the chest (Fig 2). A liver-spleen scan performed after the intravenous injection of 500 μCi of technetium sulfur colloid confirmed herniation of part of the liver from the abdominal cavity into the right side of the chest (Figs 3, 4). The following day, a central defect of the diaphragm, with herniation of a large part of the liver, was surgically repaired. The infant's recovery was uneventful, and he was discharged from the hospital on the 35th day of life.

From the Departments of Pediatrics/Neonatology (Drs Checola, Kandall, and Weinberger), and Nuclear Medicine (Drs Ongseng and Goldfarb), Beth Israel Medical Center, Mount Sinai School of Medicine, New York.

Address correspondence to Dr Kandall, Chief, Division of Neonatology, Beth Israel Medical Center, First Ave at 16th St, New York, NY 10003.

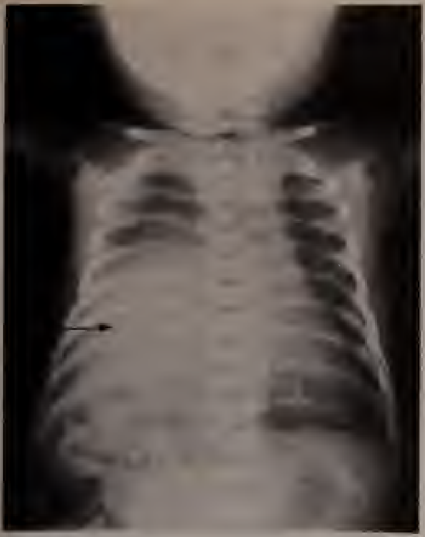


FIGURE 1. Frontal chest film shows density (arrow) occupying lower half of right hemithorax.

DISCUSSION

This case of delayed-onset diaphragmatic herniation associated with neonatal group B streptococcal sepsis followed a pattern similar to cases described in the literature.[2-5] With prompt antibiotic therapy for sepsis, the patient showed initial improvement, subsequently followed by clinical deterioration. This change in clinical status coincided with the development of a density in the right lower lobe and a right pleural effusion. Although a diaphragmatic hernia was suspected on the basis of plain radiographs and ultrasound examination, a liver-spleen scan alone proved diagnostic and allowed timely surgical intervention.

While a chest film remains the initial diagnostic approach to diaphragmatic herniation, such a study is not always definitive. Right-sided hernias may escape radiographic diagnosis if the liver alone, without associated bowel, herniates into the thoracic cavity.[5] Plain radiographs also may not distinguish herniation from an eventration with an effusion. Fluoroscopy may aid diagnosis, but may not distinguish herniation from eventration or diaphragmatic paralysis.[6] Although an ultrasound study may suggest herniation, this technique rarely provides enough diagnostic precision to allow surgical intervention. In this questionable diagnostic setting, radionuclide visualization of a localized elevation of a portion of the right lobe, when matched with chest radiographs, can identify the herniation. This can be confirmed by the presence of a photon-deficient collar due to constriction by the margins of the diaphragmatic rupture (Figs 3, 4). Also known as a band sign, the latter finding is virtually pathognomonic of rupture.[7]

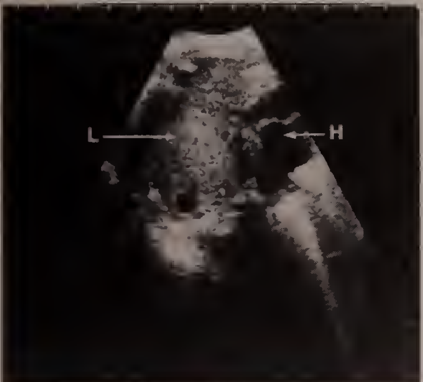


FIGURE 2. Transverse sonographic view at level of lower thorax showing heart (H) and solid structure posteriorly (L).

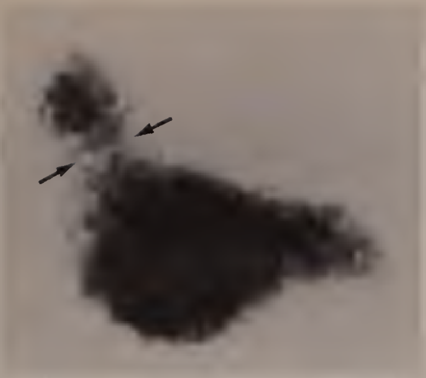


FIGURE 3. Anterior view shows photon-deficient collar (arrows) separating uptake of technetium sulfur colloid in chest from abdomen, pathognomonic of diaphragmatic herniation (band sign).

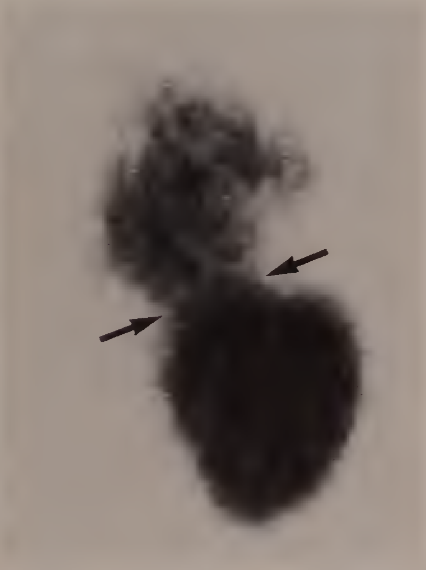


FIGURE 4. Right lateral view demonstrates the posterior location of the rupture. Arrows demarcate photon-deficient collar.

Although the etiology of this late herniation is not fully understood, speculation has focused on the relationship of an adjacent inflammatory process to subsequent diaphragmatic rupture.[2,3] Acute rupture through a normal diaphragm that has been weakened by inflammation seems unlikely. It seems more reasonable that a pre-existing congenital defect might impair ventilation or lymphatic drainage of the right lung. In the setting of neonatal infection, this would promote pulmonary localization of an invasive organism, resulting in reduced pulmonary compliance, increased negative intrapleural pressure, and diaphragmatic herniation. Still unexplained, however, is the occurrence of late rupture only with group B streptococcal sepsis, but not with other organisms, such as *Haemophilus influenzae*, that are known to cause neonatal sepsis.

Successful radionuclide imaging of diaphragmatic herniation associated with neonatal group B streptococcal disease has not been well emphasized in the literature. Ease of imaging, even in extremely ill infants, as well as lack of risk, minimal radiation exposure, and the diagnostic precision of the procedure, all support the use of nuclear liver-spleen scanning in this setting.

Acknowledgment. The authors thank Stacy B. Danheiser for her secretarial services.

REFERENCES

1. Davies PA, Gothefors LA: Bacteremia, infections of the central nervous system and respiratory systems, in *Bacterial Infections in the Fetus and Newborn Infant*. Philadelphia, WB Saunders, 1984, p 123.
2. Harris MC, Moskowitz WB, Engle WD, et al: Group B streptococcal septicemia and delayed-onset diaphragmatic hernia. *Am J Dis Child* 1981; 135: 723–725.
3. McCarten KM, Rosenberg HK, Borden S, et al: Delayed appearance of right diaphragmatic hernia associated with group B streptococcal infection in newborns. *Radiology* 1981; 139:385–389.
4. Akierman AR, Mayock DE: Group B streptococcal septicemia and delayed-onset congenital right-sided diaphragmatic hernia. *Can Med Assoc J* 1983; 129:1289–1290.
5. Kirchner SG, Burko H, O'Neill JA, et al: Delayed radiographic presentation of congenital right diaphragmatic hernia. *Radiology* 1975; 115:155–156.
6. Ashcraft KW, Holder TM, Amoury RA, et al: Diagnosis and treatment of right Bochdalek hernia associated with group B streptococcal pneumonia and sepsis in the neonate. *J Pediatr Surg* 1983; 18:480–485.
7. Gastrointestinal nuclear medicine, in Sty JR, Starshak RJ, Miller JH (eds): *Pediatric Nuclear Medicine*. Norwalk, Conn, Appleton-Century-Crofts, 1983, p 58.

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Diaphragmatic hernia

TO THE EDITOR: In the February 1987 issue of the *Journal,*[1] Robert G. Sperber, MD, described a patient with a diaphragmatic hernia mimicking pyopneumothorax. The difficulty of making a correct diagnosis by various radiographic techniques is described in an editorial by Dr Heiberg in the same issue.[2] We recently had the opportunity of seeing a patient who had sustained blunt trauma to the chest in an automobile accident 18 years previously. He presented with low grade fever, cough, and malaise thought to be due to a viral illness. A chest radiograph obtained in the course of evaluation suggested a large, right-sided pleural effusion. The diagnosis of a diaphragmatic hernia was made by chest ultrasonography performed both in the sitting and the right decubitus positions. An area of marked echogenicity was identified in the right lower lung field. A single, right-side-down decubitus radiograph of the chest identified the area as bowel (Fig 1). A previous chest radiograph taken 12 years earlier was located and proved to be similar in appearance. As the patient was now without symptoms, further evaluation was not undertaken. The combination of decubitus radiography and ultrasonography proved helpful in this case.

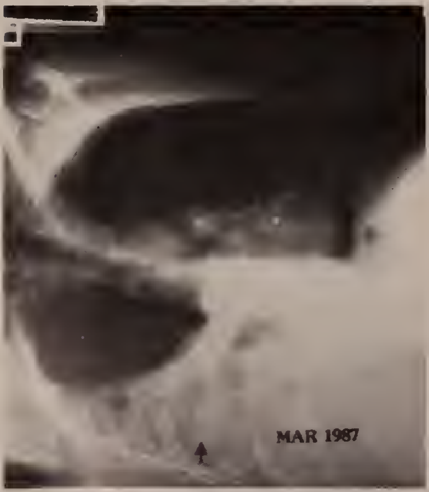


FIGURE 1. Right-side-down decubitus radiograph of chest showing loops of bowel within thoracic cavity and small quantity of layering fluid.

ROBERT H. POE, MD
Associate Professor of Medicine
Director, Pulmonary Unit

CHARLES D. MASKIELL, MD
Clinical Instructor
Preventive, Family and Rehabilitation Medicine
Highland Hospital
University of Rochester School of Medicine and Dentistry
1000 South Ave
Rochester, NY 14620

1. Sperber RG: Diaphragmatic hernia mimicking pyopneumothorax and colonic mass. *NY State J Med* 1987; 87:122-123.
2. Heiberg E: Ruptured diaphragm: A diagnostic dilemma. *NY State J Med* 1987; 87:87-88.

Computed tomography of Morgagni hernia

TO THE EDITOR: The computed tomographic (CT) appearance of Morgagni hernia has rarely been illustrated in the literature.[1,2] At times, only omental fat is contained within the hernia, and thus the hernia may be confused on computed tomography with a fatty mass originating in the chest. The omental vessel sign on computed tomography[1,2] can suggest that a fatty chest mass is in fact a Morgagni hernia.

Case Report. A 31-year-old man in good health was found on a routine chest film to have a large mass in the right cardiodiaphragmatic angle. A previous chest film taken four years previously was reported as normal. The history and physical examination were unremarkable. A CT scan of the chest revealed a large mass in the right cardiodiaphragmatic angle consistent with fat density. The mass was homogeneous except for fine linear and curvilinear soft tissue densities within it (Fig 1). At thoracotomy, a Morgagni hernia with omental fat was successfully repaired. The patient had an uneventful recovery.

Discussion. Morgagni hernias occur through potential anterior-medial defects in the diaphragm, with the majority occurring on the right side, possibly because the left space is reinforced by the pericardium. The most common contents of these hernias are omentum and/or colon.[3] When bowel is contained within the hernia, the diagnosis is usually made by plain film and barium studies.

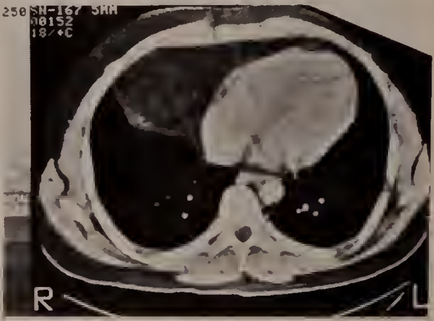


FIGURE 1. A large fatty mass in the right cardiodiaphragmatic angle showing homogeneous fat density except for fine linear and curvilinear soft tissue densities.

Morgagni hernias will sometimes present on chest film as a mass in the right cardiodiaphragmatic angle[3] or as cardiomegaly.[4] If cardiomegaly is ruled out, a CT scan should be done, which would demonstrate a fatty mass in the right cardiodiaphragmatic angle. The differential diagnosis on CT includes Morgagni hernia, lipoma, and a pericardial fat pad. If fine linear or curvilinear densities are seen within the fatty mass (Fig 1), the diagnosis of Morgagni hernia is strongly suggested. The fine soft tissue densities probably represent omental vessels.[1,2]

CT multiplanar reconstruction is also useful in the diagnosis of Morgagni hernias to show a discontinuous diaphragm and the continuity of the thoracic fatty mass with the abdominal fat.[1] If the fatty mass in the right cardiodiaphragmatic angle is not homogeneous and/or contains areas of soft tissue density, other entities such as teratoma, thymolipoma, or liposarcoma must be considered.[1] The omental vessel sign is useful in the differential diagnosis of fatty masses in the cardiodiaphragmatic angle of the chest.

WILLIAM H. SUTRO, MD
Department of Radiology
Syosset Community Hospital
221 Jericho Tpke
Syosset, NY 11791

STEVEN J. KING, MD
Department of Radiology
Flushing Hospital and Medical Center
4500 Parsons Blvd
Flushing NY 11355

1. Naidich DP, Zerhouni EA, Siegelman SS: *Computed Tomography of the Thorax*. New York, Raven, 1984, pp 44-49.
2. Kurtz VB, Konig H, Walter E: Computed tomographic and sonographic diagnosis of diaphragmatic hernias (German). *ROFO* 1983; 139:502-510.
3. Modic MT, Janicki PC: Computed tomography of mass lesions of the right cardiophrenic angle. *J Comput Assist Tomogr* 1980; 4:521-526.
4. Kumar KL, Forse MA: Herniation through the foramen of Morgagni presenting as cardiomegaly. *South Med J* 1982; 75:894.

Tissue injury and breast carcinoma

TO THE EDITOR: Although the incidence of previous breast injury is greater in men than in women with cancer of the breast (29.6% vs 11.1%),[1] I agree with Drs Raviglione and Graham[2] that there has been no reliable scientific evidence to support the contention that injury to the breast is an initiating factor in carcinogenesis.[3] Their report is interesting in that it raises a number of intriguing questions. Among these is the relationship of nipple obstruction (not the injury itself) and the associated stasis of breast secretions, which has previously been invoked to explain the high incidence of breast cancer in such patients.[4] Ample experimental evidence exists to document an increased incidence of tumors in occluded glands.[5-7] Likewise, in animals given 3-methylcholanthrene—a carcinogenic compound—the incidence of mammary carcinoma was higher in nipple-excised glands.[8,9]

Although no mention is made of any prostatic symptoms or findings, nothing in this case report can repudiate the possibility of metastatic prostatic carcinoma to the breast. This has been reported in the past and is not unlikely, particularly among elderly men.[10] Chronic tissue injury may well act as a co-carcinogen, as has been shown recently in experimental animals.[11] Finally, evidence is accumulating to suggest that any form of tissue injury may facilitate the lodgement and growth of tumor cells at injured sites. Endothelial damage, retardation of the circulation, and hypercoagulability of the blood, all induced by local tissue injury, may play a role in carcinogenesis. The forcing of tumor cells into the circulation during surgical manipulation and the promotion of cancer growth by factors that stimulate repair after injury may all favor the entrapment of circulating cells as well as stimulate their growth at the site of injury. This is an important consideration and, if shown to be true—as has recently been suggested[12]—could mean that operative manipulations themselves may enhance local recurrence.

A. N. PAPAIOANNOU, MD
Program for the Treatment and
Prevention of Breast Problems
Mt Vernon Hospital
Mt Vernon, NY 10550

1. Donegan WL, Perez-Mesa CM: Carcinoma of the male breast. A 30-year review of 28 cases. *Arch Surg* 1973; 106:273-279.
2. Raviglione MC, Graham PE: Breast carcinoma in a man following local trauma. *NY State J Med* 1987; 87:186-187.
3. Papaioannou AN: Trauma, in Papaioannou A (ed): *The Etiology of Human Breast Cancer; Endocrine, Genetic, Viral, Immunologic and Other Considerations*. New York, Springer-Verlag, 1974, pp 155-156.
4. Adair FE, Bagg HJ: Breast stasis as the cause of mammary cancer. *Internat Clinics* 1925; 4:19-26.
5. Bagg HJ: Experimental status concerning the functional activity of the breast in relation to mammary carcinoma in mice. *J Cancer Res* 1925; 9:498-502.
6. Bogen E: Cause of breast cancer. *Am J Public Health* 1935; 25:245-250.
7. Fekete E, Green CV: The influence of complete blockage of the nipple on the incidence and location of spontaneous mammary tumors in mice. *Am J Cancer* 1936; 27:513-515.
8. Marchant J: Chemical induction of breast tumors in mice of the C57B1 strain. The influence of pseudopregnancy, pregnancy and lactation on induction by methylcholanthrene. *Br J Cancer* 1961; 15:568-573.
9. Biancifiori C, Bonser GM, Caschera F: Chemically induced mammary tumours following unilateral excision of the nipples in pseudopregnant and lactating breeding BALB/C mice. *Br J Cancer* 1962; 16:232-237.
10. Cambell TH, Cummins SD, Kirk DL, et al: Secondary breast cancer of prostatic origin. *JAMA* 1962; 179:458-460.
11. Konstantinidis A, Smulow JB, Sonnenschein C: Tumorigenesis at a predetermined oral site after one intraperitoneal injection of *N*-nitroso-*N*-methylurea. *Science* 1982; 216:1235-1237.
12. Papaioannou AN: Hypothesis: Increasingly intensive locoregional treatment of breast cancer may promote recurrence. *J Surg Oncol* 1985; 30:33-41.

In reply. Dr Papaioannou's letter is certainly interesting and proposes a number of possible explanations for the development of breast carcinoma following local trauma as it occurred in the case we reported.[1] For purposes of completeness we would like to say that the initial work-up of our patient also addressed the remote possibility of a prostatic neoplasia. At that time, we were dealing with a metastatic involvement of the axillary lymph nodes without an obvious primary cancer. However, the rectal examination showed prostatic hypertrophy and the subsequent prostate biopsy revealed "adenofibromyomatous hyperplasia." Thus, we concluded that metastatic prostatic carcinoma was extremely unlikely.

In his final statement, Dr Papaioannou also suggests that even a minor tissue injury may facilitate the implantation of tumor cells at injured sites and he speculates that operative manipulations may favor local cancer cell growth. However, we cannot propose this mechanism as the causative event in our patient; the time interval does not seem to support such a possibility.

We conclude that an obvious trauma to the left breast in the patient we described had occurred years before and that subsequently a cancer developed in the injured area. Again, a number of similar such cases are required in order to sustain the value of this observation.

MARIO C. RAVIGLIONE, MD
PETER E. GRAHAM, MD
Department of Medicine
Cabrini Medical Center
227 E 19th Street
New York, NY 10003

1. Raviglione MC, Graham PE: Breast carcinoma in a man following local trauma. *NY State J Med* 1987; 87:186-187.

Subcutaneous nonparenchymal hemangioma of the breast

TO THE EDITOR: Venous malformations, while common in many parts of the body, rarely occur in the breast. This report describes the clinical, radiographic, and pathologic features of a subcutaneous nonparenchymal venous malformation of the breast in a patient seen at the New York University Medical Center.

Case Report. A 39-year-old woman was seen in consultation for evaluation of a lump in the upper outer quadrant of her right breast, adjacent to the axilla. The lesion had been present for eight years, during which time it had slowly increased in size. There was no fluctuation in size with either menarche or pregnancy. Following examinations by various physicians, she had been advised that the lump represented either ectopic breast tissue or lymph nodes.

Physical examination revealed a rubbery, mildly tender, 6-cm mass in the right breast, which was slightly fluctuant, with the overlying skin intact and normal in color. The

FIGURE 1. Mammogram with an axillary view of right breast demonstrates well-circumscribed multilobulated mass with sharp margins. Several coarse calcifications are phleboliths.

remainder of the examination was unremarkable.

Low-dose film screen mammography demonstrated a multilobulated mass in the right breast which measured 6 × 3 cm, with well-defined margins. There were coarse calcifications consistent with the appearance of phleboliths (Fig 1). The mass was excised and was found to be a soft multilobulated tumor with hemorrhagic cavities present. On microscopic examination, the tumor was found to be a hemangioma, predominantly venous, with minor cavernous and capillary components (Figs 2, 3). It was located in fat, with no breast ductal or lobular elements identified in the surrounding tissues.

Discussion. Venous angioma of the breast is an unusual tumor which may be diagnosed in any age group, and in some cases is present at birth. At a later date, for unclear reasons, the lesion may suddenly enlarge, and the patient presents with a mass. It is typically a painless, soft to rubbery lesion, which is movable and may change in size with pressure. It may also pulsate. These tumors arise in connective tissue, and generally have round or irregularly shaped cavities containing liquid or coagulated blood separated by fibrous septa. They are identical to hemangiomas arising from other organs. The mammographic appearance of the tumor in the patient described corresponds to the findings observed on pathologic evaluation: a well-defined, multilobulated area of increased density and associated coarse calcifications representing the sanguinous cavities and phleboliths. It is not uncommon for venous hemangiomas to exhibit areas that are indistinguishable from cavernous hemangiomas. In the patient described here, although the tumor was predominantly venous, minor capillary and cavernous elements were present.

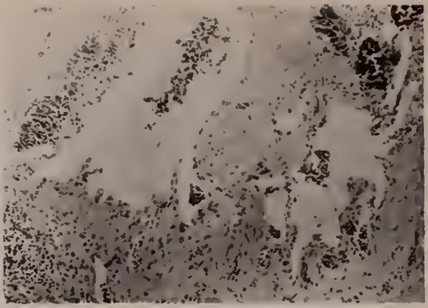


FIGURE 2. Most of the lesion consisted of large, thick-walled vascular spaces shown here. (There were minor cavernous and capillary components, not illustrated.) Hematoxylin-eosin stain; original magnification × 220.

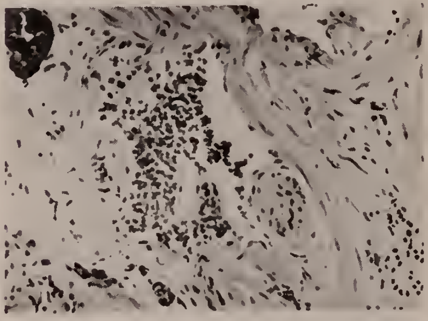


FIGURE 3. The walls of the vascular spaces contain prominent smooth muscle, confirming the presence of veins. Hematoxylin-eosin stain; original magnification × 400.

Recently, attention has been directed to distinguishing subcutaneous from intraparenchymal vascular tumors of the breast. The majority of intraparenchymal lesions frequently prove to be angiosarcomas, whereas the subcutaneous hemangiomas are benign. The latter do not recur or metastasize, and the treatment is surgical excision. Angiosarcomas recur locally and metastasize to other organs. Emphasis has been placed on the importance of complete excision of the hemangioma to verify that it is not a subcutaneous extension of an angiosarcoma, in which case mastectomy is indicated. The size of the lesion (6.3 cm) in the patient presented here is unusual, since vascular lesions larger than 2 cm often prove to be angiosarcomas. However, size is not as important as location when assessing the malignant potential of a nonparenchymal vascular lesion of the breast. The differential diagnosis also includes lymphangiosarcoma of the breast, which along with angiosarcoma has a poor prognosis.

JULIE S. MITNICK, MD
MATTHEW HARRIS, MD
HELEN FEINER, MD
New York University Medical Center
550 First Ave
New York, NY 10016

1. Rodman W: *Diseases of the Breast*. Philadelphia, P. Blakiston's Son & Co, 1908, pp 155–159.

2. Jozefczyk MA, Rosen PP: Vascular tumors of the breast. II. Perilobular hemangiomas and hemangiomas. *Am J Surg Pathol* 1985; 9:491–503.

3. Enzinger FM, Weiss SW: *Soft Tissue Tumors*. St Louis, CV Mosby, 1983, p 391.

4. Rosen PP: Vascular tumors of the breast. V. Nonparenchymal hemangiomas of mammary subcutaneous tissues. *Am J Surg Pathol* 1985; 9:723–729.

5. Gulesserian HP, Lawton RI: Angiosarcoma of the breast. *Cancer* 1969; 24:1021–1026.

6. Donnell RM, Rosen PP, Lieberman PH, et al: Angiosarcoma and other vascular tumors of the breast. *Am J Surg Pathol* 1981; 5:629–642.

INFECTIOUS DISEASES IN CHILD DAY CARE: MANAGEMENT AND PREVENTION

Edited by Michael T. Osterholm, Jerome O. Klein, Susan S. Aronson, and Larry K. Pickering. 175 pp, illustrated. Chicago, University of Chicago Press, 1987. $30.00 (hardcover)

The first day nursery for children opened in Boston in the early 1800s. Since then, the number of day care facilities in the United States has increased dramatically. Currently, approximately half of the women whose children are too young to care for themselves are in the labor force, and this number is expected to increase to 75% by the end of the century. While much attention has been paid to the social and economic implications of expanding day care, until recently there has not been much concern about the infectious diseases associated with the day care setting.

Why should infectious diseases be a problem in the day care setting? Attack rates of common respiratory and enteric diseases as well as severe conditions such as *Haemophilus influenzae* type b meningitis are highest in young children. In addition, evidence is accumulating that these infections occur more frequently in children in day care centers than in their home-bound peers. With increased recognition of disease spread in these settings comes greater interest by regulatory authorities.

The material presented in this monograph contains the proceedings of a symposium held in June 1984. Much of what is presented here previously appeared in 1986 in *Review of Infectious Diseases* (vol 8, no. 4). The purpose of this symposium was to characterize infectious diseases in child day care and to develop appropriate measures for prevention and management. The material was divided into five thematic sections, each containing from two to 12 presentations.

The first section provides an overview of the growth of child day care and the benefits and drawbacks of these programs, as well as a brief description of some of the infectious diseases associated with day care. The section entitled "Clinical and Epidemiologic Aspects of Infectious Diseases in Child Day Care" is the longest and most detailed. In this section, specific syndromes (such as acute respiratory infections and infectious diarrhea) and pathogens (*Haemophilus influenzae*, cytomegalovirus) are addressed, as well as methods for preventing the spread of these diseases in day care populations. Appropriately, there is also a paper addressing the issue of human immunodeficiency virus transmission in this setting. The third section reviews methods of surveillance of communicable diseases in day care settings, as well as policies for dealing with ill children. The fourth section presents two papers dealing with the regulatory environment that currently exists, as well as liability issues. The fifth section presents a number of papers dealing with parental and day care worker training.

This text is a long overdue contribution to a rapidly emerging public health issue, and appropriately deals not only with the microbiologic and epidemiologic implications of infections in the day care setting, but also the social and legal ones. Information is presented in a clear and comprehensive manner and, most importantly, the text deals with the practical issues that arise when communicable disease occurs in the day care setting.

This book is an excellent summary of the current state of knowledge of infectious diseases in day care and their implications. While this book should be required reading for pediatricians and infectious disease specialists, it would also be useful for day care center directors as well as governmental, regulatory, and public health authorities.

JACK A. DEHOVITZ, MD
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

PATHOLOGY OF NEOPLASIA IN CHILDREN AND ADOLESCENTS

By Milton Finegold, MD. 512 pp, illustrated. Philadelphia, WB Saunders Co, 1986. $60.00 (hardcover)

The purpose of this book is to provide a review of the pathology of all major childhood tumors with an updated analysis of recent progress in classification, cytology, and histochemical techniques. In addition, there is a brief review of some of the hereditary, cytogenic, and host factors in the etiology and development of pediatric malignancies. The authors contributing to the 17 chapters in this text are well qualified and thoroughly informed in their subject. The text has great usefulness not only to the pediatric pathologist, but also to general pediatricians, internists, radiotherapists, surgeons, and medical students, as well as pediatric hematologists/oncologists.

The chapters in this text cover the spectrum of pediatric malignancy, including leukemia, lymphoma, solid tumors, and brain and bone tumors. In addition, three chapters are devoted to the hereditary, genetic, and immunodeficiency syndromes associated with childhood cancer. Six of the chapters are outstanding in their scope and content. Chapter 4 on leukemia in childhood is extremely well done, with a superb bibliography. There are excellent tables describing the histologic changes associated with leukemia therapy, extramedullary involvement, and organisms isolated in children with leukemia, and there is an excellent summary of the commonly used marker studies in the monoclonal antibody evaluation of leukemia subtypes. Chapter 5 on malignant lymphoma in children is an excellent and concise review, with extremely clear figures of high quality. One figure will be extremely useful in assisting the pathologist in dividing the tissue for the complicated studies now underway in childhood lymphoma. There is an excellent table reviewing the morphologic, cytochemical, and immunotypic features of childhood lymphoma. Chapter 7 on neuroblastoma, Ewing's sarcoma, and the differential diagnosis of small-, round-, blue-cell tumors is a masterful treatise by one of the world authorities on this subject. Unfortunately, the figures in this particular chapter are too light, which detracts from the text. Table 7-7 is an excellent summary of the dilemma facing the pathologist in the diagnosis of small and round tumors in children. Chapter 13 on the primary hepatic tumors in childhood offers an excellent review of the subject, with a superb bibliography. The figures in this chapter are excellent. Table 13-1 summarizes the major reviews of primary liver tumors in children as reported in the medical literature. Chapter 16, entitled "Retinoblastoma: A Prototype of Childhood Malignancy," is a short but very concisely written and well-referenced chapter. The treatment of the pathology of the Flexner-Wintersteiner rosette is especially well done. The discussion of the genetics involved in this disease is current, with pertinent references to recent molecular genetic insights into this malignancy of neural crest origin. Last but not least among the outstanding chapters in this book is chapter 17, which describes the complications of treatment of neoplasia, including secondary malignancies. This is an extremely well-referenced and comprehensive chapter which is appropriately included in this text. Late effects are the harvest which follows the cure of childhood malignancy. Increased attention will be focused on this area in the future. This chapter provides an excellent background for this enormous challenge which almost equals the challenge of childhood malignancy itself. It is estimated that by the years 1990–2000 one in every thousand young adults will be survivors of childhood malignancy.

Chapters on oncogenes, cytogenetics, and immunodeficiency in childhood cancer are very brief and overly capsulized. Chapter 9 on soft-tissue neoplasms in childhood has excellent figures but desperately needs a table to summarize the field and provide an overview. Chapter 10 dealing with skeletal tumors in childhood and adolescence has an excellent table dealing with the differential diagnosis of cartilaginous tumors. Gonadal and extragonadal germ cell neoplasms-teratomas in childhood are reviewed in chapter 11. A table in this chapter provides an excellent summary of the sites for germ cell tumors. Chapter 12 on Wilms' tumor and other renal tumors of childhood has excellent figures and furnishes the reader with a holistic view in Figure 12-1. The chapter suffers from being too brief. It might have been well to reiterate the prognostic factors related to Wilms' tumors, although the authors note this has been described in other publications. A discussion of mesoblastic nephroma is included as an addendum to this chapter, making it appear as though the subject had been almost forgotten. The astrocytic tumors in childhood are reviewed in a superb manner, with an excellent figure showing the distribution of high- and low-grade astrocytomas. A chapter is also devoted to primitive neuroepithelial tumors of the central nervous system. This tumor system is very complicated and becoming more so as electron microscopic data is available. Overlap with extra-osseous Ewing sarcoma occurs as this classification is redefined. The authors have managed an excellent review of this complex and changing field.

In summary, this is an excellent text with several outstanding chapters. There are no major weaknesses in the text except for the inclusion of addenda at the end of some chapters. This practice is rather disconcerting and results in considerable loss in continuity. Nonetheless, this is an excellent text which belongs on the shelf of every student of childhood malignancy.

FREDERICK B. RUYMANN, MD
Children's Hospital
Columbus, Ohio 43205

HANDBOOK OF PEDIATRIC CARDIOLOGY

By Allan H. Rees, MD, Robert E. Solinger, MD, and Francisco Elbl, MD. 179 pp, illustrated. St Louis, Mo, Walter H. Green, Inc, 1987. $23.75 (softcover)

The authors have set out, as stated in the preface, to create a handbook of pediatric cardiology for nurses, technicians, and allied health professionals. The authors are certainly well qualified to cover the field. However, a careful review of this volume indicates that they have lost sight of their goal at times. There are areas where the coverage is too superficial, and others where there is far too much detail. For example, the section on genetics and congenital heart disease could have benefited from more information concerning the incidence of cardiac defects and their relative frequency compared to other major malformation groups. A brief discussion concerning the utility and goals of genetic counseling would be helpful for nurses and others with parental contact. In this context, perhaps the advantages and limitations of prenatal diagnosis by ultrasound should have been mentioned. Conversely, the section on cardiac catheterization provides substantial detail on the method of introducing catheters, which is probably lost on technical personnel. It could have been more informative by dealing with the indications for catheterization in an organized fashion, the importance of obtaining precise hemodynamic and angiographic data, and how these parameters are integrated to produce a diagnostic study. (Special procedures such as interventional techniques are dealt with briefly and electrophysiologic studies are not mentioned.)

The section on flow, shunt, and resistance calculations may be misleading. The formula for systemic and pulmonary blood flow does not provide the unit of measure for the O_2 content. As given, if mL/100dL is used, then the flow calculation will be in dL/min. Further, it is a serious error not to indicate that the units of vascular resistance are indexed (ie, reported as resistance units/m^2 body surface area). If this is not provided for in the calculations, erroneous values will be derived, especially for younger children.

While the text is logically organized, nurses and technical personnel might have found a unified section on cardiac anatomy and physiology easier to assimilate; this would allow one to find normal values for chamber pressures and hemodynamic calculations quickly (these are not identified in the index). The section on embryology is sparse. The ability to derive meaning from a description of the development of the aortic arch system without diagrams is limited. The sections dealing with the specific lesions from the diagnostic and surgical points of view should be very informative and helpful for the nurse and technician. I believe the excellent illustrations are supported by just the right amount of narrative. There is some duplication between the two sections but it is not significant.

The section on the management of newborns with critical cardiovascular disease lacks a discussion of prostaglandin use and transport team problems. The coverage of congestive heart failure is excessively long and does not provide clear insights into some therapeutic interventions. By contrast, the section on special problems is excellent.

The volume contains a number of grammatical and typographical errors, and in some instances the illustrations are either inappropriate or poorly reproduced. The lack of a comprehensive index to the volume is a drawback to its use.

Well-informed nurses and allied health professionals, cognizant of the book's overall therapeutic goals as well as its limitations, will better serve both the patient and family. Thus, I think the authors have identified a real need in pediatric cardiology. However, it seems evident that the authors have strayed from their stated purpose. This handbook would have profited from the collaboration of a nurse-practitioner or an intensive care nursery transport nurse, as well as a respiratory therapist.

ROBERT L. GINGELL, MD
Children's Hospital of Buffalo
Buffalo, NY 14222

SPECIAL CARE. MEDICAL DECISIONS AT THE BEGINNING OF LIFE

By Fred M. Frohock. 263 pages. Chicago, University of Chicago Press, 1986. $19.95 (hardcover)

If you can read only one book this year, do not let it be this one. While it has some redeeming features, it was clearly not written for physicians. If this review were not appearing in a medical journal it might read differently. But the time, energy, and effort required to cover the 263 pages are not sufficiently rewarded. The most significant contribution of the book is the last chapter, in which the author attempts to clarify the concepts of rights and harm and illustrates, at least in the neonatal setting, why it is more useful and appropriate to base therapy decisions on considerations of harm rather than on those of rights.

The purpose of the book seems two-fold: first, to convey to the reader what it is actually like in a neonatal intensive care unit (NICU) in terms of the atmosphere, milieu, or ambience; and second, to convince the reader that the use of language about rights of patients should be replaced by the use of language about harm to or interests of patients. Because of the two-fold nature of the book, perhaps it would have been better if the author had just written two books. The first purpose of the book is of little value or interest to most physicians, many of whom have a more thorough and accurate understanding of neonatal intensive care units than does the author. The second purpose is of immense importance and interest to physicians. However, it is not worth wading through all the unnecessary verbiage to ferret out the value of the use of harm language over that of rights language. No doubt Professor Frohock is qualified to write such a book. His reflections as a "participant" observer in a neonatal intensive care unit seem to describe clearly and accurately what they are really like. Furthermore, the data gathered support his contention that the concept of harm is more useful and appropriate than the concept of rights for making therapy decisions. However, the format of the book, which consists of journal entries interspersed with transcripts of interviews of people involved in various NICU dramas, is unengaging and does not serve the author's intended purposes. Perhaps for a lay audience this style would be more acceptable or even desirable. It could make more real a place with which they are completely unfamiliar. But for physicians it serves no useful function. As a result the book's usefulness as an addition to the medical literature is rather limited. This is extremely unfortunate because the book has a significant contribution to make through its discussion of the role of the concept of rights versus that of harm. In the last chapter, where this important distinction is set out with some clarity and force, there are other important insights that the author alludes to but inadequately develops, such as a list of useful virtues in medicine which "include honesty, tolerance, openness, a capacity to employ fair arguments and use facts honestly and a willingness to compromise." An elaboration and analysis of this list would be infinitely more useful and interesting to physicians than a description of a neonatal intensive care unit and the people in it. Indeed the last chapter has the seeds of an entire book in itself. As a result, I would recommend the last chapter of the book to all physicians, but not the entire book.

DONNIE J. SELF, MD
Texas A&M University
College of Medicine
College Station, TX 77843

TRAUMA IN CHILDREN

Edited by Randall E. Marcus, MD. 288 pp, illustrated. Rockville, Md, Aspen Publishers, Inc, 1986. $54.00 (hardcover)

The unusual feature of *Trauma in Children* is that the majority of its authors are orthopedic surgeons and the predominant emphasis is on orthopedic management of skeletal injuries. For completeness, the editor has included appropriate chapters on other aspects of injury in children. These chapters are well handled by established authorities in their respective fields. However, these authors practice at other children's hospitals; the lack of close professional relationships in a common hospital detracts from both the continuity of the text and its development of an overall system of trauma care. It is surprising and unfortunate that the management of minor trauma which is not life threatening is not addressed. Neither is there a discussion of the role of the pediatric emergency physician in the overall management of the child. The chapter on initial management of pediatric trauma, by an orthopedic surgeon, is adequate, but fails to consider that an orthopedic surgeon generally would not be involved in the initial treatment of multiple systems injury in children. It would have seemed logical to have either a general pediatric surgeon or someone actually involved in this phase of care write this chapter. In general, the discussion of the management of orthopedic injuries in children is excellent, authoritative, and well written, but some of the other chapters are pedestrian in content and cursory in scope. This is a good book for pediatric orthopedic surgeons and pediatric neurosurgeons, but will be of little help to general surgeons or pediatricians.

J. ALEXANDER HALLER, MD
The Johns Hopkins University
School of Medicine
Baltimore, MD 21205

CASE STUDIES IN PEDIATRIC EMERGENCY MEDICINE

Edited by Anthony J. Haftel, MD. 309 pp. Rockville, Md, Aspen Publishers, Inc, 1986. $33.50 (hardcover)

This book consists of 50 case studies of emergency conditions physicians encounter in busy pediatric emergency rooms. The editor is director of the Division of Emergency Medicine at the Children's Hospital of Los Angeles. Contributors include attendings, fellows, and residents in the division.

Each case study is divided into sections that include a title, chief complaints (provocative enough to stir one's diagnostic imagination), history, physical examination, suggested interventions, case discussion, and a suggested list of references.

The presentation is clear, concise, and organized, and topics are arranged according to age groups. They include sepsis, pyloric stenosis, near-miss sudden infant death syndrome, bronchiolitis, asthma, croup, epiglottitis, meningitis, sickle cell disease, diarrhea with dehydration, febrile seizure, accidental poisoning, child abuse, sexual abuse, appendicitis, testicular torsion, intracranial tumor, multiple trauma, cardiac arrhythmia, attempted suicide, and adolescent pregnancy.

I find the list of suggested interventions in some cases long and unnecessary, eg, ordering erythrocyte sedimentation rate, blood culture, and urine culture tests in a straightforward case of pyloric stenosis. In general, the discussions are instructive although the depth of discussion on individual topics varies according to the individual contributor. Appropriately, the list of references is short.

There are some significant, albeit minor, technical errors. The patient described as Case Number 12 (p 12) entitled "ten month old male with vomiting and seizures" did not have seizures. On page 160, the statement, "He was discharged on oral antibiotics four days after admission with sequelae," should read "with no sequelae."

Overall, this is a book that should prove valuable to emergency room and pediatric intensive care unit physicians and nurses. It is also a good reference text for pediatric house officers in general. It is well worth the price.

TERESITA A. LAUDE, MD
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

THE DACHMAN DIET FOR KIDS. A COMPLETE GUIDE TO HEALTHY WEIGHT LOSS

By Ken Dachman. 224 pages. New York, World Almanac Publications, 1986. $15.95 (hardcover)

This book aims at showing parents how to help their young, obese children successfully undergo weight reduction and then maintain their lower weights. The author holds degrees in behavioral psychology and human physiology from the University of Illinois and is currently enrolled in its PhD program in psychology. He lectures, conducts workshops and seminars, and writes magazine articles and newspaper columns on the subject of weight reduction. He has established 15 weight loss centers in the Chicago area. He also holds special seminars for obese children. (He himself lost 250 pounds as an obese and unhappy adolescent and has apparently continued to maintain his reduced weight to this time.) However, as in many weight loss books, there is a dearth of statistics on long-term follow-up, successful and unsuccessful.

The book places great emphasis on the emotional aspects of overeating and weight control, and very little on the now more accepted contribution of genetic influences to obesity. The importance of exercise is addressed and this is a major strength of the book. However, there are disturbing statements to the effect that it is not necessary for a child to engage in a regimented program of exercise. Daily calisthenics are seen to overburden a child and can lead to diet failure in the opinion of the author, who contents himself with saying, "Exercise your child truly enjoys won't be seen as a chore." Thus, it is possible that parents and their children will get the wrong idea concerning exercise. Weight reduction diets without exercise simply do not work in the long run according to most studies. The book also assures the safety of an artificial sweetener whose safety is currently being scientifically debated.

The book's main theme is that overweight is a response to emotional hunger. Mr Dachman also states, "I feel a maintenance diet is counterproductive, even destructive. It gives your child the notion that he is still a fat person who temporarily happens to be thin. After all a maintenance diet is simply a reducing diet with several hundred calories added. If you had to consider the prospect of a structured diet for the rest of your life, I am sure you would come to the conclusion that losing weight simply was not worth the effort. Imagine how grim those endless years of deprivation stretching far into the future look to the child." However, we know from metabolic studies that truly obese young children will need to be on a low calorie diet for the rest of their lives, whatever the reasons are that make them less efficient in utilizing calories.

Pages 68 to 220 of the book provide menus at different calorie levels. This portion of the book was written with the help of Lorna M. Castellanos and Susan Swedo. Tables are provided to enable parents to determine how many calories their child needs. These are based on the low ideal weight which is then converted into kilograms and multiplied by slightly less than the expected number of calories per kilogram of body weight required for normal growth and development. Two hundred calories are then subtracted. In slightly overweight children, this approach should work and provide a slow weight loss, which might be as low as one third of a pound per week. However, available studies indicate that obese children will require even fewer calories to maintain their weight and therefore weight reduction may not be accomplished on the diet level suggested. Diet plans A through D as given provide a different range of calories. Mr Dachman suggests that if weight is not lost on one diet, the child should reduce his intake to the level of the next lower diet. This approach may or may not work depending on the number of calories required for weight loss.

An interesting concept is bonus calories, between 300 and 500 per day, that the child can use for any type of food desired. The menus show what foods can be eaten at each calorie level. The diets are basically well balanced with reasonable amounts of protein and are geared to the normal likes of children. Menus from fast food restaurants are provided which are useful for children undergoing weight reduction. The book generally has an upbeat view of encouraging children to lose weight which is not really borne out by long-term studies of overweight children. The genetic effects and the changing caloric needs with obesity are not well described. There is little scientific documentation of data.

FREDDA GINSBERG-FELLNER, MD
Mt Sinai School of Medicine
New York, NY 10029

AMA addresses key issues at annual meeting

At its annual meeting in June, the American Medical Association (AMA) recommended mandatory AIDS testing only for prison inmates, immigrants, military personnel, and donors of blood, organs, and other biological materials. Screening of other populations, including those at high risk of contracting AIDS, should be on a voluntary basis, and the findings should be confidential, according to the AMA's House of Delegates. The AMA also emphasized the need for antidiscrimination laws to protect persons with the AIDS virus, and for accuracy in filling out death certificates in cases of AIDS—rather than omitting the AIDS diagnosis to protect the patient's privacy and family.

In other actions, the AMA was three votes short of supporting a ban on television advertisements of alcohol products. Unlike its support of a ban on all advertising and promotion of tobacco products, the AMA is not prepared to commit to a firm position on alcohol advertising.

Further recommendations included the use of seat belts in taxis, and drug testing solely for employees whose work affects the health and safety of others, and whose job performance suggests a drug problem.

In support of the surgeon general's plan for a smoke-free society by the year 2000, the AMA voted in favor of legislation to raise the minimum age for purchasing tobacco products to 21 and proposed a ban on smoking for all commercial passenger planes.

The expanding role of women in medicine

By the end of 1985, 15% of all physicians were women. The number of women physicians had increased by nearly 200% since 1970, far surpassing the 66% rate of growth in the total number of practicing physicians. Forty-two percent of female physicians were under 35 years of age in 1985. They tended to work out of an office, rather than at a hospital, and many of them chose to practice medicine in New York, California, or Illinois. Data compiled by the American Medical Association (AMA) indicate that women physicians prefer the specialties of pediatrics, general practice, psychiatry, internal medicine, anesthesiology, obstetrics/gynecology, and pathology.

Despite the assimilation of women into all aspects of medical practice, women have not achieved representation in organized medicine proportionate to their growing numbers and, therefore, are able to play only a minor role in shaping the policies that will affect the future of the medical profession and of women in medicine. According to the AMA, 28% of active women physicians are members of organized medicine, as compared to 45% of male physicians.

To promote further participation of women in organized medicine and, specifically, in leadership positions, the AMA established the Women in Medicine Project in 1984. This initiative has included a nationwide membership drive, a survey of the attitudes of women physicians toward their state and county medical societies, the creation of the AMA Women Leaders Databank, and the publication of various resource materials. Project officials believe that "Organized medicine has much to gain from mainstreaming women members. It also has a lot to lose if their opinions are not represented." The number of women in medicine is growing. State, local, and specialty medical societies should actively promote the membership and participation of women physicians.

Educational and legal efforts needed to combat smoking by minors

Children who smoke or who are likely to develop the habit greatly underestimate their health risks from smoking, according to a study published in the June 26 issue of *JAMA* (1987; 257:3373). Investigators from the University of Wisconsin, Madison, evaluated the attitudes toward smoking of a group of students in grades two through 12. They found that although almost all of the students acknowledged that smoking is harmful, many felt themselves to be invulnerable to the health risks. In addition, the students overestimated the prevalence of smoking among their peers and among adults, while underestimating negative attitudes toward smoking. Misconceptions among the study group were also noted with regard to the addictive nature of tobacco products and the difficulty of breaking a nicotine habit. The authors stressed the need for educational programs to better inform children of the health hazards associated with smoking.

One means of discouraging minors from smoking would be to restrict the the availability of tobacco products, as 95% of adult smokers started their habit before the age of 20. Although legal measures to this effect do exist, researchers at the University of Massachusetts Medical School, Worcester, have shown poor compliance with the law (*JAMA* 1987; 257:3323). In Massachusetts, a law prohibiting the sale of tobacco to persons under 18 years of age has been in effect since 1985. But the authors documented the routine sale of cigarettes to an 11-year-old girl participating in the study. Noting both the lack of compliance with and enforcement of the law, the authors recommended severe legal action. "Violators should risk the loss of their license to sell tobacco."

The politics of smoking

In Italy, it appears that smoking, or rather an antismoking stance, and politics do not mix. In *The Lancet* of June 6, 1987 (1:1310), Italian correspondents chronicled the turbulent history of antismoking legislation in Italy during the previous year. In the spring of 1986, Signor Degan, the Minister of Health, proposed legislation that would have restricted or prohibited smoking in hospitals, schools, restaurants, public transportation, national airline flights, and any offices that would be open to the public. In addition, the legislation would have banned cigarette promotion by sponsorship, and would have required health education efforts and labelling of cigarettes with health warnings and tar and nicotine content.

NEWS BRIEFS are compiled and written by Vicki Glaser.

An independent survey of 1,515 Italians demonstrated support for the legislation by 76% of the sample, including 70% of smokers. Despite such positive public sentiment, which had been reported in a national daily newspaper, the Minister of Health was transferred to another position, and the proposed legislation was abandoned. The new Minister of Health has taken no action against smoking, believing it not to be a health priority.

The correspondents compare this sequence of events to the fate of other antismoking advocates: former US Secretary of Health Califano and Sir George Young, the junior Minister of the Health Department in Great Britain. They cite a previous comment by the British junior Minister: "Smoking is in every sense a political issue, and those politicians who concern themselves with it find themselves unexpectedly promoted or demoted."

CDC to study AIDS

In an effort to better understand the natural history of the human immunodeficiency virus, which causes acquired immunodeficiency syndrome (AIDS), the Centers for Disease Control (Atlanta) plans to follow up patients who acquired the AIDS virus through blood transfusions received between 1978 and 1985. Physicians with such patients who would like to participate in the study should contact Dr Holmberg, Bldg 6-274, Mailstop G-22, Centers for Disease Control, Atlanta, GA 30333; (404) 320-2718.

FDA revises drug regulations

The FDA's revised drug regulations will make experimental drugs more easily available to patients with serious or life-threatening diseases. The new regulations enable drugs that are under study in controlled clinical trials to be used by patients not involved in the trials.

The new FDA procedures for use of experimental drugs include several restrictions. For example, the patient must be fully informed of the drug's risks, and he or she must consent to the treatment. The manufacturer of the drug can charge for the product, but may not promote the sale of the drug. The drug must be in ongoing clinical trials, with the ultimate goal of receiving marketing approval. The sponsor of the drug—a pharmaceutical company, university, or governmental institution—must notify the FDA 30 days before making the drug available or charging for it and must distribute the drug only to licensed medical practitioners. The FDA can prohibit the distribution of an experimental drug if the agency's commissioner finds the drug's effectiveness to be in question or the associated risks to be unreasonable.

CDC reports increase in syphilis in US

The number of cases of syphilis reported in the US during the first three months of 1987 was 23% greater than the number of cases reported during the first three months of 1986 (*MMWR* 1987; 36:393). This increase, the largest in more than ten years, follows a five-year period of decreasing incidence of syphilis.

Furthermore, the composition of the affected population appears to have changed during the past decade. Whereas two thirds of male patients with syphilis in the 1970s were homosexual or bisexual, the current increase in syphilis cases is primarily affecting heterosexuals. During the first quarter of this year, California, Florida, and New York City reported the greatest increases in syphilis cases.

These alarming findings pose multiple problems for health care officials, including controlling the spread of congenital syphilis. In addition, persons with a history of sexually transmitted disease are at an increased risk of having been infected with human immunodeficiency virus (HIV), and officials fear that the increasing incidence of syphilis could precede further spread of HIV infection and AIDS among both homosexuals and heterosexuals.

New hope for treating skin cancer

Within a year, physicians at Fremantle Hospital (Australia) hope to test a new drug that has shown promise against skin cancer. The drug is a monoclonal antibody that recognizes and binds to melanoma cells. Although the antibody itself is not cytotoxic, it can carry a radioisotope, specifically, samarium 153, which destroys the melanoma cell. Such a drug could prove beneficial as an adjunct to surgical removal of skin cancer, especially deep tumors, by seeking out cancerous cells that may have spread to other parts of the body.

Ulcers linked to bacterial infection

Challenging the conventional belief that excessive stomach acid causes ulcers, Dr Barry Marshall, of Royal Perth Hospital in Australia, has proposed that most stomach ulcers result from infection with the spiral-shaped bacterium *Campylobacter pyloridis*. According to Marshall's studies, *C pyloridis* produces heightened sensitivity of the stomach and upper intestines to even normal levels of acid. He estimates that the organism is responsible for approximately 70% of stomach ulcers and 90–95% of duodenal ulcers.

If this theory proves accurate, new modes of treatment could be instituted. Marshall has shown that patients with ulcers who are treated with a combination of antibiotics and bismuth to control the infection are less likely to experience relapses. Critics of this theory explain the infection as a byproduct, rather than a cause, of ulcers.

The Food and Drug Administration has granted approval to the University of Virginia Medical School (Charlottesville)—where Marshall is a research fellow—for limited use of a breath test to detect ammonia released by *C pyloridis* in the stomach.

Symposium on beta blockers

"A Quarter-Century of Beta Blockade: Lessons From the Past, an Eye Toward the Future" was the title of a symposium this spring that focused on current uses and potential new applications for beta blockers. Participants learned that beta blockers offer multiple benefits when given intravenously for treatment of chest pain suggestive of a heart attack: they reduce the severity of heart attacks; reduce the proportion of patients who ultimately suffer a heart attack; reduce arrhythmia; and reduce the risk of early death, repeat heart attacks, and ventricular fibrillation. Beta blockers have also shown promise in the treatment of hypertension, symptoms associated with alcohol withdrawal syndrome, and premenstrual syndrome.

Voluntary testing for HIV infection

The Council approved a resolution concerning testing for human immunodeficiency virus (HIV) infection proposed by the Committee on Preventive Medicine. In its resolution the Council endorsed the expansion of HIV testing and encouraged the offering of testing to patients who are at high risk. Among the latter are those attending venereal disease clinics, sexual partners of intravenous drug abusers, pregnant women, women seeking family planning services in neighborhoods with a high incidence of AIDS infections, and certain persons who received blood transfusions or blood components within the period from 1978 to mid-1985. The Council's June 11, 1987, resolution also urges physicians to recommend that HIV-infected persons notify their sexual or IV-drug-using partners of the need to consider testing for HIV. Physicians are also urged to inform marital candidates about the availability of HIV testing for their personal decision making.

Facilitating enrollment of nonmember physicians

The Council recently approved a pilot program that will be undertaken in cooperation with interested county medical societies for facilitating qualified physician membership enrollment in the state and county medical societies. At regular intervals the Medical Society of the State of New York will obtain the names of duly licensed nonmember physicians in the State of New York and forward them to the appropriate county medical society; the county medical society will then have the duty of approving or disapproving the physicians for enrollment based on whatever manner of review their respective membership committee deems useful, such as (a) immediate approval based on personal knowledge of the physician; (b) review of AMA profile; (c) brief application; (d) formal application; and/or (e) interview. Having received the county approval, the MSSNY will advise each physician that he or she is now eligible to have his or her name placed on the membership rolls of the state and county medical societies, and ask the physician to either activate or decline the membership. Dues requirements will accompany the notification of eligibility for membership. This pilot program will be in effect for a two-year period, after which time its usefulness will be evaluated by the participating county societies and the MSSNY Committee on Membership.

Nursing shortages in hospitals

Donald F. Foy, Executive Vice-President of the Society, reported to the Council on June 11, 1987, that the nursing shortage in New York hospitals is creating havoc for managers trying to fill empty nursing positions and that the nursing crisis is likely to get worse. This has prompted questions about the quality of care being delivered in many hospitals.

Hospital head-hunting firms have increasingly turned to nursing schools in Canada, Ireland, the United Kingdom, Australia, and the Philippines. Those nations have English-speaking workers who are familiar with American medical practice and are eager for US salaries and benefits. The $20,000 US hospital entry salary is at least double the amount these nurses can expect to earn at home. More than 40 American hospitals had representatives at a Toronto job fair this year, up from eight the previous year.

Evidence suggests a significant number of foreign nurses are applying for US hospital jobs. A record 8,000 nurses applied to take the April 1 certification exam given by the Commission on Graduates of Foreign Nursing Schools (CGFNS), a group which tests English proficiency and nursing knowledge in 37 centers around the world. Potential nurse emigres must pass the exam to obtain an H-1 visa and other documents needed to work in American hospitals. H-1 status is good for a two-year stay as a nonresident alien, with extensions possible.

At the same time, the nursing lobby is pressuring federal lawmakers to remove Medicare restrictions that prohibit nurses from billing the government. Medicare currently pays for Part B services only with the approval of a physician. Legislation introduced by Rep Richard Gephardt (D-Mo) would allow "nursing organizations" to compete for a Medicare risk contract to provide certain Part B services to elderly beneficiaries. Those nursing groups would provide home health, physical therapy, and other nursing services to beneficiaries for a fixed payment. These providers would not offer physician, x-ray, or laboratory services but would be in a referral position. This is consistent with the American Nurses Association's (ANA) plans to secure an independent and more direct influential role for nurses in delivery of care in institutions, in the community, and in home care. Decision-making power is sought to replace the reported "waning influence of physicians and hospitals." Models or frameworks for the delivery of nursing services have been developed, and legislative strategies have been suggested to codify a new, independent, and autonomous role for nurses. This framework is set forth in a recent ANA publication entitled, "New Organizational Models and Financial Arrangements for Nursing Services."

The new power brokers who, it is claimed, are replacing physicians are fiscal intermediaries composed of employers' insurance companies and government. The objectives of their activities are to provide nursing practice models and payment arrangements to enable fiscal intermediaries to view "nursing therapy" as a high quality, cost effective substitute for physician services eligible for direct reimbursement. This was a topic of considerable discussion at a meeting Mr Foy attended in early June in Chicago of the Advisory Committee to the AMA Executive Vice-President. This matter was brought to the attention of the AMA House of Delegates at its recent meeting in order to alert the federation to state initiatives from organized nursing.

MEDICAL SOCIETY NEWS is compiled and written by Vicki Glaser.

Attention Delegates
Your Next Meeting

The 182nd Annual Meeting of the Medical Society of the State of New York will be held at the New York Hilton Hotel, New York City, on April 21–24, 1988

•

Nominating Committee, 1988

(The committee nominates Officers, Councilors, and Trustees of MSSNY, and Delegates to the American Medical Association.)

Member-at-Large—John A. Finkbeiner, M.D., New York, *Chairman*
First District—Stuart Orsher, M.D., New York
Second District—John A. Ostuni, M.D., Nassau
Third District—Gloria S. Aitken, M.D., Sullivan
Fourth District—John D. Fulco, M.D., Schenectady
Fifth District—Robert M. George, M.D., Oneida
Sixth District—Antonio F. LaSorte, M.D., Broome
Seventh District—James M. Flanagan, M.D., Wayne
Eighth District—Leo E. Manning, M.D., Erie
Ninth District—Seymour R. Stall, M.D., Dutchess
Member-at-Large—James H. Cosgriff, Jr., M.D., Erie

Residents/Fellows

A $500 AWARD will be presented by the Medical Society of the State of New York to the intern, resident or fellow who submits the best paper on a clinical subject for the Society's 1987 Merit H. Cash Competition.

Entrants need not be members of the MSSNY but must be in training at a hospital within the State of New York.

Entries should be "sponsored" by a faculty member, and all papers submitted should be of sufficient quality to be considered for original publication in the *New York State Journal of Medicine.*

The deadline for receipt of entries is November 1, 1987, and the prize for the winning paper will be awarded at the MSSNY's annual meeting in April of 1988.

Address all entries to: Division of Scientific Activities
Medical Society of the State of New York
420 Lakeville Road
P.O. Box 5404
Lake Success, NY 11042

Questions? Contact either the Division of Scientific Activities or MSSNY's Resident Physicians Section at (516) 488-6100.

In New York City, for the Newest and Finest Medical Space, Remember:

THE 4 ESSENTIAL REQUIREMENTS

1. LOCATION!
Liberty Residences at Battery Park City

2. LOCATION!
Windsor Pavilion
(3rd Avenue & 32nd Street)

3. LOCATION!
205 East 64th Street (3rd Avenue)

AND

4. LOCATION!
Normandie Pavilion
(3rd Avenue & 95th Street)

For Further Information Contact Lester Schwalb
(212) 708-0846
Milstein Properties Corp.

ORTHOPEDISTS

Tired of the hassles of practice?
Tired of the malpractice burden?

I need two additional full-time orthopedists to join our 15 man group. One for our facility in Syracuse, and one for Rochester.

We do evaluations only, no treatment.

Banker's hours, pleasant work, good remuneration. No nights or weekends.

If you are ready to give up surgery for a more placid life, send your C.V. to me:

Harvey Lewis, M.D.
3155 G. Eggert Road
Tonawanda, NY 14150

BUILD YOUR PRACTICE with a structured marketing and public relations program developed by experts in the medical field.

℞: PESIN PUBLIC RELATIONS

- Design all print materials — press releases, brochures, newsletters, direct mail pieces, ad copy
- Arrange interviews on TV and radio and in newspapers and magazines
- Produce direct mail pieces to send to patients and/or colleagues in your practice region
- Establish your presence at professional meetings/conventions

For a **FREE CONSULTATION** call:
Ella Pesin at 212-737-3344

PPR
PESIN PUBLIC RELATIONS

301 East 79th Street, Suite 26G
New York, N.Y. 10021

---

ecmc erie county medical center

MEDICAL DIRECTOR

The Erie County Medical Center, a major teaching hospital in Western New York, offers an excellent opportunity in a 490-bed, acute care, and 120-bed Skilled Nursing Facility in a public hospital in Buffalo, New York.

The Medical Director will report to the Chief Executive Officer and Board of Managers and will plan activities designed to enhance the medical staff and recruit members in the community to ensure continuing excellence in medical care.

Candidates must possess board certification in a medical specialty and have four (4) years of post-residency experience in a medical specialty, two (2) years of which must have been in an administrative/supervisory capacity.

If interested, send resume outlining your employment history and salary requirements to George McCoy, Chief Executive Officer, 462 Grider Street, Buffalo, NY 14215.

ECMC is an AA/EO Employer.

FOR SEVERE COUGHS, CAN YOU REMEMBER THESE LETTERS?

You know Robitussin®, and the "DAC" formula gives you just what you need to manage those severe coughs complicated with nasal congestion . . . Decongestant And Codeine!

Robitussin®-DAC ⓥ

Each 5 ml (1 teaspoonful) contains:

Guaifenesin, USP	100 mg
Pseudoephedrine Hydrochloride, USP	30 mg
Codeine Phosphate, USP (Warning: May be habit forming)	10 mg

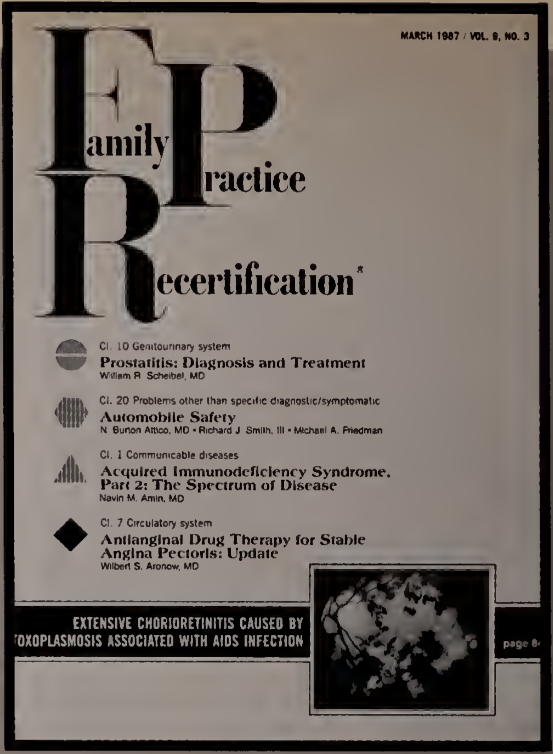
Alcohol 1.4 percent

℞ ONLY

A·H·ROBINS
Pharmaceutical Division
Richmond, Virginia 23261-6609

The complete journal for family practice physicians

- Reaches 79,000 family physicians monthly
- Presents the most commonly seen patient problems in family practice
- Written by physicians for physicians
- The most current clinical updates in:
  - Cardiology
  - Diabetes
  - Pediatrics
  - Ob/Gyn
  - Psychiatry
  - Gastroenterology
- Provides 20 hours of CME Category 1 Credit



MARCH 1987 / VOL. 9, NO. 3

Family Practice Recertification®

Cl. 10 Genitourinary system
Prostatitis: Diagnosis and Treatment
William R. Scheibel, MD

Cl. 20 Problems other than specific diagnostic/symptomatic
Automobile Safety
N. Burton Attico, MD • Richard J. Smith, III • Michael A. Friedman

Cl. 1 Communicable diseases
Acquired Immunodeficiency Syndrome, Part 2: The Spectrum of Disease
Navin M. Amin, MD

Cl. 7 Circulatory system
Antianginal Drug Therapy for Stable Angina Pectoris: Update
Wilbert S. Aronow, MD

EXTENSIVE CHORIORETINITIS CAUSED BY TOXOPLASMOSIS ASSOCIATED WITH AIDS INFECTION

page 8

PRACTICAL ▪ CLINICAL ▪ EDUCATIONAL ▪ CURRENT

It's Here At Last...
A Choice of Two Low-Cost Practice Evaluations.

Basic Practice Evaluation
Provides a benchmark for your practice.

Expanded Option Evaluation
Provides 15 different option categories, with 180 evaluations, under a variety of conditions.

Here's Why You Should Consider a Pro-Tech Practice Evaluation:

- Analysis can be used in buying, selling, merging or dissolving of a practice.
- Analysis can serve to confirm other appraisals.
- Analysis is processed quickly — usually within 10 days.

Get the hard facts.

Name ______________ Phone ______________
Address ______________
City ______________ State ______ Zip ______

Pro-Tech Information Systems℠
© 1986 All Rights Reserved

240 Cedar Knolls Rd., Suite 310, Cedar Knolls, NJ 07927-9990

Exclusive Marketing Agents: Sarantos & Co., Inc.

DOCTOR
DRUG
DISPENSING

"Catching On"

- Greater convenience & savings for patients
- More effective patient treatment
- A new profit center. . . no additional overhead
- Computerized labeling, inventory and ordering

For more information please call or write:

DOCTORS'

DOCTORS' Rx
145 Algonquin Parkway
Whippany, NJ 07981
(201) 428-1117
1 (800) 752-5588

Patients and physicians alike prefer

Transderm-Nitro®
nitroglycerin

Over half a billion* patches prescribed in the U.S. since 1982

The only patch with a rate-limiting membrane

Familiar, distinctive tan color and unique shape recognized by patients everywhere

There's no substitute for experience

CIBA

(See Brief Summary of Prescribing Information on the next page.)

*Data on file, CIBA Pharmaceutical Co.

629-7750-A

Transderm-Nitro®
nitroglycerin
Transdermal Therapeutic System

BRIEF SUMMARY (FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE INSERT)

INDICATIONS AND USAGE
This drug product has been conditionally approved by the FDA for the prevention and treatment of angina pectoris due to coronary artery disease. The conditional approval reflects a determination that the drug may be marketed while further investigation of its effectiveness is undertaken. A final evaluation of the effectiveness of the product will be announced by the FDA.

CONTRAINDICATIONS
Intolerance of organic nitrate drugs, marked anemia, increased intraocular pressure or increased intracranial pressure.

WARNINGS
In patients with acute myocardial infarction or congestive heart failure, Transderm-Nitro system should be used under careful clinical and/or hemodynamic monitoring.
In terminating treatment of anginal patients, both the dosage and frequency of application must be gradually reduced over a period of 4 to 6 weeks to prevent sudden withdrawal reactions, which are characteristic of all vasodilators in the nitroglycerin class.
Transdermal nitroglycerin systems should be removed before attempting defibrillation or cardioversion because of the potential for altered electrical conductivity which may enhance the possibility of arcing, a phenomenon associated with the use of defibrillators.

PRECAUTIONS
Symptoms of hypotension, such as faintness, weakness or dizziness, particularly orthostatic hypotension may be due to overdosage. When these symptoms occur, the dosage should be reduced or use of the product discontinued.
Transderm-Nitro system is not intended for immediate relief of anginal attacks. For this purpose occasional use of the sublingual preparations may be necessary.

ADVERSE REACTIONS
Transient headaches are the most common side effect, especially when higher doses of the drug are used. These headaches should be treated with mild analgesics while Transderm-Nitro therapy is continued. When such headaches are unresponsive to treatment, the nitroglycerin dosage should be reduced or use of the product discontinued.
Adverse reactions reported less frequently include hypotension, increased heart rate, faintness, flushing, dizziness, nausea and vomiting. These symptoms are attributable to the known pharmacologic effects of nitroglycerin, but may be symptoms of overdosage. When they persist the dose should be reduced or use of the product discontinued. In some patients, dermatitis may occur.

DOSAGE AND ADMINISTRATION
Therapy should be initiated with application of one Transderm-Nitro 5 system to the desired area of skin. Many patients prefer the chest; if hair is likely to interfere with system adhesion or removal, it can be clipped prior to placement of the system. Each system is designed to remain in place for 24 hours, and each successive application should be to a different skin area. Transderm-Nitro system should not be applied to the distal parts of the extremities.
The usual dosage is one Transderm-Nitro 5 system every 24 hours. Some patients, however, may require the Transderm-Nitro 10 system. If a single Transderm-Nitro 5 system fails to provide adequate clinical response, the patient should be instructed to remove it and apply either two Transderm-Nitro 5 systems or one Transderm-Nitro 10 system. More systems may be added as indicated by continued careful monitoring of clinical response. The Transderm-Nitro 2.5 system is useful principally for decreasing the dosage gradually, though it may provide adequate therapy for some patients when used alone.
The optimal dosage should be selected based upon the clinical response, side effects, and the effects of therapy upon blood pressure. The greatest attainable decrease in resting blood pressure that is not associated with clinical symptoms of hypotension especially during orthostasis indicates the optimal dosage. To decrease adverse reactions, the size and/or number of systems should be tailored to the individual patient's needs.
Do not store above 86°F (30°C).

PATIENT INSTRUCTIONS FOR APPLICATIONS
A patient leaflet is supplied with the systems.

HOW SUPPLIED

Transderm-Nitro System Rated Release *in vivo*	Total Nitroglycerin in System	System Size	Carton Size
2.5 mg/24 hr	12.5 mg	5 cm²	30 Systems (NDC 0083-2025-26) *100 Systems (NDC 0083-2025-30)
5 mg/24 hr	25 mg	10 cm²	30 Systems (NDC 0083-2105-26) *100 Systems (NDC 0083-2105-30)
10 mg/24 hr	50 mg	20 cm²	30 Systems (NDC 0083-2110-26) *100 Systems (NDC 0083-2110-30)
15 mg/24 hr	75 mg	30 cm²	30 Systems (NDC 0083-2115-26) *100 Systems (NDC 0083-2115-30)

*Hospital Pack 100's

C85-35 (Rev. 11/85)

Dist. by:
CIBA Pharmaceutical Company
Division of CIBA-GEIGY Corporation
Summit, New Jersey 07901

All transdermal nitroglycerin products are being marketed pending final evaluation of effectiveness by the FDA.

© 1986, CIBA 629-7750-A C I B A

(continued from p 9A)

Postgraduate Course-Radiology. 28 Cat 1 Credits. Hotel del Coronado, Coronado (San Diego). Contact: Dawne Ryals, Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

Oct 26–31. **38th Annual Workshops and Scientific Program of the Society for Clinical and Experimental Hypnosis.** 46 Cat 1 Credits. Ambassador Garden Hotel, Los Angeles. Contact: Marion Kenn, Administrative Director, The Society for Clinical and Experimental Hypnosis, 128A Kings Park Dr, Liverpool, NY 13090. Tel: (315) 652-7299.

COLORADO

Oct 5–10. **Family Practice Review.** The University of Colorado Health Sciences Center, Denver. Contact: University of Colorado School of Medicine, Office of Continuing Medical Education, 4200 E 9th Ave, Box C-295, Denver, CO 80262. Tel: (303) 394-5195.

DISTRICT OF COLUMBIA

Oct 15–18. **The Politics of Caring.** 14 Cat 1 Credits. J. W. Marriott Hotel, Washington. Contact: Barbara Lauter, American Society of Internal Medicine, 1101 Vermont Ave, NW, Suite 500, Washington, DC 20005-3457. Tel: (202) 289-1700.

Oct 30–Nov 1. **Second Annual Conference on Clinical Immunology.** Sheraton-Washington Hotel. Contact: Clinical Immunology Society, PO Box 827, Bowie, MD 20715.

FLORIDA

Oct 7–9. **Emerging Trends in Critical Care—1987.** 15 Cat 1 Credits. Crowne Plaza Hotel, Orlando. Info: Rick Mace, Executive Director, Florida Heart Institute, 500 E Rollins, Suite 101, Orlando, FL 32803. Tel: (305) 897-1575.

Oct 28–31. **28th Workshop in Electrocardiography.** 19 Cat 1 Credits. Sheraton Sand Key Hotel, Clearwater Beach. Contact: Anne Criss Taylor, Rogers Heart Foundation, 601 12th St, North, St Petersburg, FL 33705. Tel: (813) 894-0790.

GEORGIA

Oct 26–30. **53rd Annual Scientific Assembly of the American College of Chest Physicians.** Atlanta Hilton and Towers, Atlanta. Contact: Division of Education, American College of Chest Physicians, 911 Busse Hwy, Park Ridge, IL 60068-2375. Tel: (312) 698-2200.

HAWAII

Oct 10–18. **12th Annual International Body Imaging Conference.** 30 Cat 1 Credits. Maui. Info: Annual Body Imaging Conference, Department of Radiology, Nu Med Regional Medical Center, 22141 Roscoe Blvd, Canoga Park, CA 91304. Tel: (818) 340-0580, ext 280.

ILLINOIS

Oct 8–11. **AMA National Conference on the Impaired Health Professional.** Drake Hotel, Chicago. Contact: Janice J. Robertson, AMA Department of Substance Abuse, 535 N Dearborn St, Chicago, IL 60610. Tel: (312) 645-5083.

Oct 9–11. **Controversies in Geriatric Medicine.** Drake Hotel, Chicago. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

MICHIGAN

Oct 11–14. **Industrial Problems of the Upper Extremity in the US and Canada.** Ann Arbor. Contact: American Society for Surgery of the Hand, 3025 South Parker Rd, Suite 65, Aurora, CO 80014.

PENNSYLVANIA

Oct 5–9. **13th Annual Meeting of the American Association of Pathologists Assistants (AAPA).** Sheraton University City Hotel. Contact: Department of Pathology, New York University Medical Center, 550 First Ave, New York, NY 10016. Tel: (212) 340-5747.

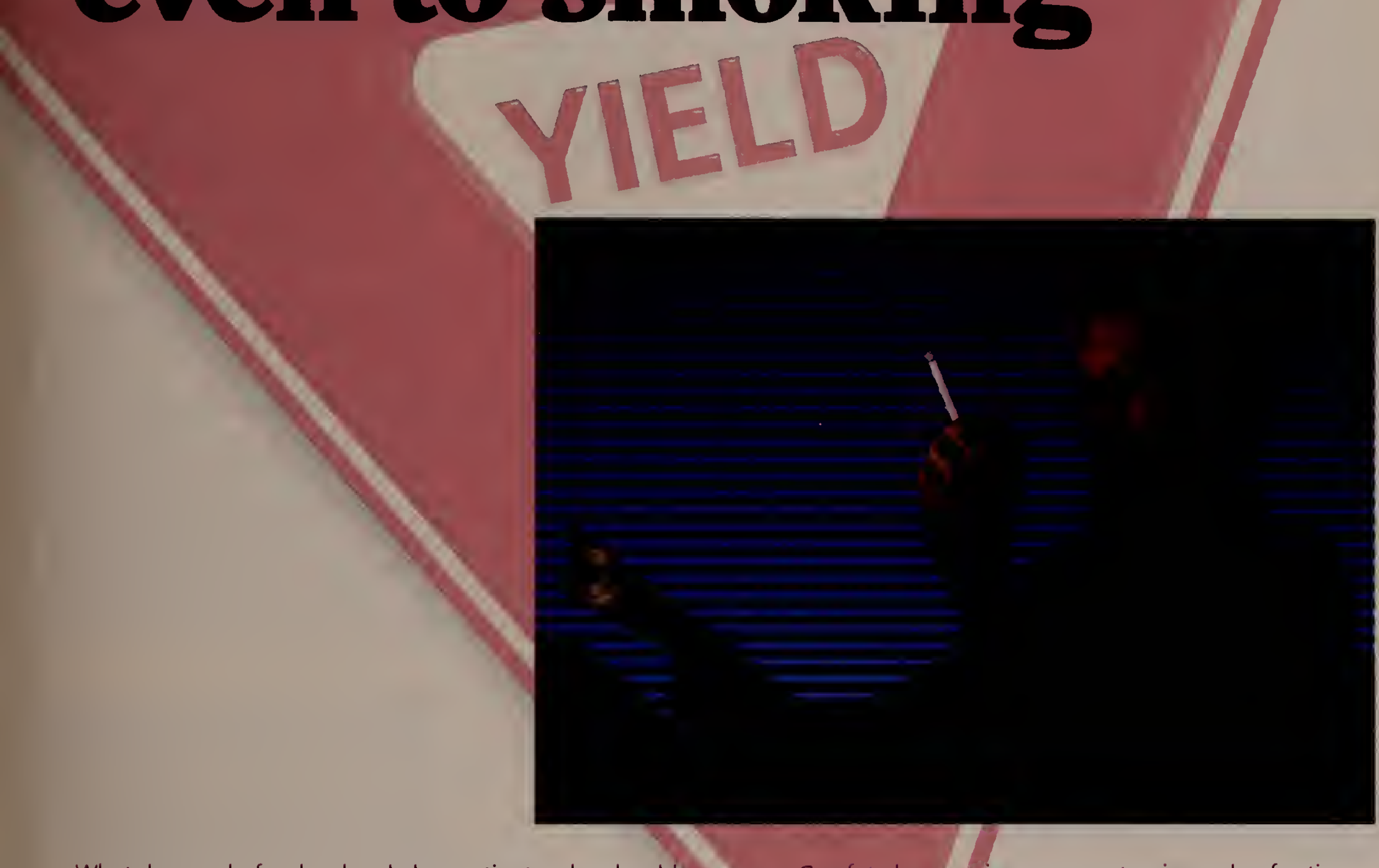
Oct 14–17. **Critical Care Medicine: A Board Review Course.** Penn Tower Hotel, Philadelphia. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

TEXAS

Oct 15–18. **Flexible Fiberoptic Sigmoid-**

(continued on p 22A)

Ulcer therapy that won't yield, even to smoking



What do you do for duodenal ulcer patients who should stop smoking, but won't? Both cimetidine[1] and ranitidine[2] have been shown less effective in smokers than nonsmokers.

Choose CARAFATE® (sucralfate/Marion). Two recent studies show Carafate to be as effective in smokers as nonsmokers.[3,4] A difference further illustrated in a 283-patient study comparing sucralfate to cimetidine[5]:

Ulcer healing rates:
(at four weeks of therapy)[5]

Sucralfate:	
All patients	79.4%
Smokers	81.6%*
Cimetidine:	
All patients	76.3%
Smokers	62.5%

*Significantly greater than cimetidine smoker group ($P < .05$).

Carafate has a unique, nonsystemic mode of action that enhances the body's own ulcer healing ability and protects the damaged mucosa from further injury.

When your ulcer patient is a smoker, prescribe the ulcer medication that won't go up in smoke: safe, nonsystemic Carafate.

Nothing works like

CARAFATE®
sucralfate/Marion

Please see adjoining page for references and brief summary of prescribing information.

1594H7

CARAFATE®
(sucralfate)

BRIEF SUMMARY

CONTRAINDICATIONS

There are no known contraindications to the use of sucralfate.

PRECAUTIONS

Duodenal ulcer is a chronic, recurrent disease. While short-term treatment with sucralfate can result in complete healing of the ulcer, a successful course of treatment with sucralfate should not be expected to alter the post-healing frequency or severity of duodenal ulceration.

Drug Interactions: Animal studies have shown that the simultaneous administration of CARAFATE with tetracycline, phenytoin, or cimetidine will result in a statistically significant reduction in the bioavailability of these agents. This interaction appears to be nonsystemic in origin, presumably resulting from these agents being bound by CARAFATE in the gastrointestinal tract. The bioavailability of these agents may be restored simply by separating the administration of these agents from that of CARAFATE by two hours. The clinical significance of these animal studies is yet to be defined.

Carcinogenesis, Mutagenesis, Impairment of Fertility: No evidence of drug-related tumorigenicity was found in chronic oral toxicity studies of 24 months' duration conducted in mice and rats at doses up to 1 gm/kg (12 times the human dose). A reproduction study in rats at doses up to 38 times the human dose did not reveal any indication of fertility impairment. Mutagenicity studies have not been conducted.

Pregnancy: Pregnancy Category B. Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Adverse reactions to sucralfate in clinical trials were minor and only rarely led to discontinuation of the drug. In studies involving over 2,500 patients, adverse effects were reported in 121 (4.7%). Constipation was the most frequent complaint (2.2%). Other adverse effects, reported in no more than one of every 350 patients, were diarrhea, nausea, gastric discomfort, indigestion, dry mouth, rash, pruritus, back pain, dizziness, sleepiness, and vertigo.

DOSAGE AND ADMINISTRATION

The recommended adult oral dosage for duodenal ulcer is 1 gm four times a day on an empty stomach.

Antacids may be prescribed as needed for relief of pain but should not be taken within one-half hour before or after sucralfate.

While healing with sucralfate may occur during the first week or two, treatment should be continued for 4 to 8 weeks unless healing has been demonstrated by x-ray or endoscopic examination.

HOW SUPPLIED

CARAFATE (sucralfate) 1-gm pink tablets are supplied in bottles of 100 and in Unit Dose Identification Paks of 100. The tablets are embossed with MARION/1712. Issued 3/84

References:
1. Korman MG, Shaw RG, Hansky J, et al: *Gastroenterology* 80:1451-1453, 1981.
2. Korman MG, Hansky J, Merrett AC, et al: *Dig Dis Sci* 27:712-715, 1982.
3. Brandstaetter G, Kratochvil P: *Am J Med* 79(suppl 2C):36-38, 1985.
4. Marks IN, Wright JP, Gilinsky NH, et al: *J Clin Gastroenterol* 8:419-423, 1986.
5. Lam SK, Hui WM, Lau WY, et al: *Gastroenterology* 92:1193-1201, 1987.

Another patient benefit product from
PHARMACEUTICAL DIVISION
MARION LABORATORIES INC.
KANSAS CITY MO 64137

1594H7

"SELLING PRACTICES IS OUR BUSINESS"

Want to maximize the return on your investment? Before you buy or sell, call for an appraisal to assure the best financial and transfer terms. We are a full service practice broker, not simply a listing service. Since 1981, Countrywide has been instrumental in helping hundreds of doctors like yourself buy and sell their practices. We guide you through the entire sales process from initial meeting to closing. Our offices serve New York, New Jersey and New England.

Countrywide Business Brokerage, Inc.
319 East 24th Street, Suite 23-G, New York, NY 10010
(212) 686-7902 (203) 868-3666 (617) 232-1075

MISCELLANEOUS—CONT'D

HAVE YOU RECEIVED ANY COMMUNICATION FROM THE OFFICE OF PROFESSIONAL MEDICAL CONDUCT? If affirmative, contact Susan Kaplan, Attorney-at-Law, (212) 877-5998. Practice limited to the crucial legal problems directly affecting your license to practice medicine—with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professions), and as an Assistant District Attorney in Nassau County. Susan Kaplan, Esq., 165 West End Avenue, Suite 27P, New York, N.Y. 10023.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta, GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

ADVICE, COUNSEL & REPRESENTATION of physicians and other health care professionals in licensure and professional conduct matters, reimbursement issues and practice-related litigation. David E. Ruck, Esq., former Chief, Criminal Division, Office of the New York State Special Prosecutor for Medicaid Fraud Control and Alain M. Bourgeois, Esq., former Acting Justice, Supreme Court of the State of New York, announce the opening of their offices for the practice of law under the name of Bourgeois & Ruck, 101 Park Avenue, New York, New York 10178. Telephone 212-661-8070.

PORTABLE RADIOLOGY SERVICE: abdominal + pelvic ultrasound, cardiac echos including 2D + m-mode portable x-rays and portable osteoporosis bone densitometry screening. NYC and suburbs. Schedule weekly/bi-weekly in your office or visits to your patient's home. New portable hi-tech equipment. Hi-quality like standard office images, far superior to hospital portable images. No lead shielding needed for x-rays. University Radiology Service, (212) 265-7392 or (718) 339-5363.

FIRM OF GERALD GOLDBERG, CPA-ATTORNEY, formerly with IRS, announces his availability to prepare taxes and plan under changing tax environment, accounting, tax appeals, legal representation or practice transfers. (212) 406-1156 and (516) 565-6260.

SIGNATURE LOANS—$5,000 to $60,000 doctors only. No collateral, simple interest, 4, 5, or 6 year terms. No points, no fees, no prepay penalties. No use restrictions. Residents, D.O.'s, new practitioners welcome. For application and info call: (NY) Northstar Funding (212) 323-8076 or Mediversal 1-800-331-4952 Dept. #135.

MISCELLANEOUS—CONT'D

1988 CME CRUISE/CONFERENCES on Medico-legal Issues & Risk Management—Caribbean, Mexico, Alaska, China/Orient, Europe, New England/Canada, Trans Panama Canal, South Pacific. Approved for 24-28 CME Cat. 1 Credits (AMA/PRA) and AAFP prescribed credits. Distinguished lecturers. Excellent Group Rates on Finest Ships. Registration limited. Pre-scheduled in compliance with IRS requirements. Information: International Conferences, 189 Lodge Ave., Huntington Station, NY 11746. (516) 549-0869.

PROFESSIONAL MISCONDUCT ATTORNEYS. William L. Wood Jr., formerly Executive Director of the New York State Office of Professional Discipline and Anthony Z. Scher, formerly Director of Prosecutions, announce the opening of their law office, Wood & Scher. Our recent tenure as chief enforcement officers has given us experience which will allow us to represent physicians in professional misconduct proceedings, malpractice licensure, license restoration, controlled drug proceedings, reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practitioners. Wood & Scher, Attorneys at Law, One Chase Road, Scarsdale, New York 10583. Telephone 914-723-3500.

PACEMAKER PATIENTS WANTED by nationwide transtelephonic follow-up service. Physician will receive complete computerized reports and will be notified if pacemaker malfunction or serious arrhythmia is detected. Interested in enrolling single patients as well as entire patient roster. Reply: Pacer-Care, Post Office Box 1988, Lake Oswego, Oregon 97035, (800) 547-0394.

PROFESSIONAL MISCONDUCT ADVOCACY with dignity and concern. George Weinbaum, Esq. former deputy of the New York State Attorney General's office for Medicaid Fraud Control and also former Director of Medicare and private insurance investigations for Empire Blue Cross/Blue Shield is pleased to announce the opening of his office for the practice of health law. Attention is devoted to misconduct avoidance, disciplinary proceedings, reimbursement maximization, controlled drug issues and medical staff representation amongst other exclusive physician needs. George Weinbaum, Esq., 3 Barker Avenue, White Plains, New York 10601. Telephone (914) 686-9310 office hours, (212) 621-7776-24 hours.

Buy Bonds

Classified Advertising

New York State Journal of Medicine

GENERAL INFORMATION

All classified advertisements are payable in advance, except for government agencies and advertising agencies; a 15% commission to recognized advertising agencies.

Frequency: Monthly
Mailing date: 7th of the month
Closing date: 30 days before mailing date

Circulation: 27,000 (mainly physicians & medical libraries)

DISPLAY ADVERTISING

	1 time	*6 time*	*12 time*	*Width*	*Depth*
1/4-page	$540	$480	$420	3-1/2″ ×	4-7/8″
1/6-page	252	228	204	2-1/8″ ×	4-7/8″
1/8-page	228	204	180	3-1/2″ ×	2-3/8″
1/16-page	114	102	90	3-1/2″ ×	1-1/8″
1/18-page	84	72	60	2-1/8″ ×	1-1/2″

PARAGRAPH ADVERTISING

The first several words are set in bold face caps.

Rates: $35.00 per insertion for 50 words or less; additional words are 40 cents each. On request, Dept. numbers will be assigned by the Journal for replies to be forwarded to the advertiser. Dept. numbers are 50 cents per insertion. There is no frequency discount for paragraph ads.

Counting Words: Two initials, each abbreviation, figures consisting of 5 numerals or less, are counted as separate words. For replies, your name and address, or telephone number should appear at the end of the ad and be counted in the total number of words.

For further information, please call the Advertising Department at 516-488-6100.

CLASSIFIED ADVERTISING ORDER FORM

New York State Journal of Medicine
Attn: Classified Advertising
420 Lakeville Rd., P.O. Box 5404
Lake Success, N.Y. 11042

Date____________________

Name______________________________________

Payment enclosed: $______________

Address______________________________________

Number of Insertions: ____________

City/State/Zip__________________________________

Assign Dept. Number: ____________

Please make check payable to New York State Journal of Medicine.

____

____

____

____

____

____

SIGNATURE LOANS—$5,000 to $60,000

DOCTORS ONLY. No collateral, simple interest, 6 years to repay. No points, no fees, no prepay penalties. Use for any reason. Residents, D.O.'s, start-up welcome. For appointment and info call:

(NY) NORTHSTAR FUNDING (212) 323-8076
or MEDIVERSAL 1-800-331-4952 Dept. #135

EQUIPMENT LEASING—$10K-$10 MILLION
NORTHSTAR FUNDING (212) 323-8076 Apply by Phone

(*continued from p 18A*)

oscopy in the Diagnosis of Colorectal Diseases. 15 Cat 1 Credits. Hilton Palacio Del Rio, San Antonio. Contact: Seminars & Symposia, Inc, 132 East 70th Street, New York, NY 10021. Tel: (212) 517-7520.

Oct 27–31. **Doppler Ultrasound.** 30 Cat 1 Credits. Houston. Contact: Lisa Krehbiel, Institute for Medical Studies, 30131 Town Center Dr, Suite 215, Laguna Niguel, CA 92677. Tel: (714) 495-4499.

VERMONT

Oct 3–10. **Effective Diagnosis and Treatment of Infectious Diseases.** Southwest Vermont. Contact: Kathy Rairigh, EPIC Expeditions, PO Box 209, Sun Valley, ID 83353. Tel: (208) 788-4995.

AROUND THE WORLD

BERMUDA

Oct 14–18. **The Problem Patient: From the Case Records of the NYU Medical Center.** 16 Cat 1 Credits. Sonesta Beach Hotel, Southampton, Bermuda. Contact: NYU Medical Center Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

ITALY

Oct 13–16. **Bombesin-like Peptides in Health and Disease.** Rome. Contact: Joyce Fried, Brain Research Institute, University of California, Center for the Health Sciences, Los Angeles, CA 90024. Tel: (213) 825-5061. □

Doctor . .

Do you, or a physician you know, have a problem with alcohol, drugs, or emotional illness?

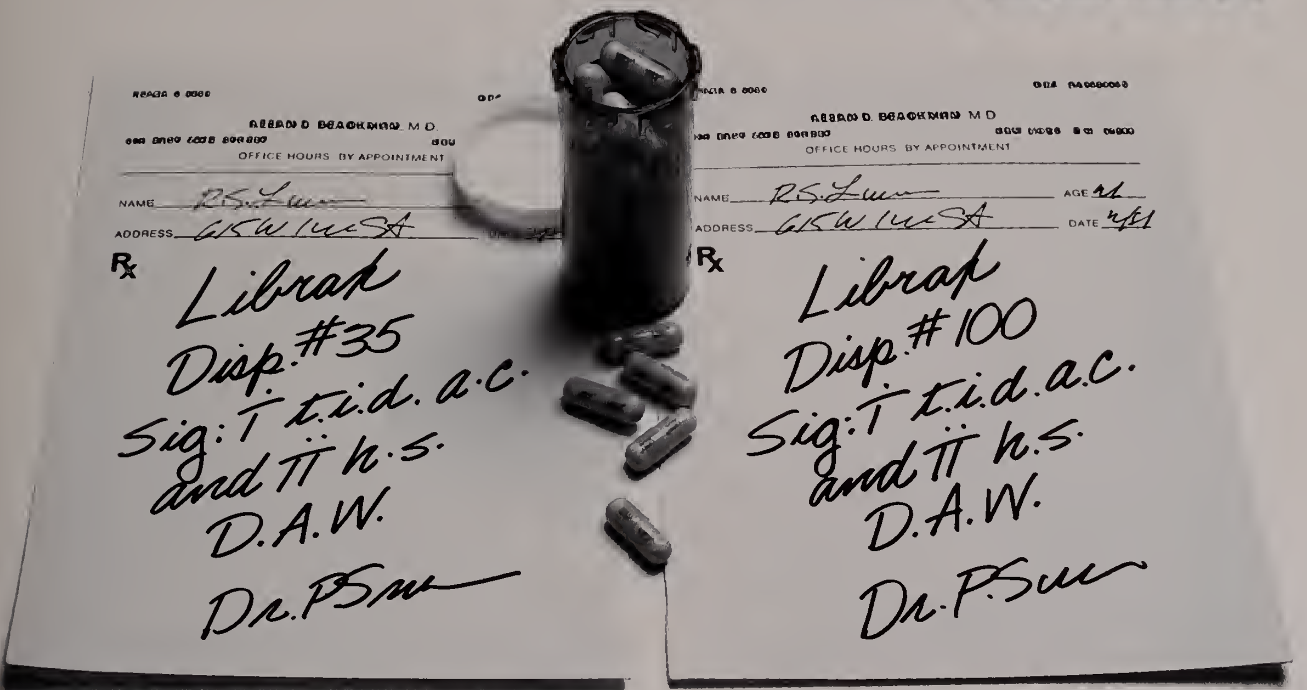
If so, please contact the Medical Society of the State of New York's Committee for Physicians' Health, whose function is to assist, *confidentially.*

Telephone: 516-488-7777.

Index to Advertisers

Ayerst Laboratories 4A, 5A, 6A
CIBA Pharmaceuticals 17A, 18A
St. Clare's Hospital 7A
Classified Advertising 11A, 14A, 20A, 22A
Doctors' RX 16A
Family Practice Recertification Magazine 16A
General Electric Information Services 8A, 9A
Eli Lilly and Company 12A
Marion Laboratories 1A, 2A, 19A, 20A
Milstein Properties Corp, ... 14A
Pisen Public Relations 14A
Pro-Tech Information Systems 16A
A. H. Robins Company 10A, 15A
Roche Laboratories .... 3rd & 4th Cover
Upjohn Company .... 2nd Cover

Specify Adjunctive LIBRAX®



OFFICE HOURS BY APPOINTMENT

NAME R.S. Lum

ADDRESS 65 W 14 St

℞

Librax
Disp. #35
Sig: i t.i.d. a.c.
and ii h.s.
D.A.W.
Dr. P.Sm

OFFICE HOURS BY APPOINTMENT

NAME R.S. Lum AGE 41

ADDRESS 65 W 14 St DATE

℞

Librax
Disp. #100
Sig: i t.i.d. a.c.
and ii h.s.
D.A.W.
Dr. P.Sm

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide.

Please consult complete prescribing information, a summary of which follows:

* **Indications:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:
"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl/Roche) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.
Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

P.I. 0186

Roche Products Inc.
Manati, Puerto Rico 00701

To insist on the brand, be sure to specify "Dispense as Written."

When brain and bowel conflict...

It's time for the Peacemaker.

In irritable bowel syndrome* anxiety can aggravate intestinal symptoms, which may further intensify anxiety — a distressing cycle of brain/bowel conflict. Librax intervenes with two well-known compounds. The Librium® (chlordiazepoxide HCl/Roche) component safely relieves anxiety. And Quarzan® (clidinium bromide/Roche) provides antisecretory and antispasmodic action to relieve discomfort associated with intestinal hypermotility.

Dual action — for peace between brain and bowel. Because of possible CNS effects, caution patients about engaging in activities requiring complete mental alertness. Specify Adjunctive

LIBRAX®

Each capsule contains 5 mg chlordiazepoxide HCl
and 2.5 mg clidinium bromide

*Librax has been evaluated as possibly effective as adjunctive therapy in the treatment of peptic ulcer and the irritable bowel syndrome.

Copyright © 1987 by Roche Products Inc. All rights reserved. Please see summary of prescribing information on adjacent page.

STACKS

NEW YORK STATE JOURNAL OF MEDICINE

OCTOBER 1987 • Volume 87, Number 10

MEDICAL SOCIETY OF THE STATE OF NEW YORK ORGANIZED 1807

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA
OCT 14 1987
M. D. S.

Contents

COMMENTARIES

The myth called Medicare 531
JOHN T. PRIOR, MD

A view of the proposed New York State regulations pertaining to the activities of medical students 533
ASSOCIATED MEDICAL SCHOOLS OF NEW YORK

The "exaggerated death" of peritoneal lavage in the management of trauma patients 534
CARL A. SODERSTROM, MD, PHILIP R. MILITELLO, MD

Nonoperative management of blunt abdominal trauma: The cost/benefit margin 536
JOHN A. MARX, MD, ERNEST E. MOORE, MD

IN MEMORIAM

Joseph F. Shanaphy, MD 538

RESEARCH PAPERS

Physicians' attitudes toward and knowledge about generic drug substitution 539
DANIEL FRIEDMAN, MD; ARNOLD JAFFE, PhD; SUSAN STEINHARDT, MPA

The diagnostic significance of free erythrocyte protoporphyrin and the FEP/hemoglobin ratio in plumbism 542
SOLOMON RAJKUMAR, MD; VALERIE GEIBEL, PhD; BHASKAR DEVANAGONDI, MD; BALAKRISHNA KAUL, PhD; AUDREY K. BROWN, MD

HISTORY

The role of physicians in the life insurance industry in the 19th century 546
JOHN S. HALLER, JR

SPECIAL ARTICLE

The physician-patient relationship in interesting times 553
JOSEPH A. STANKAITIS, MD, MPH

MSSNY WHITE PAPER

Mandatory Medicare assignment 560
MARIO V. MENGHINI

CASE REPORTS

Bile peritonitis following expectant management of liver fracture 565
STEPHEN L. BARKER, MD, DAVID FROMM, MD

Modified radical neck dissections necessitated by a retropharyngeal abscess 567
JAY H. KLARSFELD, MD; STEVEN H. SACKS, MD; ROBERT P. GREEN, MD

Fulminant lymphocyte-depleted Hodgkin disease in a homosexual man with HIV infection 570
DONALD COLBOURN, MD; HARRY STASZEWSKI, MD; STEVEN GOLDENBERG, MD; VIRGINIA DONOVAN, MD; FRANK RAIO, MD

Hypersensitivity reaction to anticonvulsants following head injury 571
JAMES M. HASSETT, JR, MD; THOMAS DOOLITTLE, MD; MICHAEL MOLLOY, BS; DAVID LALKA, PhD

LETTERS TO THE EDITOR

Responses to "Death of a dentist" 574
MAURICE TULIN, MD; ALFRED P. INGEGNO, MD; CHARLES S. HIRSCH, MD; CHARLES A. PERERA, MD; BARRY M. ZIDE, DMD, MD

Moses Maimonides and air pollution 574
FRED ROSNER, MD

Rare *Streptococcus pneumoniae* soft tissue abscess 574
LAKSHMANAN SATHYAVAGISWARAN, MD; P. C. T. DICKINSON, MD; JAY DOBKIN, MD

Isolated endometriosis in a Bartholin gland 575
STEPHEN MATSEOANE, MD; TOMMY HARRIS, MD; EDWARD MOSCOWITZ, MD

Tetracycline reduces the need for insulin 576
VINCENT P. GARBITELLI, MD

BOOK REVIEWS 577
NEWS BRIEFS 581
OBITUARIES 583
MEDICAL MEETINGS AND LECTURES 4A

Motrin® 800 TABLETS mg
ibuprofen

Extra strength
Convenience
Economy

Upjohn
100 A Century of Caring
1886·1986

©1986 The Upjohn Company

J-6138 January 1986

HIV Positive Confirmed. Then What?

AIDS is tough to treat. Ruthless to an increasing patient population, it challenges physicians and nurses as never before in their commitment to quality care.

The Spellman Center for HIV-Related Disease at St. Clare's Hospital and Health Center in New York City, accepts the challenge posed by AIDS and HIV infection. The first facility in the tri-state region to devote its resources solely to the care of AIDS patients, the Spellman Center designates its highest priority as compassionate patient care. A dedicated staff provides comprehensive medical, nursing, psychosocial, and dental treatment in an atmosphere that is warm and understanding.

The Spellman Center—a 60-bed Acute Care Unit, Emergency Service, Outpatient, Ambulatory Infusion and Dental Clinics—accepts referrals of patients with CDC-confirmed AIDS or ARC (AIDS-related complex), and those who are seropositive for HIV. Spellman also reaches out to offer testing, counseling and treatment to persons at risk for AIDS.

If you need consultation concerning your patients, or more information, please call Dr. Debra Spicehandler, the Medical Director, at (212) 459-8409.

The Spellman Center also maintains an information hotline for patients with questions about AIDS: 1-800-433-AIDS.

The Spellman Center
for HIV-Related Disease
A Designated AIDS Treatment Facility
St. Clare's Hospital and Health Center

415 W 51st St
New York, NY 10019

An Affiliate of New York Medical College

NEW YORK STATE JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
ELIZA H. CALDWELL, MD
JOSEPH F. MURATORE, MD
JOHN T. PRIOR, MD
GITA S. SINGH*
STANFORD WESSLER, MD

**Medical student*

Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Consulting Editor and Book Review Editor	RICHARD B. BIRRER, MD
Consulting Editor	NAOMI R. BLUESTONE, MD, MPH
Consulting Editor	CARL POCHEDLY, MD
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Managing Editor	CAROL L. MOORE
Advertising Director	ROBERT MILLER
Consulting Medical Writer	VICKI GLASER
Editorial Assistant	MILDRED J. AREMANN
Secretary	ELIZABETH J. SOMERS
Librarian	ELLA ABNEY
Assistant Librarian	ELEANOR BURNS

ASSOCIATE EDITORIAL BOARD 1987

MICHAEL E. BERLOW, MD
RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MYLES S. GOMBERT, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
JAMES M. MORRISSEY, ESQ
STEPHEN NORDLICHT, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. United Media Associates, Inc, 16 Bruce Park Ave, Greenwich, CT 06830. Telephone: (203) 661-9702.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

SAMUEL M. GELFAND, MD,	*President*
JOHN A. FINKBEINER, MD,	*Past-President*
CHARLES D. SHERMAN, JR, MD,	*President-Elect*
DAVID M. BENFORD, MD,	*Vice-President*
JOHN H. CARTER, MD,	*Secretary*
GEORGE LIM, MD,	*Assistant Secretary*
MORTON KURTZ, MD,	*Treasurer*
ROBERT A. MAYERS, MD,	*Assistant Treasurer*
CHARLES N. ASWAD, MD,	*Speaker*
SEYMOUR R. STALL, MD,	*Vice-Speaker*

Councilors

Term Expires 1988

JAMES H. COSGRIFF, JR, MD,	Erie
RICHARD A. HUGHES, MD,	Warren
ANTONIO F. LASORTE, MD,	Broome
SIDNEY MISHKIN, MD,	Nassau

Term Expires 1989

ROBERT E. FEAR, MD,	Suffolk
STANLEY L. GROSSMAN, MD,	Orange
THOMAS D. PEMRICK, MD,	Rensselaer
RALPH E. SCHLOSSMAN, MD,	Queens

Term Expires 1990

STUART I. ORSHER, MD, (*elected to serve until 1988*)	New York
DUANE M. CADY, MD,	Onondaga
WILLIAM A. DOLAN, MD,	Monroe
ROBERT E. GORDON, MD,	Kings

Resident Councilor (representing the resident physician membership)

KATHLEEN E. SQUIRES, MD,	New York

Student Councilor (representing the medical student membership)

MICHAEL PACIOREK,	Onondaga

Trustees

RICHARD D. EBERLE, MD, (*Chairman*)	Onondaga
EDGAR P. BERRY, MD,	New York
JAMES M. FLANAGAN, MD,	Wayne
ALLISON B. LANDOLT, MD,	Westchester
DANIEL F. O'KEEFFE, MD,	Warren
BERNARD J. PISANI, MD,	New York
VICTOR J. TOFANY, MD,	Monroe

Executive Vice-President	DONALD F. FOY
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Vice-President Emeritus	GEORGE J. LAWRENCE, JR, MD
Director, Division of Policy Coordination	IRMA A. ERICKSON
Director, Division of Scientific Publications	PASCAL JAMES IMPERATO, MD
Director, Division of Communications	EDWARD A. HYNES
Director, Division of Computer Services	IVAN H. NAJMAN
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Governmental Affairs	GERARD L. CONWAY, ESQ
General Counsel	DONALD R. MOY, JD
Director, Division of Management and Meeting Services	ROBERT MILLER
Director, Division of Membership Support Services	EUNICE M. SKELLY
Director, Division of Scientific and Educational Activities	PETER B. FARNSWORTH, MD
Director, Division of Socio-Medical Economics	MARIO V. MENGHINI
Medical Director, Committee for Physicians' Health	BARTON A. HARRIS, MD
Consultant	EDWARD SIEGEL, MD

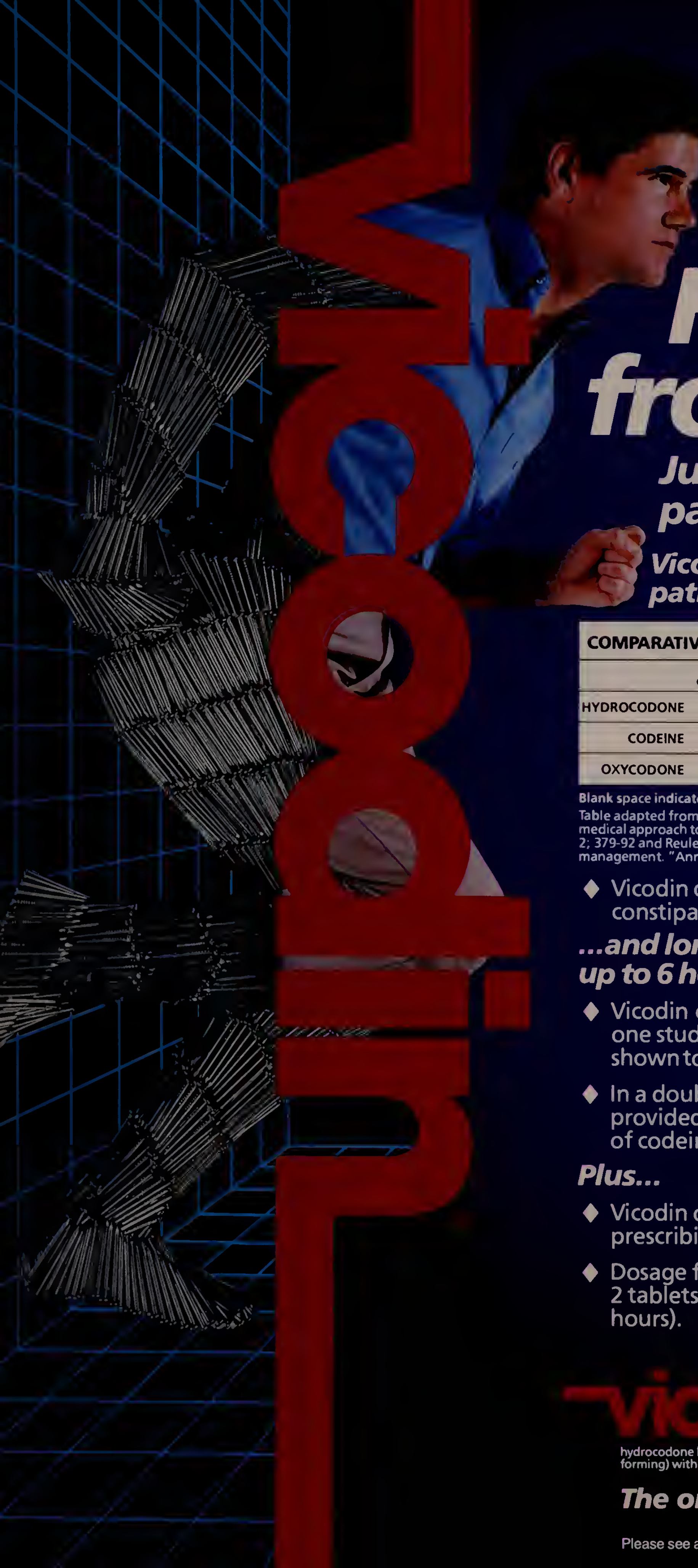
The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York.

Copyright © 1987, Medical Society of the State of New York. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.



Freedom from pain

Just one part of pain relief therapy.

Vicodin® provides greater patient acceptance

COMPARATIVE PHARMACOLOGY OF THREE ANALGESICS

	CONSTIPATION	RESPIRATORY DEPRESSION	SEDATION	EMESIS	PHYSICAL DEPENDENCE
HYDROCODONE		X			X
CODEINE	X	X	X	X	X
OXYCODONE	XX	XX	XX	XX	XX

Blank space indicates that no such activity has been reported.

Table adapted from Facts and Comparisons (Nov.) 1984 and Catalano RB. The medical approach to management of pain caused by cancer. "Semin Oncol" 1975; 2; 379-92 and Reuler JB, et. al. The chronic pain syndrome: misconceptions and management. "Ann Intern Med" 1980; 93; 588-96.

- ◆ Vicodin offers: less nausea, less sedation, less constipation.

...and longer lasting pain relief— up to 6 hours.

- ◆ Vicodin contains hydrocodone not codeine. In one study, 10 mg. of hydrocodone alone was shown to be as effective as 60 mg. of codeine.[1]
- ◆ In a double-blind study, Vicodin (2 tablets), provided longer lasting pain relief than 60 mg. of codeine.[2]

Plus...

- ◆ Vicodin offers the convenience of CIII prescribing.
- ◆ Dosage flexibility—1 tablet every 6 hours or 2 tablets every 6 hours (up to 8 tablets in 24 hours).

1. Hopkinson JH III: *Curr Ther Res* 24; 503-516, 1978
2. Beaver, WT *Arch Intern Med*, 141:293-300, 1981.

vicodin®

hydrocodone bitartrate 5 mg. (Warning: May be habit forming) with acetaminophen 500 mg.

The original hydrocodone analgesic.

Please see adjacent page for brief summary of prescribing information.

vicodin®

hydrocodone bitartrate 5mg (Warning: May be habit forming) and acetaminophen 500mg

INDICATIONS AND USAGE: For the relief of moderate to moderately severe pain
CONTRAINDICATIONS: Hypersensitivity to acetaminophen or hydrocodone
WARNINGS
Drug Abuse and Dependence: VICODIN is subject to the Federal Controlled Substances Act (Schedule III). Psychic dependence, physical dependence and tolerance may develop upon repeated administration of narcotics, therefore, VICODIN should be prescribed and administered with the same caution appropriate to the use of other oral-narcotic-containing medications.
Respiratory Depression: At high doses or in sensitive patients, hydrocodone may produce dose-related respiratory depression by acting directly on brain stem respiratory centers. Hydrocodone also affects centers that control respiratory rhythm, and may produce irregular and periodic breathing.
Head Injury and Increased Intracranial Pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a preexisting increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.
Acute Abdominal Conditions: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions.
PRECAUTIONS
Special Risk Patients: VICODIN should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture.
Information for Patients: VICODIN, like all narcotics, may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery; patients should be cautioned accordingly.
Cough Reflex: Hydrocodone suppresses the cough reflex; caution should be exercised when VICODIN is used postoperatively and in patients with pulmonary disease.
Drug Interactions: The CNS-depressant effects of VICODIN may be additive with that of other CNS depressants. When combined therapy is contemplated, the dose of one or both agents should be reduced. The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone. The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus.
Usage in Pregnancy: Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose. There are no adequate and well-controlled studies in pregnant women. VICODIN should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.
Nonteratogenic Effects: Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent. The intensity of the syndrome does not always correlate with the duration of maternal opioid use or dose.
Labor and Delivery: Administration of VICODIN to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used.
Nursing Mothers: It is not known whether this drug is excreted in human milk, therefore, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.
Pediatric Use: Safety and effectiveness in children have not been established.
ADVERSE REACTIONS
Central Nervous System: Sedation, drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, dizziness, psychic dependence, mood changes.
Gastrointestinal System: Nausea and vomiting may occur; they are more frequent in ambulatory than in recumbent patients. Prolonged administration of VICODIN may produce constipation.
Genitourinary System: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported.
Respiratory Depression: (See WARNINGS.)
DOSAGE AND ADMINISTRATION: Dosage should be adjusted according to the severity of the pain and the response of the patient. However, tolerance to hydrocodone can develop with continued use, and the incidence of untoward effects is dose related. The usual dose is one tablet every six hours as needed for pain (If necessary, this dose may be repeated at four-hour intervals.) In cases of more severe pain, two tablets every six hours (up to eight tablets in 24 hours) may be required.

Revised January 1987 MR 1987 5786

Knoll Pharmaceuticals
A Unit of BASF K&F Corporation
Whippany, New Jersey 07981

BASF Group

knoll®

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

NOVEMBER 1987

AROUND THE STATE

MANHATTAN

Nov 3–5. **Structure and Activity of Heparin and Related Polysaccharides.** Vista International Hotel. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd St, New York, NY 10021.

Nov 4. **Involuntary Hospitalization and Treatment in New York 1987. The State of Debate.** Beth Israel Medical Center. Contact: Beverly R. Baptiste, Medical Education Office, Beth Israel Medical Center, First Ave at 16th St, New York, NY 10003. Tel: (212) 420-2849.

Nov 6. **New Dimensions in the Management of Facial, Head and Neck Pain.** Conference Room, New York Eye and Ear Infirmary. Contact: Barry C. Cooper, DDS, Conference Coordinator, 310 East 14th St, New York, NY 10003. Tel: (212) 598-1600.

Nov 6. **New York Allergy Society—Annual Teaching Day.** 5 Cat 1 Credits. Cornell Medical Center. Contact: Ms Noell Sexton, The New York Allergy Society, c/o St Lukes-Roosevelt Hospital Center, 428 West 59th St, New York, NY 10019. Tel: (212) 554-7192.

Nov 7–8. **The Cardiac Patient for Non-Cardiac Surgery—The Experts Opine.** Mount Sinai Medical Center. Contact: Department of Anesthesiology, Mount Sinai Medical Center, One Gustave L. Levy Place, Box 1010, New York, NY 10029. Tel: (212) 650-7630.

Nov 7–8. **New York Otology: '87.** Manhattan Eye, Ear and Throat Hospital. Contact: Joanne Lupetin, Administrator, Department of Otolaryngology, Manhattan Eye, Ear and Throat Hospital, 210 East 64th Street, New York, NY 10021. Tel: (212) 838-9200, ext 2448.

Nov 19–21. **Obstetric and Gynecologic Emergencies.** 16 Cat 1 Credits. Grand Hyatt Hotel. Contact: Registrar, The American College of Obstetricians and Gynecologists, 600 Maryland Ave, SW, Suite 300 East, Washington, DC 20024-2588. Tel: 1-800-633-8811.

Nov 20. **First International Breast Cancer Chemoprevention Workshop-Conference.** Essex House. Contact: Jaclyn Silverman, Chemoprevention Workshop Coordinator, Division of Medical Oncology, Mount Sinai Medical Center, One Gustav L. Levy Place, New York, NY 10029. Tel: (212) 369-5440.

AROUND THE NATION

ARIZONA

Nov 2–5. **Update in Clinical Medicine.** The Sheraton Scottsdale Resort. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

Nov 5–7. **Gynecologic Surgery: Principles, Techniques, and Management of Complications.** 16 Cat 1 Credits. Contact: Registrar, The American College of Obstetricians and Gynecologists, 600 Maryland Ave SW, Suite 300 East, Washington, DC 20024-2588. Tel: 1-800-633-8811.

CALIFORNIA

Nov 5–7. **San Francisco Otology Update—1987.** 17 Cat 1 Credits. Holiday Inn Union Square Hotel. Contact: University of California, Extended Programs in Medical Education, Room U-569, San Francisco, CA 94143-0742. Tel: (415) 476-5808.

Nov 6–8. **The 33rd Annual Group Therapy Symposium.** Cathedral Hill Hotel, San Francisco. Contact: Extended Programs in Medical Education, University of California, Room U-569, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

Nov 11–15. **16th Annual Meeting, International Congress of Gynecologic Endoscopy.** Sheraton Palace, San Francisco. Contact: Jordan M. Phillips, MD, American Association of Gynecologic Laparoscopists, 13021 East Florence Ave, Santa Fe Springs, CA 90670. Tel: (213) 946-8774.

(*continued on p 16A*)

Mutual trust...

As a physician, you know the importance of trust between you and your patients. What's more, you're well aware that the foundation of that trust lies in the knowledge and experience you have demonstrated as a professional.

Expertise is also the cornerstone of AMA Advisers, Inc., a subsidiary of the American Medical Association. We naturally have your investment interests in mind. That's why we dedicate our efforts to physicians like you...and why we provide a variety of alternative investment strategies that can be tailored to your specific needs.

You can build your investment program around The AMA Group, the mutual funds managed by AMA Advisers, Inc., designed to help you meet your financial goals with a broad choice of investment strategies. Plus, we offer a full range of investor services, including our newest service, Asset Allocation, and, of course, professional management of the highest quality.

Whether you're new to investing or an "old pro", AMA Advisers' money management expertise may help you reach your financial goals. Simply complete and mail the coupon for your FREE Information Kit on the funds in The AMA Group. We'll send you a prospectus with more complete information detailing fees and expenses. Please read the prospectus carefully before you invest or send money.

Services and products as described herein are not offered for sale in any state where they are not lawfully registered.

Building Mutual Trust

AMA ADVISERS, INC.

AMA Advisers, Inc. is a subsidiary of the American Medical Association and manages the mutual funds in The AMA Group.

FOR FASTER ACTION, CALL TOLL FREE 1-800-AMA-FUND EXTENSION 2043, ANYTIME!

MAIL TO: The AMA Group NY2043
Mutual funds managed by AMA Advisers, Inc.
P.O. Box 1923, West Chester, PA 19380-1923

☑YES. I want my FREE Information Kit on the funds in The AMA Group, as well as a prospectus with more complete information detailing fees and expenses.

Name ______________________________

Address ______________________________

City ______________________________

State ____________________ Zip __________

Daytime Telephone (________) ______________

Classified Advertising

"SELLING PRACTICES IS OUR BUSINESS"

Want to maximize the return on your investment? Before you buy or sell, call for an appraisal to assure the best financial and transfer terms. We are a full service practice broker, not simply a listing service. Since 1981, Countrywide has been instrumental in helping hundreds of doctors like yourself buy and sell their practices. We guide you through the entire sales process from initial meeting to closing. Our offices serve New York, New Jersey and New England.

Countrywide Business Brokerage, Inc.
319 East 24th Street, Suite 23-G, New York, NY 10010
(212) 686-7902 (203) 868-3666 (617) 232-1075

PHYSICIANS WANTED

EMERGENCY MEDICINE POSITIONS—Full/part time emergency medicine physicians sought by multi state professional association for openings in metropolitan NY, PA, MD, DC, FL, New England and throughout U.S. Contact or send CV to Liberty Healthcare Corporation, 399 Market Street, Suite 400, Philadelphia, PA 19106, (215) 592-7400 or outside Pa (800) 331-7122.

ARIZONA-BASED PHYSICIAN RECRUITMENT firm has quality opportunities coast to coast. Available positions in most primary care and surgical specialties to include OB/GYN, Orthopedics, ER, and ENT. "Quality Physicians for Quality Clients since 1972." Call 602-990-8080; or send CV to: Mitchell & Associates, Inc., PO Box 1804, Scottsdale, AZ 85252.

GENERAL INTERNIST—Immediate need due to recent death of respected physician. Take over established practice, fully equipped office; coverage with 4 other internists who enthusiastically support this effort. Unlimited potential with first year compensation guaranteed. Colorful community in western New York; close to Finger Lakes with excellent family environment. Reply Dept. 449 c/o NYSJM.

GREAT OPPORTUNITY for Directorship and staff positions in newly designed emergency room located near the mountains in south central New York; just three hours from New York City. 28,000 annual patient visits. Lucrative compensation package for director including benefits. Professional liability insurance procured on your behalf. Flexible scheduling gives you the free time to enjoy the recreational aspects of the area. Contact: Christine Fairchild, Coastal Emergency Services, Inc., 550 Latona Rd., Building C, Rochester, NY 14626; (716) 225-4090 (collect).

INTERNIST WANTED—Busy three man General Internal Medicine practice in community setting seeks board eligible/board certified internist for primary care responsibilities. Excellent benefits and opportunities offered for association. Contact (201) 837-3200 or send C.V. to Dr. Peter W. Coppola, 751 Teaneck Road, Teaneck, NJ 07666.

DISABILITY EVALUATING PHYSICIANS: Full or part-time positions available performing independent disability determinations, no-fault examinations and various other physical examinations in rapidly expanding Albany office. Send C.V. to AIP, P.O. Box 13964, Albany, New York 12212.

POSITIONS WANTED

SURGEON FOR HIRE. Experienced general surgeon available 3 days a week, for first assisting, Westchester area. O.R. work only. Must include monetary offer in your reply. Dept. 450 c/o NYSJM.

POSITIONS WANTED—CONT'D

ENDOCRINE PRACTICE DESIRED: M.D., 1983, ABIM 1986; B/E Endocrinology/Metabolism, 1988; University trained; clinical experience in general endocrinology, diabetes, reproductive endocrinology, and lipoprotein disorders. Available July, 1988. Desire practice in NYC/metropolitan area. Reply to Alex Stagnaro-Green, M.D., 228 Voorhis Ave., River Edge, New Jersey 07661. (201) 265-4859.

PRACTICES AVAILABLE

FOR SALE: FAMILY PRACTICE. Retiring, immediate occupancy. Professional Building, long standing active practice. In excellent community on main thoroughfare in Nassau County, 200 feet across Queens border. Ideal for new ambitious M.D. or relocation. 1200 square feet modern rooms, fully equipped with x-ray. Low overhead. 10 minutes from 4 major hospitals. Reply Dept. 447 c/o NYSJM.

DERMATOLOGY PRACTICE FOR SALE. Terms negotiable, 70's upper east side. Call (212) 744-6809.

EQUIPMENT

50% OFF PREVIOUSLY OWNED MEDICAL—Laboratory, x-ray, ultrasound equipment. Buy, Sell, Broker, Repair. Appraisals by certified surgical consultants. Medical Equipment Resale & Repair, 24026 Haggerty Road, Farmington Hills, MI 48018. 1-800-247-5826. (313) 477-6880.

DISCOUNT HOLTZER SCANNING SERVICES starting at $35.00. Spacelabs holter recorders available from $1,275.00. Smallest and lightest holters update. Fast service (24–48 hours) turnover. Hook-up kits starting at $4.95. Special introductory offer of three free tests at the purchase or lease of the holter. Stress test electrodes available at 34 cents. Scanning paper available at $18.95. Cardiologist over-read available at $15.00. If interested call 1-800-248-0153.

REAL ESTATE FOR SALE OR RENT

FOR SALE—Combination professional office and home, Oceanside, NY; opposite South Nassau Community Hospital on 1/3rd acre lot. Never pay rent again. Telephone (516) 536-6279.

REAL ESTATE FOR SALE OR RENT—CONT'D

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

MANHATTAN, 220 EAST 69TH STREET. Share expenses. Luxury building. Own 12 × 15 consultation room, garden window; or share two days. Share 3 equipped examination rooms. Laboratory, x-ray services on premises. Suitable for Internist, Surgeon or Psychiatrist. (212) 737-4020.

FOR RENT completely equipped office in upstate New York. Office has large waiting, consultation, treatment, x-ray, lab, physiotherapy, and 2 bathrooms. Utilities included. Attached garage. Only physician here for 54 years. Contact: Francis D. Brown, M.D., Maple-Bank, Hobart, NY 13788.

THE CONTINENTAL—Professional Co-op office space for sale. 70-20 108 St. Premier, prestigious building in the heart of Forest Hills, Queens, NY. Superb location, subway and buses at your door. 24 hour attended garage, central A/C, separate professional wing. 370–1600 sq. ft. possible to combine. Call: Mr. Marvin Weingart, (718) 792-1081.

OFFICE SPACE NEW ROCHELLE, NY. 3 Internists in office, all lab and x-ray. Call (914) 632-1952.

FOR SALE—OFFICE BUILDING, L.I. Suffolk County. Family practitioner retiring after 33 years. Building has 3 office suites, C/A, W/W carpeting, paneling, separate entrances, private parking and 3 zone heat. Excellent location, near hospitals. Suitable all specialties. For information please call (516) 667-7872.

OFFICE FOR RENT: Furnished, excellent location. Center of Village, Schuyler Hospital two blocks away. Paul F. Willwerth, M.D., 105 Genesee Street, Montour Falls, N.Y. 14865 (607) 535-9671 or (607) 546-2450.

CUSTOM BUILD superbly designed professional home located in the East Northport area on highly visible 1/2 acre, just minutes from local hospitals, parkways and schools. Reception room plus 900 sq. ft. useable space on lower level. Upper level features wonderful contemporary living area with highhat lighting, skylights and cathedral ceilings. Asking $425,000. The Daniel Gale Agency, Camille Sonesen (516) 754-3400 or Joan Gannon (516) 692-6770.

DAC
FOR SEVERE COUGHS, CAN YOU REMEMBER THESE LETTERS?
NDC 0031-8680-25
A-H-ROBINS
Robitussin®-DAC
EXPECTORANT
ANTITUSSIVE
Alcohol 1.4 per cent
KEEP THIS AND ALL DRUGS OUT OF REACH OF CHILDREN. IN CASE OF ACCIDENTAL OVERDOSE, SEEK PROFESSIONAL ASSISTANCE OR CONTACT A POISON CONTROL CENTER IMMEDIATELY.
16 FL.OZ. (1PT.)

You know Robitussin®, and the "DAC" formula gives you just what you need to manage those severe coughs complicated with nasal congestion . . . Decongestant And Codeine!

Robitussin®-DAC Ⓒ

Each 5 ml (1 teaspoonful) contains:
Guaifenesin, USP .... 100 mg
Pseudoephedrine
Hydrochloride, USP .... 30 mg
Codeine Phosphate, USP .... 10 mg
(Warning: May be habit forming)
Alcohol 1.4 percent

℞ ONLY

A-H-ROBINS
Pharmaceutical Division
Richmond, Virginia 23261-6609

SARANTOS & COMPANY

S. Randy Sarantos, D.D.S., President

THE DOCTORS' MONEY DOCTOR

1. ***Investment Management Services***
   Pension, Personal and Childrens' Educational Asset Management Programs
2. ***Total Financial Planning Services***
   Wide selection of 10 different financial plans for every budget level
3. ***Insurance Advisory Services***
   Custom-designed Insurance and Risk Management programs
4. ***Practice Management Services***
   Buy/Sell, Associate Advisory Services, Practice Evaluations, Cash-Flow Analysis
5. ***Seminar Education Services***
   Special Financial Seminar programs for professional societies, groups & institutions

Exclusive Marketing Agents For:

Pro-Tech Practice Evaluations
Low-cost Expanded Option Analysis, including 180 values for your practice.

National Association of Doctors (NAD)
Exclusive agents in Eastern PA, NJ and CT, providing low-cost, high quality financial services.

YES: I would like to know more about Professional Financial Services. Please send me a free brochure.

Name ______________________________

Address ______________________________

City ______________ State ________ Zip ________

Sarantos & Company, Inc. 240 Cedar Knolls, Rd., Suite 310
Cedar Knolls, NJ 07927 (201) 539-4000

— OR CALL 1-800-223-0164 —

The principals of Sarantos & Company, Inc., a Registered Investment Advisor, are also representatives of Integrated Resources Equity Corp. Member NASD & SIPC, and a registered broker dealer

ORTHOPEDISTS

Tired of the hassles of practice?
Tired of the malpractice burden?

I need two additional full-time orthopedists to join our 15 man group. One for our facility in Syracuse, and one for Rochester.

We do evaluations only, no treatment.

Banker's hours, pleasant work, good remuneration. No nights or weekends.

If you are ready to give up surgery for a more placid life, send your C.V. to me:

Harvey Lewis, M.D.
3155 G. Eggert Road
Tonawanda, NY 14150

In New York City, for the Newest and Finest Medical Space, Remember:

THE 4 ESSENTIAL REQUIREMENTS

1. LOCATION!
   Liberty Residences at Battery Park City
2. LOCATION!
   Windsor Pavilion
   (3rd Avenue & 32nd Street)
3. LOCATION!
   205 East 64th Street (3rd Avenue)

AND

4. LOCATION!
   Normandie Pavilion
   (3rd Avenue & 95th Street)

For Further Information Contact Lester Schwalb
(212) 708-0846
Milstein Properties Corp.

Essential reading for every physician

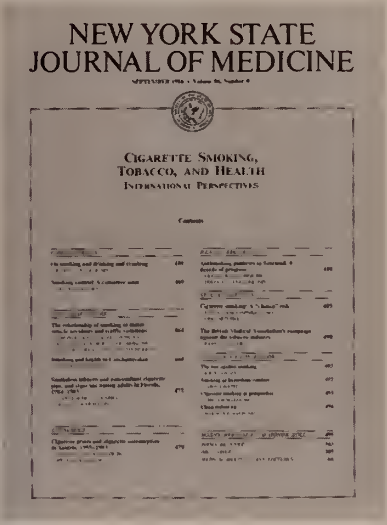
NEW YORK STATE
JOURNAL OF MEDICINE

THE WORLD CIGARETTE PANDEMIC

NEW YORK STATE
JOURNAL OF MEDICINE

THE WORLD CIGARETTE PANDEMIC
Part II

PRIMUM NON NOCERE


NEW YORK STATE
JOURNAL OF MEDICINE

CIGARETTE SMOKING,
TOBACCO, AND HEALTH
INTERNATIONAL PERSPECTIVES

Contents

The December 1983, July 1985 and September 1986 issues of the **New York State Journal of Medicine** constitute the most comprehensive coverage on the subject of smoking ever published by a medical journal.

SEND A COPY TO COLLEAGUES AND MEDICAL STUDENTS in the US and abroad who might not normally receive the Journal. Or order an extra copy for your patients and yourself.

Extra press runs have enabled the New York State Journal of Medicine to offer single or bulk copies of its issues on smoking. As the official publication of the Medical Society of the State of New York since 1900, the Journal is one of the foremost independent peer-reviewed general medical journals in the country. Providing timely and stimulating commentary, challenging research papers, analysis of clinical trends, debate on topical issues, and in-depth coverage of medical news, the Journal makes important reading—and a welcome gift—to physicians and students in the United States or abroad. A free copy of the July 1985 issue will be included with each subcription.

The Following, Reduced Rates Are Now Being Offered On The Smoking Issues

Quantity	Per Copy*
1–24	$4.00
25–49	$3.50
50–99	$3.00
100–149	$2.50
150–500	$2.00

ORDER FORM

Name____

Address______________________________________

City/State/Zip__________________________________

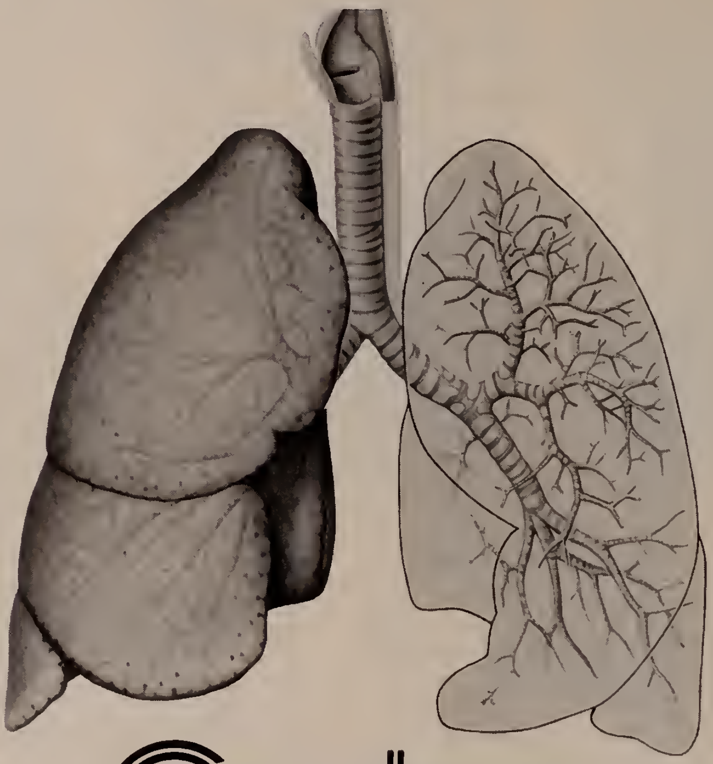
Please send ______ copies of December 1983, ______ copies of July 1985, ______ copies of September 1986 issue of the Journal.

Enter my subscription to the Journal. ☐ $25.00 U.S. & Canada; ☐ $33.00 Foreign

Total amount enclosed $ ________ ***Note:** Domestic postage included. Please add New York state & local sales tax where applicable, or sales tax exemption certificate. Foreign postage additional.

Mail to: Circulation Department, **New York State Journal of Medicine**
420 Lakeville, Rd., P.O. Box 5404, Lake Success, N.Y. 11042.

Consider the causative organisms...



Ceclor®
cefaclor

250-mg Pulvules® t.i.d.

offers effectiveness against the major causes of bacterial bronchitis

Haemophilus influenzae, Streptococcus pneumoniae
(ampicillin-susceptible and ampicillin-resistant)

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

Ceclor® (cefaclor)

Summary. Consult the package literature for prescribing information.

Indications: Lower respiratory infections, including pneumonia, caused by susceptible strains of *Streptococcus pneumoniae, Haemophilus influenzae,* and *Streptococcus pyogenes* (group A β-hemolytic streptococci).

Contraindication:
Known allergy to cephalosporins.

Warnings:
CECLOR SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY. POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS.

Administer cautiously to allergic patients.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea. Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.

Precautions:
- Discontinue Ceclor in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of nonsusceptible organisms.
- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- Ceclor should be administered with caution in the presence of markedly impaired renal function. Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk. Exercise caution in prescribing for these patients.

Adverse Reactions: (percentage of patients)

Therapy-related adverse reactions are uncommon. Those reported include:
- Gastrointestinal (mostly diarrhea): 2.5%.
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
- Hypersensitivity reactions (including morbilliform eruptions, pruritus, urticaria, and serum-sickness-like reactions that have included erythema multiforme [rarely, Stevens-Johnson syndrome] or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever): 1.5%; usually subside within a few days after cessation of therapy. Serum-sickness-like reactions have been reported more frequently in children than in adults and have usually occurred during or following a second course of therapy with Ceclor. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.
- Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.
- As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely.
- Rarely, reversible hyperactivity, nervousness, insomnia, confusion, hypertonia, dizziness, and somnolence have been reported.
- Other: eosinophilia, 2%; genital pruritus or vaginitis, less than 1%; and, rarely, thrombocytopenia.

Abnormalities in laboratory results of uncertain etiology
- Slight elevations in hepatic enzymes.
- Transient fluctuations in leukocyte count (especially in infants and children).
- Abnormal urinalysis; elevations in BUN or serum creatinine.
- Positive direct Coombs' test.
- False-positive tests for urinary glucose with Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (glucose enzymatic test strip, Lilly).

[072886R]

PA 8794 AMP

©1987, ELI LILLY AND COMPANY

Lilly

Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285

Eli Lilly Industries, Inc
Carolina, Puerto Rico 00630

700241

Residents / Fellows

A $500 AWARD will be presented by the Medical Society of the State of New York to the intern, resident or fellow who submits the best paper on a clinical subject for the Society's 1987 Merit H. Cash Competition.

Entrants need not be members of the MSSNY but must be in training at a hospital within the State of New York.

Entries should be "sponsored" by a faculty member, and all papers submitted should be of sufficient quality to be considered for original publication in the *New York State Journal of Medicine.*

The deadline for receipt of entries has been extended to December 1, 1987, and the prize for the winning paper will be awarded at the MSSNY's annual meeting in April of 1988.

Address all entries to: Division of Scientific Activities
Medical Society of the State of New York
420 Lakeville Road
P.O. Box 5404
Lake Success, NY 11042

Questions? Contact either the Division of Scientific Activities or MSSNY's Resident Physicians Section at (516) 488-6100.

1988 ANNUAL CME ASSEMBLY

Medical Society of the State of New York
The New York Hilton, New York City

Newer Approaches to Habit Abuse and Dependency

FRIDAY, APRIL 22

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Plenary Session: Human Abuses Through the Ages: "From the Womb to the Tomb," *Sutton South/Regent Parlor*
2:00 p.m. - 5:00 p.m.	Women's Medical Society/Occupational Health, *Gramercy B*
	Cardiology, *Sutton South*
	Physical Medicine & Rehabilitation/Family Practice/Orthepedics, *Gramercy A*
	Emergency Medicine/Critical Care, *Murray Hill B*
	Pathology/Pediatrics, *Bryant Suite*
	General Surgery/Radiology, *Regent Parlor*
	Neurosurgery, *Murray Hill A*
	Medical Liability Mutual Insurance Co., *Rendezvous Trianon*

SATURDAY, APRIL 23

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Internal Medicine (All Day), *Regent Parlor*
	Ophthalmology (All Day), *Sutton South*
	Hand Surgery/Neurology (All Day), *Sutton Parlor North*
	Psychiatry (All Day), *Gramercy B*
	Urology/Radiology, *Nassau A*
	Dermatology, *Murray Hill A*
2:00 p.m. - 5:00 p.m.	Internal Medicine, *Regent Parlor*
	Ophthalmology, *Sutton South*
	Hand Surgery/Neurology, *Sutton Parlor North*
	Psychiatry, *Gramercy B*
	Chest Diseases/Otolaryngology, *Murray Hill B*
	Computers in Medicine, *Nassau A*

SUNDAY, APRIL 24

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Allergy & Immunology, *Gramercy B*
	Obstetrics and Gynecology, *Murray Hill A*
	Gastroenterology/Colon and Rectal Surgery, *Nassau A*
	Orthopedics/Family Practice/Physical Medicine & Rehabilitation/Anesthesiology, *Regent Parlor*
	Alcoholism, *Sutton Parlor Center*
	Sleep Disorders—Apnoea, *Nassau B*

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis. The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians.

◆

Technical Exhibits

NEW YORK STATE
JOURNAL OF MEDICINE

October 1987 • Volume 87, Number 10

COMMENTARIES

The myth called Medicare

In 1965 Lyndon Johnson created Medicare. At that time he said, "No longer will older Americans be denied the healing miracle of modern medicine. No longer will illness crush and destroy the savings they have so carefully put away over a lifetime so that they might enjoy dignity in their later years."[1] Since 1965 a half a trillion dollars have been spent on Medicare. Medicare spending has grown from $1 billion in 1966 to $70 billion in 1985. This sum, however, pays less than half the total health care costs of its beneficiaries.

The elderly population has been lulled into believing that the Medicare program will provide quality care compatible with their ability to pay at a time in life when their resources are limited. Few understand that under the rules as currently constituted the Medicare beneficiary must pay $492 deductible for each period of hospital confinement; pay $123 for each day of hospitalization from the 61st through the 90th day; pay $246 for each day of hospitalization from the 91st through the 150th day; pay all costs after 150 days; pay an additional $75 deductible each year for certain other covered services; pay at least 20% of all surgeons' and physicians' fees as well as fees for certain supplies and services; and pay any out-of-hospital nursing charges, as well as charges for private duty nursing.

Older people with an income of less than $10,000 a year can expect to spend one half of this on health care, most of it on long-term care. Seventy percent of the elderly believe that Medicare covers long-term nursing home care. The fact is that only Medicaid provides extended nursing home coverage, and only the impoverished qualify. The cost of nursing home care averages $20,000 a year. Obviously there is a need for long-term care insurance, but insurance companies are only now testing the water. Remember, too, that the well-to-do are not immune from catastrophe, since one third of all couples and two thirds of all single people who move into nursing homes will become impoverished within 13 weeks. The federal bureaucrats responsible for operating this program are forcing the elderly with major illnesses and disabilities to be further harassed by painful hospital bills and the debt collection process as the bureaucrats attempt to remove what is left of their meager income. The most outrageous proposal to surface recently is a plan advocated by the Inspector General of the US Department of Health and Human Services. Under this plan, sums would be deducted from the monthly Social Security checks of Medicare beneficiaries who are unable to pay their share of Medicare hospital debts.

The Great Society programs are today in serious financial trouble, and most economists predict bankruptcy by early 1990. In an attempt to hold down costs, in 1983 the Congress put together a legislative program for changing the way hospitals are paid for the care of Medicare patients.[2] The program is called a prospective payment system (PPS), and its key ingredient is diagnosis related groups (DRGs). New York State was a late entry into the program, which has been in effect here only since January 1, 1986. Before PPS, hospitals were reimbursed on a fee-for-service basis. Payment depended upon length of stay and any ancillary services the patient received. With PPS the reimbursement rates have been predetermined for 468 different diagnoses ranging from hip fractures to cataract surgery. Factors that establish a patient's DRG include the diagnosis and surgical procedures performed, any complications or comorbidities, and the patient's age and sex. If the hospital uses more resources than are allocated for a specific DRG, it will lose money. If the hospital uses fewer resources, it will realize a savings. It is because of this that there is anxiety on the part of the Medicare population that they will be catapulted out of the hospital doors prematurely. A Senate Special Committee on Aging has investigated a large number of alleged premature patient discharges and the problem was found to be more severe and widespread than the Health Care Financing Administration estimates had indicated.[3]

The prospective payment system, by reason of both the DRGs and the current peer review quality assurance program, is causing fear and misunderstanding among the elderly population. As physicians become aware of the inequities of Medicare, they will be better able to work in concert with those groups as they try to develop a more responsive and higher quality health care system.

It appears that the DRGs can cut costs, but at the expense of quality of care for our older Americans. It seems that senior citizens are being abandoned to fend for themselves in a health care system that is inadequately prepared to care for them. Financially efficient hospitals are rewarded and those that exceed the target DRG payment are penalized, hence the emphasis on early discharge. The elderly must "get well or get lost."

To guard against unnecessary expense and to guarantee quality health care, the government created monitoring and review agencies called peer review organizations (PROs). They have been in place since 1972 and employ hundreds of physicians and nurses, but until recently they have been more concerned with reducing lengths of stay than with increasing quality of care. There is general agreement that PROs have effected a decline in hospital admissions—4.0% since the PROs began their reviews.[4] In 1982 there was no change in the Medicare admission rate, and there was only a 0.5% decline in 1983. There has also been a continued decline in the average length of stay for patients over age 65, which measured 7.6% between 1983 and 1984. Critics emphasize, however, that this concentration on shortening stays and avoiding unnecessary admissions has been at the expense of quality concerns. The PROs have until recently addressed quality by a contractual agreement to concentrate on a selected number of surgical and medical quality objectives. These have proven to be restrictive and unimaginative, and lack any baseline data to validate the existence of quality problems. Health Care Financing Administration officials have agreed to a new concept using "generic screens" to identify quality and to develop standards for measuring it. This calls for screening charts for a variety of quality indicators, such as hospital-acquired infection, unscheduled return to the operating room, abnormal temperature or blood pressure on discharge, and so on. With the emphasis on reduction of admissions and a shift towards ambulatory care, many feel the scope of PRO review is too narrow. PROs ought to examine what happened before the patient entered and after he left the hospital. A test of this approach will come through PRO review of the care Medicare patients have received in health maintenance organizations (HMOs). The PRO should investigate deaths occurring at home associated with a hurried hospital discharge as well as medical complications, including death, brought about by denial of hospital admission. Other areas which should rightfully be subjects for PRO review are instances of failure to prepare patients for home care as well as the reliability of home health services. Frequently patients require more extensive care and supervision than many of these agencies can provide. Quality of care problems resulting from inadequate outpatient care could be identified if all admissions for ruptured appendix, gangrene, cellulitis, digitalis toxicity, diabetic acidosis, perforated ulcers, etc, were retrospectively analyzed. Finally, the PRO should be funded to undertake studies to explain the extreme variations in admission, length of stay, and cost of hospitalization that occur in this country today for identical disease processes. We must explain variations in rates of hernia repair, hysterectomy, hemorrhoidectomy, pneumonia, and prostatectomy—the diseases are identical but the disparity in incidence rates and patient fee statistics is shocking! We must narrow this range of variation in practice and to do this we need research aimed at establishing a consensus within the medical profession with respect to appropriate care. We must be certain that these variations are the result of sound medical judgments formed by the most thorough analysis of the outcomes of treatment alternatives.

Vita Ostrander,[5] past president of the American Association of Retired People (AARP), has stressed that the Gramm-Rudman spending cuts, $36 billion annually until the budget is balanced, will have profound effects on the elderly people who are trying to live on a moderate income. The elderly wonder how the Gramm-Rudman amendment will change what they can expect to pay for health care when they are ill, or what will happen when their housing subsidies are cut, their telephone bills go up, or their community support services are reduced. Will hospitals discharge them prematurely under PPS? Can skilled nursing facilities and home health agencies provide the needed services under existing guidelines? Will HMOs and competitive medical plans (CMPs) assure access to and provide for quality care? Concerns that the elderly will be deinstitutionalized are receiving increasing publicity, since deinstitutionalization carries the risks of increased death rates, more postoperative infections, and an overall deterioration in the quality of care provided. The AARP has been supportive of the PROs, and has called for comprehensive uniform data about providers, particularly hospitals and physicians. They, too, are demanding a reevaluation of PRO quality objectives along the lines outlined above. The AARP is also requesting a smoothing out of variations in physicians' practice patterns and a mandate to review outpatient services. It should be emphasized that the population of Medicare recipients has a very limited appeals process, namely, the PRO intervention. There is no access to the courts, and at present the providers do not have the right to represent their patients. It is also obvious that the elderly often do not have the stamina and will to see an appeals process all the way through.

Does the future provide any good news for change in the health care system as it serves the geriatric population? Every year we witness changes in the way Medicare and Medicaid are financed and managed. Leaders in the health economy field tell us that Medicare's health insurance fund, which pays hospital costs for the elderly and disabled, will be bankrupt by 1990 and possibly earlier.

Reforms are needed not only to control costs but to correct serious drawbacks in both Medicare and Medicaid. Much of Medicare's resources are spent on expensive hospital care and sophisticated techniques to treat patients in the last phase of a terminal illness. Some 9% of the elderly consume 70% of Medicare's funds. Medicare's most serious flaw is its lack of coverage for nursing home care and rehabilitative treatment for chronic disease, even though 80% of problems affecting the elderly on Medicare are long-term ailments such as diabetes, stroke, or arthritis.

In 1984, former Colorado governor Richard Lamm, speaking at a meeting of the Colorado Health Lawyers

Association, said the terminally ill elderly "have a duty to die." He said, "We are approaching the day when medical technology can keep people alive in hospitals hooked up to tubes and things far beyond when any quality of life is left at all." On the question of how many heart transplants the nation can afford, Lamm said that the elderly should "get out of the way . . . and let the other society—our kids—build a reasonable life."[6]

Riddick[7] has called attention to the role of rationing in some countries, where scarce resources are doled out in limited quantities. Either there is a long queue and prolonged waiting time for elective procedures, or there is simply failure to provide the service at a certain point. For example, renal dialysis is not available in Great Britain for those beyond age 55. When asked for the justification of this cutoff, a British physician said, "We all get a bit crumbly around that age, and patients of that age are not suitable candidates for dialysis."[7] He added that as more and more medical care is delivered on a price-sensitive basis, as research-based institutions are increasingly squeezed, there may not be much extension of the "gee-whiz" technology to the general public.

A *Wall Street Journal* editorial[8] suggests that since the Medicare regulatory framework is not working, we should try deregulated competition in health care. The article agrees that up until now most Medicare reforms have taken the form of price controls, which is window dressing. It suggests dismantling the entire Medicare bureaucracy and dividing the budgeted dollars among the beneficiaries as a yearly health stipend. This program would allow the Medicare population to do their own shopping and would throw the entire health care field open to the marketplace.

Payment reforms as currently practiced are certainly not the answer. Anderson and Raposa[9] point out that these result in decreased funding for new technology and graduate medical education, and in uncompensated care. The result will be a decline in the development and marketing of new technology, the size of graduate medical education programs, the amount of hospital-based research, and the number of hospitals financially willing and able to care for the elderly and indigent population. At the same time there will be more nonhospital services as they unbundle their services to maximize reimbursement. Competition will grow as providers lower their pricing with an eye to the competing market. There will follow a redistribution of wealth in the health care industry—among the hospitals, across regions of the country, between urban and rural settings, and among physician specialties. The formula would seem to be: more elderly people and more technology equals more expensive health care. It seems apparent that no one knows what the future health care sector should look like or even how to gain control over the health care system.

Physicians should be in the forefront in demanding that quality not be compromised for cost containment. They should lend their support to those organizations such as the AARP and the National Council of Senior Citizens that are struggling so hard to correct the flaws in the Medicare legislation. Finally, as Butler[10] recently pointed out, physicians must be ever vigilant against "ageism," which is defined as institutionalized and individual prejudice against the elderly, stereotyping, myth making, distaste, and/or avoidance.

JOHN T. PRIOR, MD
Medical Director
Empire State Peer Review Organization

Pathologist, Director of
Laboratories
Community-General Hospital
Broad Rd
Syracuse, NY 13215

1. Medicare in need of reform. *American Medical News* 1985; 28(33):4.
2. Tax Equity and Fiscal Responsibility Act of 1982, Pub L No. 97-248, 96 Stat 324 (codified as amended in scattered sections of 42 USC); Omnibus Legislation, 1983.
3. Staff of Senate Special Committee on Aging: *Impact of Medicare's Prospective Payment System on the Quality of Care Received by Medicare Beneficiaries*, 99th Cong, 1st sess (1985) (John Heinz, Chairman).
4. Owen J: Policy outlook PRO's: A hospital perspective. *AMPRA Review* 1985; 2:3.
5. Ostrander V: Medicare DRGs pose problems for elderly [interview by Jeffrey Finn]. *Hospitals* 1985; 59(24):75-76.
6. Lamm's "duty to die" remarks draw fire. *Hospitals* 1984; 58(9):24.
7. Riddick FA Jr: Future directions in graduate medical education. *Arch Pathol Lab Med* 1986; 110:1006-1011.
8. Wohl S: Deregulate to cure health care paralysis. *Wall Street Journal*, October 9, 1985, p 32.
9. Anderson GF, Rapoza CR: Health care payment reform: Projections for the next 15 years. *NY State J Med* 1985; 85:644-646.
10. Butler RN: Thoughts on aging. *Am J Psychiatry* 1978; 135(suppl):14-16.

A view of the proposed New York State regulations pertaining to the activities of medical students

On June 2, 1987, the Ad Hoc Committee on Emergency Services, appointed by New York State Health Commissioner David Axelrod, announced several recommendations that were subsequently inserted into draft revisions of the New York State Health Code, Section 405. In a separate position statement, the Associated Medical Schools (AMS) of New York has responded to those sections of the Ad Hoc Committee recommendations and the code revisions dealing with the work of residents and attending physicians.[1] AMS, however, is also gravely concerned with other proposed regulations in Section 405 pertaining to medical students. This position statement emphasizes these concerns.

Section 405, subsection (h), dealing with medical students, states that students may only

> observe the provision of patient care services by an appropriately licensed practitioner granted privileges pursuant to that license to practice in the hospital, but shall not be assigned or directed to provide any patient care services for which a license is required pursuant to the requirements of New York State Education Law with the exception of taking patient histories and conducting physical examinations. These can be performed only under the supervision and countersignature within twenty-four hours by at least a second year resident.

This requirement, if enacted, would result in medical students who have completed more than an entire year of clinical training assuming no more responsibility than that which had existed during their second year of medical education. Such a restriction violates nationally accepted standards for the clinical education of medical students.

Students clearly are not licensed to practice medicine and should not be allowed to function independently in any procedure for which a license is required. It is equally unacceptable, however, that students under the appropriate supervision of licensed, credentialed physicians should be unable to participate in graded clinical experiences which will, in turn, legitimize their receipt of the MD degree and prepare them for their residency. Indeed, it is a fundamental principle of the medical education experience across the continuum from medical school through residency training that students and residents have gradually increasing responsibility in order to increase their knowledge, skills, and experience under appropriate supervision.

One of the most valuable aspects of medical school is the senior year clinical experience during which time students, under close supervision, take night call and assist residents and the attending staff in the clinical care of patients. There has rarely, if ever, been an adverse outcome resulting from these arrangements. When isolated events have occurred, they have been the result of inadequate supervision. To prevent students from assuming clinical responsibilities until they start their residencies will ill serve the public as well as our students. A more intense and prolonged training period will be needed in order to allow residents to progress to levels of achievement and responsibility which are now typical of medical school graduates under our current system of medical education. The result will be increased costs and less adequate care of patients by less well prepared graduates. These problems will be compounded if the medical schools of New York—as a result of these new restrictions—become less attractive to top quality candidates from New York and elsewhere.

The Associated Medical Schools strongly urges that this subsection be revised to allow medical students to engage in such activities and perform those procedures deemed appropriate by national standards and by the schools of medicine under careful supervision and, when appropriate, under direct observation of licensed and appropriately credentialed physicians.

AMS recognizes the valid concerns about proper supervision. Its members will continue to review current procedures and work with appropriate public and private agencies to assure the best possible supervision of students.

ASSOCIATED MEDICAL SCHOOLS OF NEW YORK
Bellevue Hospital Center
First Ave and 27th St
New York, NY 10016

---

1. *Position Statement on Proposed Regulations Governing the Activities of Residents*. New York, Associated Medical Schools of New York, August 3, 1987.

The "exaggerated death" of peritoneal lavage in the management of trauma patients

In this issue of the *Journal*, Barker and Fromm[1] present a patient with an unusual sequela—bile peritonitis—from a blunt trauma injury to the liver. The patient survived and is doing well.

A measure of success in the treatment of any patient is the restoration of wellness, which in this instance was achieved. Given even the best of descriptions, it is generally not possible to give a reader or listener an exact feel for a clinical scenario. While adhering to basic principles in the resuscitation, evaluation, and treatment of trauma patients, clinicians vary their management styles according to available personnel, equipment, and clinical experiences.

The management of the patient presented here illustrates an evolutionary change in the diagnosis and treatment of patients with blunt torso trauma. Just over two decades ago, the diagnosis of this patient's liver injury would have been made by four-quadrant abdominal paracentesis or by performing an exploratory laparotomy. The first of these diagnostic modalities is of historic interest, having been replaced and incorporated in part in the performance of a formal diagnostic peritoneal lavage. Now, with the growing acceptance of computed tomography (CT) scanning as a diagnostic method, the role of peritoneal lavage is undergoing scrutiny.

At the Shock Trauma Center of the Maryland Institute for Emergency Medical Services Systems in Baltimore, diagnostic peritoneal lavage is used liberally to diagnose intra-abdominal injury. Collectively, the center's trauma surgeons have employed the test several thousands of times over the past two decades to assess intra-abdominal injury. The once-favored, classic open method described by Root and colleagues in 1965[2] has given way to a semi-open technique developed by Cox and Dunham[3] at our center.

As in many other centers, lavage is performed in our center in patients with altered sensoriums, including patients with trauma due to head injury and/or drug use (usually alcohol); patients with multiple trauma (particularly above and below the diaphragm); patients with significant abdominal pain and/or tenderness without distention, who have stable vital signs; and patients who must undergo prolonged surgical procedures to treat nonabdominal injuries.

Using quantitative criteria to interpret lavage results, primarily a red blood cell (RBC) count of $\geq 100{,}000/\text{mm}^3$, Fischer and colleagues[4] noted false-negative and false-positive results of 1.2% and 0.2%, respectively, among 2,586 patients who underwent lavage for suspected intra-abdominal injury. In a report from our center, Cox[5] noted slightly higher false-negative and false-positive results of 0.3% and 3.3%, respectively, among 640 patients with intra-abdominal injuries. The criterion for exploration in those patients was a red blood count of $\geq 50{,}000/\text{mm}^3$.

After his motorcycle crashed, the patient described by Barker and Fromm[1] was admitted with vague right upper quadrant pain and mild tenderness. His hematocrit was low (37%). Given this scenario, some would have been more aggressive in diagnosing intra-abdominal injury, often within the first hours after admission. In the past, surgeons would have performed peritoneal lavage shortly after admission. Now, some would evaluate the abdomen using CT scanning. If lavage had been performed, a positive result would have been obtained in one of three ways: spontaneous drainage of blood through the lavage catheter on its insertion, aspiration of gross blood prior to performing the lavage, or lavage effluent containing gross blood. This would have led to the performance of an exploratory laparotomy and discovery of the liver injury. Most likely, the bleeding would have stopped or been minimal and easily controlled by compression of the organ (manually or by placing gauze packs about the right lobe for 10–15 minutes). The abdomen would have been closed without drains.[6,7] The biliary problem would have become apparent days later, leading to a second laparotomy.

Barker and Fromm adopted a less aggressive style of management in their young patient with minimal abdominal signs and symptoms, normal vital signs, and a low hematocrit. The patient was admitted and carefully monitored with the performance of repeated abdominal examinations and the tracking of serial vital signs and hematocrit determinations. Twenty-four hours after admission, a CT scan of the abdomen was obtained, which demonstrated the liver defect. If the patient had been transferred to this center at that time, with stable vital signs and having required only two units of blood, most of the clinicians here would have continued to manage the patient in a cautiously expectant fashion, as was done. If this rather unusual complication had not developed, he would have had no abdominal surgery. As it turned out, he had one surgical procedure compared to the two he would have had if he had undergone an early peritoneal lavage.

The report of Barker and Fromm[1] contributes to the growing body of literature that is helping to define the role of abdominal CT scans in the diagnosis and treatment of patients who sustain blunt torso trauma. Reports extolling the virtues of CT scanning in diagnosing intra-abdominal injuries continue to increase,[8–10] noting the test's ability to detect bleeding from liver injuries that do not require surgery.[11] The reports show that, in contrast to peritoneal lavage, enhanced CT studies can usually delineate specific organ injuries with great clarity. Trunkey and Federle[10] commented that the achievement of acceptable results with CT requires high-quality studies and the immediate review of equivocal studies by a staff radiologist with expertise in reading such scans.

The enthusiasm of some clinicians concerning the role of CT scanning in the diagnosis of abdominal injuries may be summarized by Trunkey and Federle's[10] editorial statement: "CT scanning is a different modality [compared to peritoneal lavage] and represents the next revolutionary step in [the] diagnosis of trauma." Is it time to shelve peritoneal lavage? To answer this question one should consider a previous statement by Blaisdell and Trunkey.[12] They noted, "We remain surprised at the reluctance of surgeons to explore the abdomen of a traumatized patient and at the same time readily accept a negative exploration rate of 20–30% for appendicitis as representing good practice." [12] This statement alludes to a cardinal principle of trauma care: one must be aggressive in identifying potentially life-threatening injuries. The introduction of peritoneal lavage has been a major advance in the management of trauma victims. As indicated, using quantitative criteria for interpretation, results greatly surpassing the standard for appendicitis have been achieved.

Currently, there is no single test without associated morbidity that is 100% sensitive and specific in detecting all intra-abdominal injuries. The introduction of peritoneal lavage allowed for a major leap forward to be made in the rapid diagnosis of life-threatening injuries. The use of CT allows for another great advance in trauma care: a reduction in the incidence of nontherapeutic laparotomies. One should remember, however, that it is neither practical nor safe to interrupt the resuscitation/evaluation of some patients by moving them to the CT scanning suite. Among critically ill patients, a quickly performed peritoneal lavage will indicate whether emergency surgery is necessary. Not uncommonly, surgery is avoided in patients who appear to need a laparotomy. An obsession with avoiding nontherapeutic laparotomies can lead to the sin of omission in the treatment of a life-threatening injury. While one does not have to apologize for an occasional nontherapeutic laparotomy, one must seek forgiveness for missing an easily identifiable life-threatening injury.

When a considerable number of clinicians have gained much needed experience with CT scanning of the abdomen in the management of trauma patients, one will then appropriately see a significant reduction in the performance of peritoneal lavage. Clinical expertise with CT scanning is not sufficiently advanced among clinicians who treat trauma victims.

Mark Twain once cabled from Europe, "The report of my death was an exaggeration." Indeed, the demise of peritoneal lavage should be considered the same.

CARL A. SODERSTROM, MD
PHILIP R. MILITELLO, MD
University of Maryland
School of Medicine
Department of Surgery/Traumatology
Maryland Institute for Emergency
Medical Services Systems
22 S Greene St
Baltimore, MD 21201

1. Barker SL, Fromm D: Bile peritonitis following expectant management of liver fracture. *NY State J Med* 1987; 87:565-567.
2. Root HD, Hauser CW, McKinley CR, et al: Diagnostic peritoneal lavage. *Surgery* 1965; 57:633-637.
3. Cox EF, Dunham CM: A safe technique for diagnostic peritoneal lavage. *J Trauma* 1983; 23:152-154.
4. Fischer RP, Beverlin BC, Engrav LH, et al: Diagnostic peritoneal lavage: Fourteen years and 2,586 patients later. *Am J Surg* 1978; 136:701-704.
5. Cox EF: Blunt abdominal trauma: A five-year analysis of 870 patients requiring celiotomy. *Ann Surg* 1984; 199:467-474.
6. Dunham CM, Militello P: Surgical management of liver trauma. *Am Surgeon* 1982; 48:435-439.
7. Fischer RP, O'Farrell KA, Perry JF Jr: The value of peritoneal drains in the treatment of liver injuries. *J Trauma* 1978; 18:393-398.
8. Federle MP, Crass RA, Jeffrey RB, et al: Computed tomography in blunt abdominal trauma. *Arch Surg* 1982; 117:645-650.
9. Goldstein AS, Sclafani SJ, Kupferstein NH, et al: The diagnostic superiority of computerized tomography. *J Trauma* 1985; 25:938-946.
10. Trunkey D, Federle MP: Computed tomography in perspective [editorial]. *J Trauma* 1986; 26:660-661.
11. Federle MP: Computed tomography of blunt abdominal trauma. *Radiol Clin North Am* 1983; 21:461-475.
12. Blaisdell FW, Trunkey DD: Preface, in Blaisdell FW, Trunkey DD (eds): *Trauma Management. Vol I: Abdominal Trauma.* New York, Thieme-Stratton, 1982.

Nonoperative management of blunt abdominal trauma: The cost/benefit margin

The appropriate utilization of computed tomography (CT) in the management of blunt abdominal trauma is currently at issue. This radiologic instrument allows visualization of the retroperitoneal as well as intraperitoneal spaces and may help identify specific organ injury, particularly to solid viscera. Formerly, diagnostic peritoneal lavage (DPL) was almost exclusively relied on to determine the need for urgent celiotomy following both blunt and penetrating injuries. Numerous reports attest to the exceptional accuracy of DPL in detecting or excluding intraperitoneal hemorrhage.[1-4] However, DPL's sensitivity for even small amounts of blood can lead to unnecessary surgery in as many as 12% of patients in whom DPL is the sole diagnostic criterion.[1,4] At laparotomy, these patients usually have self-limited injury to the liver or spleen. CT enthusiasts argue that in selected patients this consequence can be avoided with serial CT examination and clinical observation. But careful application of CT is required to avoid more serious complications. This is true for the acute as well as convalescent phases of trauma management; the report by Barker and Fromm[5] in this issue of the *Journal* underscores this fact.

CT accuracy is highest for trauma to solid viscera and is reported to exceed 95% for splenic injury.[6,7] But these exemplary results have only been achieved in uncontrolled, retrospective series. In three prospective comparisons of DPL and CT following blunt trauma, this success was not duplicated.[8-10] In fact, CT was only 40-60% sensitive in the detection of injury mandating operative intervention, compared with a 90-100% accuracy rate for DPL. In these and other reports, CT has failed to demonstrate a substantial number of liver injuries that required hepatotomy for direct suture control of bleeding, parenchymal debridement, and perihepatic drainage.[11-13] Moreover, hemoperitoneum in excess of 500 cc has escaped detection by CT. This may have particular significance in hepatic trauma, in which the quantity of hemoperitoneum seen on CT may be a decisive factor for laparotomy. False-positive CT studies of hepatic injury have also been reported.[11] Scintigraphy provides an alternative to CT in the assessment of solid organ injury. It has achieved excellent results in direct comparison to CT and has the advantage of being better able to assess the agitated patient.[13] However, its scope is limited to visualization of the liver, spleen, and kidneys.

Apparent discrepancies in the accuracy of CT can be attributed to differences in patient selection, scanning equipment, and institutional experience. Of greater significance, patient safety concerns and logistical difficulties can arise with untimely CT usage. Experienced trauma personnel must be available to monitor the patient throughout the obligatory 45 to 60 minutes required to execute the basic study. The location of the CT suite and the need to provide ongoing skilled patient care can be restrictive. The inconsistent availability of technical personnel, radiologists expert with interpretation, and the scanner itself can be limiting. The uncooperative patient will further compromise accuracy and may require sedation, with its attendant risks. Finally, the necessary administration of a relatively large volume of oral as well as intravenous contrast-enhancing agents has its own risks. Even under optimal conditions, reliance on CT as the primary triage tool for blunt abdominal trauma has incurred an unnecessary laparotomy rate of 19%.[6] It is imperative that institutions research their own resources and capabilities.

Expectant management of hepatic trauma mandates careful consideration. The liver is the intraperitoneal organ most frequently injured following blunt trauma; coincident damage to remaining intra-abdominal viscera is frequent. Pancreatic ductal disruption or hollow visceral perforation can be life-threatening, yet poorly appreciated by CT. Moreover, liver complications of recurrent bleeding, hematobilia, bile leakage, and abscess formation can ensue, further obfuscating clinical evaluation. Whether nonoperative treatment of liver injuries is cost effective is not known. Considerable clinical and laboratory monitoring together with multiple radiographic studies are required to optimize patient safety. Clearly, a single patient death due to delay in operation will nullify the cost savings of innumerable, unrewarding abdominal explorations. The paradox is that the patient in whom CT-directed expectant management is most appropriate is the individual in whom the morbidity associated with delayed laparotomy is negligible.

Clinical criteria for nonoperative management of liver injuries have been defined at the San Francisco General Hospital.[14] Patients who sustain blunt abdominal trauma, are hemodynamically stable, and have no other indications for laparotomy are candidates for CT evaluation. Observation is permissible if CT reveals a simple parenchymal laceration or intrahepatic hematoma without continued hemorrhage, and if the amount of intraperitoneal blood is less than 250 cc as estimated by restriction to the Morison pouch. In a series of 24 patients who fulfilled these guidelines, none required laparotomy or transfusions for hepatic injuries, and no complications developed. Notably, these 24 patients represent only 6% of the liver injuries evaluated during the 30-month period covered by this study.

In summary, DPL rapidly and accurately discerns the presence of intraperitoneal hemorrhage in patients who suffer blunt injury. A small number of such patients with minor liver or spleen lacerations may be subjected to unnecessary operation based on DPL results alone. However, this occurs in an acceptably low percentage. DPL also measures leukocytic and enzymatic response, reflecting damage to other intra-abdominal organs that are less well defined by CT. Conversely, the qualities of CT and scintigraphy make these diagnostic aids suitable for managing the stable patient with suspected, isolated hepatosplenic injury. In the level I patient, CT can be a valuable adjunct to DPL, but its inconsistent record and logistic problems speak against its being the sole instrument used for decision making.

JOHN A. MARX, MD
ERNEST E. MOORE, MD
Denver General Hospital
University of Colorado
Health Sciences Center
Denver, CO 80204

1. Fischer RP, Beverlin BC, Engrav LH, et al: Diagnostic peritoneal lavage: Fourteen years and 2,586 patients later. *Am J Surg* 1978; 136:701-704.
2. Parvin S, Smith DE, Asher WM, et al: Effectiveness of peritoneal lavage in blunt abdominal trauma. *Ann Surg* 1975; 181:255-261.
3. Powell DC, Bivins BA, Bell RM: Diagnostic peritoneal lavage. *Surg Gynecol Obstet* 1982; 155:257-264.
4. Thal ER, Shires GT: Peritoneal lavage in blunt abdominal trauma. *Am J Surg* 1973; 125:64-69.
5. Barker SL, Fromm D: Bile peritonitis following expectant management of liver fracture. *NY State J Med* 1987; 87:565-567.
6. Federle MP, Crass RA, Jeffrey RB, et al: Computed tomography in blunt abdominal trauma. *Arch Surg* 1982; 117:645-650.
7. Jeffrey RB, Laing FC, Federle MP, et al: Computed tomography of splenic trauma. *Radiol* 1981; 141:729-732.
8. Davis RA, Shayne JP, Max MH, et al: The use of computerized axial tomography versus peritoneal lavage in the evaluation of blunt abdominal trauma: A prospective study. *Surgery* 1985; 98:845-850.
9. Fabian TC, Mangiante EC, White TJ, et al: A prospective study of 91 patients undergoing both computed tomography and peritoneal lavage following blunt abdominal trauma. *J Trauma* 1986; 26:602-608.
10. Marx JA, Moore EE, Jorden RC: Limitations of computed tomography in the evaluation of acute abdominal trauma: A prospective comparison with diagnostic peritoneal lavage. *J Trauma* 1985; 25:933-937.
11. Moon K Jr, Federle MP: Computed tomography in hepatic trauma. *AJR* 1983; 141:309-314.
12. Toombs BD, Sandler CM, Rauschkolb EN, et al: Assessment of hepatic injuries with computed tomography. *J Comput Assist Tomogr* 1982; 6:72-75.
13. Uthoff LB, Wyffels PL, Adams CS, et al: A prospective study comparing nuclear scintigraphy and computerized axial tomography in the initial evaluation of the trauma patient. *Ann Surg* 1983; 198:611-616.
14. Meyer AA, Crass RA, Lim RC, et al: Selective nonoperative management of blunt liver injury using computed tomography. *Arch Surg* 1985; 120:550-554.

Joseph F. Shanaphy, MD

Joseph F. Shanaphy, MD, Vice-President of the Medical Society of the State of New York (MSSNY), died on Friday, July 10, 1987, after a brief illness. He had been elected Vice-President of the Society at the 1987 House of Delegates meeting in New York City in March, the same month he retired as director of the Department of Obstetrics and Gynecology at St Vincent's Medical Center of Richmond County. Dr Shanaphy had served in the latter post for ten years and had practiced obstetrics and gynecology on Staten Island since 1952.

Dr Shanaphy, who was born in 1921 in West Hoboken, New Jersey, received a BS degree from the College of the Holy Cross and went on to study medicine at the Long Island College of Medicine, from which he received an MD degree in 1945. He did his internship and residency in obstetrics and gynecology at the US Public Health Service Hospital on Staten Island, and later attended the New York University Basic Science Post-Graduate School, from which he received an MSc degree in 1951. He served as a Senior Surgeon in the USPHS from 1945 to 1953, spending part of this time as Chief of Obstetrics and Gynecology at the US Public Health Service Hospital in New Orleans.

Dr Shanaphy was active in organized medicine, serving as the President of the Richmond County Medical Society in 1964. In 1975 he was elected Vice-Speaker of the MSSNY House of Delegates, and was speaker from 1976 to 1981. Subsequently he was a member of the MSSNY Board of Trustees from 1981 until his election as Vice-President. He was an alternate delegate to the American Medical Association House of Delegates in 1976 and 1977, and from 1979 to 1981, and a full delegate from 1982 until his death. Dr Shanaphy was widely known and respected in his specialty. He served in the departments of obstetrics and gynecology at several hospitals, including Richmond Memorial Hospital and St Vincent's Medical Center in Staten Island and New York University Hospital in Manhattan. He was also a clinical instructor in obstetrics and gynecology at New York University-Bellevue Hospital Center. Dr Shanaphy is survived by his wife, Ellen, and four children, Jean, Nancy, Patricia, and Joseph, Jr.

Pascal James Imperato, MD
Editor

"The Board of Trustees of the Medical Society of the State of New York wishes to express its deep sorrow at the untimely passing of the Society's Vice-President, Joseph F. Shanaphy, MD. Dr Shanaphy served the Society and the people of New York State with compassion and skill for many years. He was greatly admired for his warm and giving personality, his intelligence, and his leadership. Dr Shanaphy was a friend to his patients and colleagues alike. Our profound sympathy goes to his widow, Ellen, and their four children."

Richard D. Eberle, MD
Chairman
Board of Trustees
Medical Society of the State of New York

"The Officers and Councilors of the Medical Society of the State of New York extend their deepest sympathy to the family of Joseph F. Shanaphy, MD, our esteemed colleague, who served the Society and the medical care interests of the people of New York State with distinction for many years. He was greatly gifted and admired by his patients, the citizens of his community of Staten Island, and by his fellow physicians. This admiration is his lasting tribute."

Samuel M. Gelfand, MD
President
Medical Society of the State of New York

Physicians' attitudes toward and knowledge about generic drug substitution

DANIEL FRIEDMAN, MD; ARNOLD JAFFE, PhD; SUSAN STEINHARDT, MPA

ABSTRACT. Recent federal and state legislation has caused considerable confusion for those who prescribe, dispense, or purchase prescription drugs. This study investigates physician understanding of and attitudes toward prescribing of generic drugs. Respondents to a survey included 245 physicians from several disciplines who were members of the Suffolk County Medical Society. Only 12.9% of respondents reported having knowledge of Food and Drug Administration procedures for testing generic drug products. Physicians are mixed in their beliefs about the efficacy of generic drugs, and the level of prescribing common generic drugs is low. A majority of respondents reported a willingness to prescribe generic drug products on a patient's request, but few such requests are made. Generic drug prescribing habits differed among the specialties included in the sample. It is recommended that information on testing and efficacy of generic drugs be provided to physicians. Perhaps such information could facilitate the decision-making process when physicians prescribe drugs.

(NY State J Med 1987; 87:539–542)

In 1938 Congress passed legislation to ensure the safety of drugs marketed in the United States.[1] In 1962, federal legislation mandated that efficacy (as well as safety) must be certified.[2] The Drug Price Competition and Patent Term Restoration Act of 1984 was intended to make prescription drugs more affordable to the public.[3] One of its primary goals was to simplify the application process by which generic drug products can gain the approval of the Food and Drug Administration (FDA). Prior to this law a New Drug Application (NDA) often required 150,000 pages of detailed documentation. The new law provides for an Abbreviated New Drug Application (ANDA), which is appropriate only for generic drug products. An ANDA does not require manufacturers to test for safety or effectiveness. They are only required to demonstrate that the generic drug product has bioequivalence of +20% or +30%, depending on the innovator product. The percentage varies by class of drug. (See the "75-75" rule below.)

The essence of the new FDA regulations includes the following:[3]

- Deriving efficacy data from 18 to 24 healthy men, ages 21 to 35, who are within 10% of their ideal body weights. Sampling of a pediatric or a geriatric population is not considered.
- Accepting a generic drug product as "pharmaceutically equivalent" if it contains the same active ingredient in the identical concentration and form as the innovator product. Excipients, defined as inert ingredients, are expected to differ.
- Testing and comparing blood concentrations of subjects in the sample. Clinical trials on patients may be required for some drugs.
- Approving the generic drug product contingent on its meeting the "75–75" bioavailability rule. The generic drug product is required, in only 75% of the test subjects, to achieve blood concentrations not less than 75% or greater than 125% of those obtained by the brand name product. (For some psychotropics, the rule is relaxed to "70–70.") Bioavailability is distinct from bioequivalence. It is defined by the Federal Registry as "the rate and extent to which the active ingredient or therapeutic moiety is absorbed from a drug product and becomes available at the site of drug action."

Passage of this new legislation coincided with the termination of patent rights on a large number of brand name drugs. There has since been a flood of ANDA applications. During the past two decades, all 50 states have passed legislation that either permits or requires pharmacists to substitute a generic drug for its brand name congener. The goal of such legislation is to enhance attempts to reduce consumer cost without sacrificing quality, reliability, or efficacy.

Legislation has led to increased confusion relating to issues of substitution policy, bioavailability, bioequivalency, and economic and fiscal impact. Twenty percent of the $17.6 billion spent for drugs during 1984 was for generic products—a figure that is expected to grow to more than $10 billion (a 25% increase) by 1995.[4,5] Though it is difficult to achieve a relative exactness of dosage of gener-

---

From the Department of Family Medicine, State University of New York at Stony Brook, Stony Brook, NY.

Address correspondence to Dr Friedman, Department of Family Medicine, Health Science Center, State University of New York at Stony Brook, Stony Brook, NY 11794-8461.

ics and innovators, it is not clear that the efficacy of generic drugs depends on such strict equivalence.[6,7] Mazullo[8] points out, in a cautionary note, that the most important determinant of drug pricing is neither the pharmaceutical manufacturer nor the physician; it is the pharmacist.

As prescriptions for generic drugs are refilled, manufacturers' products are often interchanged without the knowledge of either the physician or the patient. Both need to know the details of the laws within their state to benefit from the laws' intent and to avoid unanticipated consequences. Unfortunately, the lack of uniformity in state laws provides mechanisms by which both advocates and opponents of generic prescribing attempt to thwart the intended legislative purpose.[9]

Francetic et al[10] found wide variations in retail pharmacy prices. In nearly 30% of the cases, savings were not passed on to the customer. The additional administrative costs to state and federal governments for generic drug prescribing programs are rarely calculated into the cost of generic prescribing. Brater and Pettinger[11] question who is liable when a pharmacist "selects an equivalent" drug product as authorized by state laws. Effective July 1, 1987, New York State legislation relieves the physician of such liability.

In a study that covered 39 states and almost 900,000 prescriptions, Bloom et al[12] concluded that it is foolish to assume that generic prescribing will result in significant cost savings. Lasagna[13] believes that pharmacists favor generic drugs because their markup tends to be higher. In a concise review of these issues, Schwartz[14] delineates the dilemmas that face physicians as they try to maintain knowledge and awareness of the potential differences and dangers when substituting one drug product for another.

Do physicians understand the FDA generic drug approval procedures, and are they aware that the guidelines include a variable standard for bioavailability of the generic drug as compared to the innovator product? How have legislative and administrative issues influenced the daily prescribing habits and attitudes of the practicing physician? This study explored physicians' opinions about prescribing generic drug products.

METHODS

A questionnaire was mailed to a sample of 500 members of the Suffolk County Medical Society. Names were chosen with the use of a random numbers table from a membership list of approximately 1,600. After two mailings, a total of 245 responses were received. The questionnaire was designed to ascertain respondents' knowledge of several requirements related to prescription writing; their attitudes toward several specific drugs that have been the center of controversy concerning the bioequivalency issue; and the influence of patient opinions and other factors on the physician's prescribing behavior. Age, specialty, practice setting, terminal year of residency training, board certification status, and the average number of prescriptions written each day were ascertained. The data were analyzed to discern differences that relate to these characteristics.

RESULTS

Table I provides an analysis of respondents by specialty, practice type, and age. Specialties were distributed rather evenly between primary care and non-primary care subspecialties. Psychiatrists represented the smallest percentage of respondents (5.5%) and surgeons the largest (17.4%). Practice types were about evenly divided between solo practices and groups of two or more physicians.

TABLE I. Characteristics of Respondents

	N	%
Practice specialty		
General Internal Medicine	36	14.9
Family/General Practice	34	14.1
Obstetrics-Gynecology	28	11.6
Pediatrics	28	11.6
Psychiatry	13	5.5
Surgery	42	17.4
Subspecialty Internal Medicine	34	14.1
Other	26	10.8
No response	4	—
Total	245	100.0
Practice type		
Solo/private	112	46.5
Large private group (five or more)	26	10.8
Small private group (two to four)	88	36.5
Other	15	6.2
No response	4	—
Total	245	100.0
Age (yr)		
25–40	44	18.6
41–55	112	47.2
56 and over	81	34.2
No response	8	—
Total	245	100.0
Average number of prescriptions written daily		
5 or less	53	21.8
6–15	94	38.7
16–25	54	22.2
26 or more	42	17.3
No response	2	—
Total	245	100.0

Table II analyzes the respondents' beliefs regarding the efficacy of generic drug products. Respondents were evenly divided in their beliefs concerning the reliability of generic as compared to brand name drugs. A small majority (56.8%) agreed with the Food and Drug Administration's assertion that equivalent bioavailability indicates equal therapeutic efficacy. Only 12.9% of the respondents stated that they were familiar with the FDA procedure for determining a generic drug's bioequivalence.

TABLE II. Respondents' Beliefs and Knowledge About Generic Drugs

	N	%
Generic drugs as reliable as trade name drugs		
Agree	104	44.0
Disagree	105	44.5
Not sure	27	11.5
No response	9	—
Total	245	100.0
Equal bioavailability indicates equal efficacy		
Agree	129	56.8
Disagree	98	43.2
No response	18	—
Total	245	100.0
Respondent is knowledgeable about the FDA procedure for equivalence testing		
Yes	30	12.9
No	151	65.1
Not sure	51	22.0
No response	13	—
Total	245	100.0

TABLE III. Frequency of Prescribing Common Drugs Generically

	Penicillin		Diazepam		Digoxin		Theophylline	
	N	%	N	%	N	%	N	%
Never	30	13.7	59	29.1	83	52.5	64	37.9
Rarely	61	27.9	93	45.8	33	20.9	49	29.0
Often/very infrequently	128	58.4	51	25.1	42	26.6	56	33.1
No response	26	—	42	—	87	—	76	—
Total	245	100.0	245	100.0	245	100.0	245	100.0

The frequency of generic prescribing for several common medications (Table III) was relatively low. The majority of prescriptions for diazepam, digoxin, and theophylline were written for the trade name drug, while a majority of penicillin prescriptions were written for generic products. There were significant differences based on the number of prescriptions a physician wrote per day. Physicians who wrote few prescriptions (Table IV) reported the least knowledge of FDA procedures for equivalence testing ($\chi^2 = 13.41$, $p < 0.01$). Knowledge increased as the number of prescriptions written increased. Physicians' opinions did not differ according to their ages or practice type (solo vs group). Few respondents indicated that their willingness to prescribe generic drugs was influenced by state laws, FDA regulations, or the statements of pharmaceutical company representatives.

Differences in belief about the reliability of generic drug products by physicians' specialties are described in Table V. Pediatricians were most likely to believe that generic drug products are reliable while obstetricians-gynecologists and family/general practioners were least likely ($\chi^2 = 11.01$, $p < 0.05$).

The majority of physicians were influenced by a patient's request for a generic prescription (Table VI). More than 60% of respondents stated that they acceded to a patient's wish to receive generic drug products "often" or "very frequently." Patients do not, however, make such requests very frequently. Our sample reported that 72.8% of patients "rarely" or "never" ask for generic drug products (Table VII). Internists were most willing to prescribe generic drugs and were most frequently asked by patients for these drug products ($\chi^2 = 25.73$, $p < 0.01$). Though obstetricians-gynecologists are willing to prescribe generic products, they are least likely to report patient requests for generic drugs ($\chi^2 = 39.30$, $p < 0.01$).

DISCUSSION

The issue of deciding whether to prescribe a generic drug is complex. Some preparations are essentially equal in efficacy to the innovator drug, as in the case of oral penicillin. Some generic drugs are not bioequivalent, such as digoxin, ferrous sulfate, or thyroid hormone preparations. Other generic drugs, such as hydrochlorthiazide and phenytoin, are bioequivalent. Complexity is also the rule regarding cost savings. The use of generic drug products may result in significant cost savings. Pharmacy practices, however, may eliminate any savings to the patient.[12]

TABLE IV. Number of Prescriptions Written Daily Related to Respondents' Knowledge of FDA Procedures for Equivalence Testing

No. Prescriptions Written per Day	N	Knowledge of FDA Procedures Yes	No
5 or less	40	7.5%	92.5%
6–15	73	12.3%	87.7%
16–25	32	37.5%	62.5%
25	35	17.1%	82.9%
Total	180	16.7%	83.3%

$\chi^2 = 13.41$, $p < 0.01$.

In this survey, physicians reported considerable uncertainty about the procedures for testing and establishing efficacy of generic drug products. Only 12.9% of the sample reported knowledge of FDA procedures for testing these drugs, but the physicians were evenly divided in their beliefs about the effectiveness and reliability of substitute products.

The data from this study indicate that when physicians are asked by their patients to prescribe generic products, 60% often comply with their patients' requests. Although many physicians are willing to substitute generic drugs on the request of a patient, relatively few patients make the request. Physician willingness to prescribe generic products also varies with the type of drug in question—for example, generic prescriptions for penicillin are much more common than those for diazepam, digoxin, or theophylline.

Physician subspecialty and frequency of prescribing were related to the participants' attitudes and prescribing habits. In general, the more frequent prescriber and the primary care physician prescribed generic drugs most frequently. The less frequent prescriber and the subspecialist employed fewer generic drug products.

It is likely that a combination of both physician resistance and patient disinterest contributes to the low frequency of generic drug prescribing. A program of instruction may be necessary to improve both physician and patient awareness of the testing, efficacy, pricing, and other aspects of the generic drug dilemma. If generic drugs are to be prescribed optimally, both patients and

TABLE V. Differences in Opinion About Reliability of Generic Drugs By Specialty

Specialty	N	Generic Drugs as Reliable as Trade Name Drugs Yes	No
Family/General Practice	29	34.5%	65.5%
Obstetrics-Gynecology	24	33.3%	66.7%
Pediatrics	24	70.8%	29.2%
Internal Medicine	28	53.5%	46.4%
Surgery	39	59.0%	41.0%
Internal Medicine subspecialties	29	51.7%	48.3%
All specialties combined	173	50.9%	49.1%

$\chi^2 = 11.01$, $p < 0.05$.

TABLE VI. Frequency of Generic Prescribing Upon Patient Request By Specialty

Specialty	N	Never	Rarely	Often/ Very Frequently
Family/General Practice	32	12.5%	18.8%	68.7%
Obstetrics-Gynecology	25	4.0%	24.0%	72.0%
Pediatrics	27	11.1%	33.3%	55.6%
Internal Medicine	32	3.1%	12.5%	84.4%
Surgery	37	18.9%	48.6%	32.5%
Internal Medicine subspecialties	32	0.0%	34.4%	65.6%
All specialties combined	185	8.6%	29.2%	62.2%

$\chi^2 = 25.73$, $p < 0.01$.

TABLE VII. Frequency of Patient Requests for Generic Drug Products by Specialty

Specialty	N	Never	Rarely	Often/ Very Frequently
Family/General Practice	34	2.9%	61.8%	35.3%
Obstetrics-Gynecology	28	0.0%	96.4%	3.6%
Pediatrics	28	7.1%	71.5%	21.4%
Internal Medicine	36	2.8%	52.8%	44.4%
Surgery	42	16.7%	76.2%	7.1%
Internal Medicine subspecialties	34	0.0%	82.4%	17.6%
All specialties combined	202	5.4%	72.8%	21.8%

$\chi^2 = 39.30$, $p < 0.01$.

physicians will require instruction concerning the merits and disadvantages of generic prescribing. Opinions and practices need to be based on the full knowledge of the issues.

REFERENCES

1. Federal Food, Drug and Cosmetic Act of 1938, Public Act No. 717.
2. Harris-Kefauver Amendment of 1962, Public Law No. 87-781.
3. The Drug Price Competition and Patent Term Restoration Act of 1984, Public Law No. 98-417, 98 Stat 1585 (1984).
4. Collins PJ: Practical briefings. *Patient Care*, 1986; 20(1):21-22.
5. Sun M: Generics, Roche joust for valium market [news]. *Science* 1985; 228:472-473.
6. Chodos DJ: Generic substitution: A form of pharmaceutical Russian roulette? in Lasagna L (ed): *Controversies in Therapeutics*. Philadelphia, WB Saunders Co, 1980, pp 72-82.
7. Horvitz R: Generic substitution: A safe way to cut medical costs, in Lasagna L (ed): *Controversies in Therapeutics*. Philadelphia, WB Saunders Co, 1980, pp 63-71.
8. Mazullo JM: Generic substitution and Grandma's secret recipe, in Lasagna L (ed): *Controversies in Therapeutics*. Philadelphia, WB Saunders Co, 1980, pp 83-84.
9. Morgan JP, Schwartz LL, Sherman FT: A survey of generic drug legislation and geriatric pharmacotherapy: Opinions of those who generate the literature. *J Am Geriatric Soc* 1983; 31:535-539.
10. Francetic J, Lasagna L, Weintraub M, et al: Prescription prices under the New York generic substitution law. *Ann Intern Med* 1980; 92:419-423.
11. Brater DC, Pettinger WA: The generic prescribing issue. *Ann Intern Med* 1980; 92:426-427.
12. Bloom BS, Wierz DJ, Pauly MV: Cost and price of comparable branded and generic pharmaceuticals. *JAMA* 1986; 256:2523-2530.
13. Lasagna L: The economics of generic prescribing: Winners and losers [editorial]. *JAMA* 1986; 256:2566.
14. Schwartz LL: The debate over substitution policy. Its evolution and scientific basis. *Am J Med* 1985; 79:38-44.

The diagnostic significance of free erythrocyte protoporphyrin and the FEP/hemoglobin ratio in plumbism

SOLOMON RAJKUMAR, MD; VALERIE GEIBEL, PHD; BHASKAR DEVANAGONDI, MD;
BALAKRISHNA KAUL, PHD; AUDREY K. BROWN, MD

ABSTRACT. The diagnostic significance of elevations of free erythrocyte protoporphyrin (FEP) and the FEP/hemoglobin (Hb) ratio was evaluated in relation to anemia, iron deficiency, and iron deficiency anemia in 170 children with high blood lead levels (Pb > 30 mg/dL). Anemia was defined as an Hb level of less than 11 g/dL; transferrin saturations of less than 7% and 8–15% were considered as "definite" and "intermediate" iron deficiency, respectively. Anemia occurred in 6% (n = 10) of the children, iron deficiency in 29% (n = 49), and iron deficiency anemia in 9% (n = 16). The mean FEP as well as the FEP/Hb ratio were significantly higher ($p < 0.001$) in children with plumbism associated with anemia, iron deficiency, or iron deficiency anemia, than in those children with plumbism alone.

From these observations, we conclude that in children with high blood lead levels, there is a higher incidence of a biochemical iron deficiency state than iron deficiency anemia. Furthermore, iron deficiency associated with plumbism causes a significantly greater elevation of FEP and FEP/Hb ratio than plumbism or iron deficiency alone. Therefore, biochemical iron deficiency merits consideration in the assessment of FEP and the FEP/Hb ratio in pediatric lead poisoning.

(NY State J Med 1987; 87:542–545)

Elevation of free erythrocyte protoporphyrin (FEP) occurs not only in lead poisoning, but also in iron deficiency

---

From the Department of Pediatrics, State University of New York Health Science Center at Brooklyn, Brooklyn NY.

Address correspondence to Dr Rajkumar, Kings County Hospital Center (code 29), 451 Clarkson Ave, Brooklyn NY 11203.

anemia.[1-5] The incidence of iron deficiency anemia associated with plumbism is estimated by nationwide lead poisoning programs to be 4%.[6] Because iron deficiency anemia and lead poisoning occur in the same age groups, the interpretation of elevated FEP values in children with high blood lead levels is problematic. Elevated FEP values have not been shown to readily distinguish between the relative toxicity of lead and the degree of associated biochemical iron deficiency or iron deficiency anemia. This study was undertaken to evaluate the diagnostic significance of FEP and the FEP/Hb ratio in children with high blood lead levels.

PATIENTS AND METHODS

One hundred seventy children with lead levels greater than 30 mg/dL were studied. Forty-two children with lead levels of less than 29 mg/dL and with iron deficiency or iron deficiency anemia served as controls. Children with inherited hemoglobinopathies (eg, Hb SS, Hb SC, and Hb S-Thal diseases) were excluded from the study.

Venous blood samples were drawn from all subjects for the estimation of hemoglobin, hematocrit, lead level, serum iron, and total iron binding capacity (TIBC). The cyanmethemoglobin and Clay Adams microhematocrit methods[7,8] were used to estimate the hemoglobin level and hematocrit, respectively. Serum iron and TIBC were measured according to the method of Fischer and Price.[9] Lead levels were determined by atomic absorption spectrophotometric analysis.[10] Erythrocyte protoporphyrin was measured using the extraction technique.[11] Transferrin saturation was calculated as the ratio of serum iron to TIBC × 100. The FEP/Hb ratio was calculated as a ratio of FEP mg/dL to Hb g/dL.

Anemia was defined as a hemoglobin level of less than 11 g/dL.[12] Transferrin saturation of greater than 7% was used as an indicator of "definite" iron deficiency.[13] Values between 8% and 15% were considered as indicating "intermediate" iron deficiency.[13]

All data were punched onto cards, and statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS).[14]

RESULTS

The mean age of the children in the study population was five years and nine months (range, 1 yr, 2 mo–13 yr, 9 mo) and two years and five months (range, 6 mo–8 yr, 9 mo) for the controls. While it is generally believed that lead poisoning is not much of a problem after six years of age, 38% of the study population was over six years, one third being in class III of the Risk Classification defined by the Centers for Disease Control (CDC) of the Public Health Service.[15]

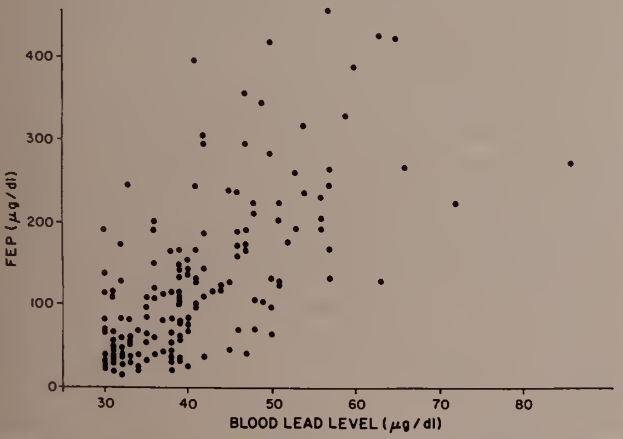


FIGURE 1. The relationship of lead levels to free erythrocyte protoporphyrin (FEP) in plumbism.

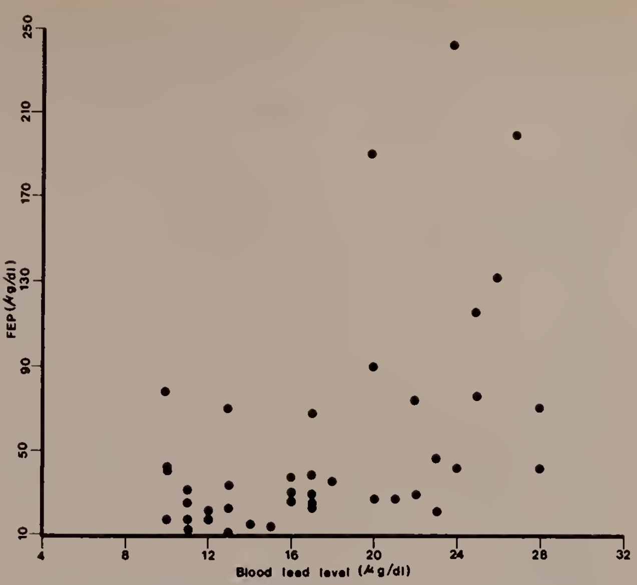


FIGURE 2. The relationship of lead levels to free-erythrocyte protoporphyrin (FEP) in iron deficiency.

Free Erythrocyte Protoporphyrin. The relationship of lead level to FEP for both study and control groups is shown in Figures 1 and 2. In both groups, higher values of FEP were associated with elevated lead levels; the relationship was of a curvilinear nature. The positive relationship was more pronounced in the study group than among controls (r = 0.7; controls, r = 0.5). A considerable variation in FEP values was observed for a given range of blood lead levels (Table I). FEP values greater than 50 mg/dL were found in 129/170 (76%) of the children in the study group and in only 12/42 (28%) of control children.

Incidence of Iron Deficiency. Forty-four percent (75/170) of children with high blood lead levels had anemia, iron deficiency, or iron deficiency anemia. Anemia occurred in 6% (n = 10), iron deficiency in 29% (n = 49), and iron deficiency anemia in 9% (n = 16).

FEP and FEP/Hb ratio. FEP and the FEP/Hb ratio for different groups of children are presented in Table II. The mean FEP for children with only lead burden was 100 mg/dL; for those with lead burden and anemia (but no iron deficiency or iron deficiency anemia), the FEP was 156 mg/dL. For children with increased lead burden and iron deficiency (but no anemia) the FEP was even higher: for intermediate iron deficiency it was 161 mg/dL (control, 47 ± 12 mg/dL); and for definite iron deficiency it was 218 mg/dL (control, 40 ± 7 mg/dL). The greatest increase in FEP was noted in children with both increased lead burden and obvious iron deficiency anemia: intermediate iron deficiency = 170 mg/dL (control, 44 ± 13 mg/dL); definite iron deficiency = 237 mg/dL (control, 92 ± 28 mg/dL). The relationship of FEP to biochemical iron deficiency, iron deficiency anemia, lead burden alone, lead burden plus iron deficiency, lead burden plus anemia, and lead burden plus iron deficiency anemia is illustrated by plotting 95% confidence limits (Fig 3).

TABLE I. Distribution of Number of Children According to Risk Classification of Free Erythrocyte Protoporphyrin Versus Blood Lead Level

Blood Lead (mg/dL)	Free Erythrocyte Protoporphyrin (mg/dL) ≥ 49	50–109	110–249	≥ 250	Total
30–49	41	47	42	6	136
50–69	0	2	15	15	32
≥70	0	0	1	1	2
Total	41	49	58	22	170

TABLE II. **Free Erythrocyte Protoporphyrin and Free Erythrocyte Protoporphyrin/Hemoglobin Ratio***

	Pb** = <29 mg/dL							Pb = >30 mg/dL						
	No.	Pb	Hb**	Iron	TIBC**	FEP	FEP/Hb Ratio	No.	Pb	Hb	Iron	TIBC	FEP**	FEP/Hb Ratio†
Plumbism														
No anemia														
No iron deficiency														
No iron deficiency anemia														
Hb > 11g														
TS** > 15%								95	40 ± 0.9	12.9 ± 0.1	94 ± 3	358 ± 5	100 ± 8	7.9 ± 0.7
Anemia														
Hb < 11g														
TS > 15%								10	42 ± 2.7	9.8 ± 0.4	89 ± 4	372 ± 21	156 ± 29	16.8 ± 3.8
Iron deficiency														
Hb > 11g														
TS, 8–15%														
(intermediate)	10	17 ± 1.9	12.1 ± 10.1	42 ± 2	365 ± 12	47 ± 12	3.9 ± 1.0	34	42 ± 1.9	12.4 ± 0.2	50 ± 2	389 ± 13	161 ± 21	13.2 ± 1.7
TS, < 7% (definite)	6	20 ± 1.7	12.4 ± 0.5	23 ± 3	425 ± 27	40 ± 7	3.3 ± 0.6	15	45 ± 3.7	12.7 ± 0.3	24 ± 2	435 ± 16	218 ± 39	17.6 ± 3.2
Iron deficiency anemia														
Hb < 11g														
TS, 8–15%														
(intermediate)	17	15 ± 1.2	10.2 ± 0.1	38 ± 2	354 ± 14	44 ± 13	4.3 ± 1.2	8	46 ± 3.4	10.5 ± .01	46 ± 4	381 ± 17	170 ± 46	13.9 ± 5.0
TS, < 7% (definite)	9	21 ± 2.1	9.7 ± 0.3	24 ± 2	449 ± 29	92 ± 20	9.9 ± 2.4	8	45 ± 4.7	10.2 ± 0.2	26 ± 2	450 ± 31	237 ± 65	23.6 ± 7.0

* Mean ± standard error of the mean.
† Analysis of variance $p < 0.0001$.
** Pb = lead, Hb = hemoglobin, TIBC = total iron binding capacity, FEP = free erythrocyte protoporphyrin, TS = transferrin saturation.

DISCUSSION

The erythrocyte protoporphyrin level, expressed as free erythrocyte protoporphyrin (FEP), is used as the primary diagnostic screening test for lead poisoning.[15,16] However, an elevated FEP level in lead poisoning may also be due to associated anemia, iron deficiency, or iron deficiency anemia.

The mechanism of anemia associated with lead poisoning, although not completely elucidated, can be attributed to either inhibition of heme synthesis or shortened life span of red blood cells. Anemia unrelated to iron deficiency or inherited hemoglobinopathies occurred in 6% of this study population. Within this group, the possibility of patients with thalassemia traits cannot be ruled out. However, Koenig et al[4,5] demonstrated that FEP levels can be used to differentiate iron deficiency anemia from alpha or beta thalassemia traits. They found the FEP levels in iron deficiency anemia to be greater than 185 mg/100 mL red blood cells, as compared to less than 107 mg/100 mL red blood cells in thalassemia traits (normal, 25–100 mg/100 mL red blood cells). In this regard, the authors suggested that FEP may even be a better parameter than the Mentzer index.[5] In a study by Cohen et al,[17] the incidence of anemia was 12%. However, the combination of anemia and microcytosis occurred only in one out of 58 patients (2%), suggesting that microcytic anemia in lead poisoning is rather uncommon. Hence, the probability of patients in this group having thalassemia traits is low.

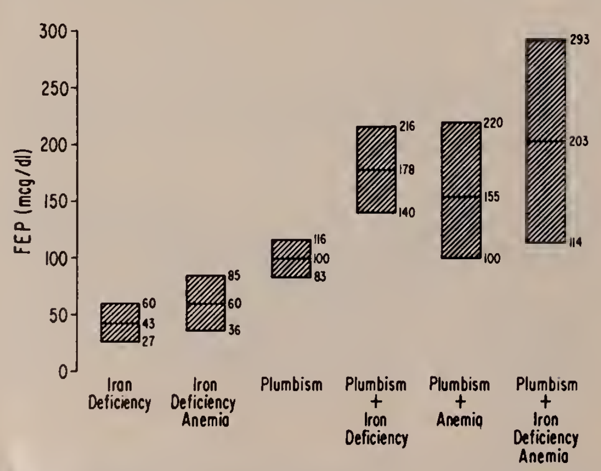


FIGURE 3. Ninety-five percent confidence limits for free erythrocyte protoporphyrin (FEP).

The most specific indicator of lead toxicity is urinary aminolevulinic acid (ALA). However, measurements of urinary ALA in random voidings are unreliable, necessitating 24-hour urine collection. On the other hand, erythrocytic protoporphyrin, another indicator of toxicity, can be estimated easily from blood obtained by venipuncture or fingerstick. Chisolm[18] has demonstrated a statistically significant linear relationship between urinary ALA and FEP. In later studies, although Chisolm was unable to confirm these findings, he attributed the difficulties to modifications in methodology[19] (personal communication, August 1984).

Iron deficiency or iron deficiency anemia, by themselves capable of elevating FEP levels, can cause such levels to become more pronounced in the setting of plumbism; this effect is referred to as background response. In fact, this response was demonstrated by Chisolm,[18] utilizing the hematocrit as an indicator of anemia. The study reported on here, utilizing hemoglobin and transferrin saturation as indicators of anemia and iron deficiency, respectively, clearly demonstrates the magnitude of this background response. This could explain, at least in part, the variation of FEP values observed for a given level of blood lead (Table I). Hannah et al[20] observed a similar phenomenon.

The FEP/Hb ratio, yet another indicator of heme bio-synthesis, has been demonstrated to increase not only with iron deficiency anemia, but also with decreasing transferrin saturation.[21] Data from the control subjects support that conclusion. In addition, these data enable comparisons of the FEP/Hb ratio with anemia, biochemical iron deficiency, and iron deficiency anemia. The pattern of increase in the FEP/Hb ratio closely paralleled that of FEP.

This study has shown that more children with high blood lead levels have biochemical iron deficiency (29%) compared to anemia (15%). Biochemical iron deficiency, even without anemia, raises the FEP and FEP/Hb ratio in children with lead poisoning. This elevation in FEP may erroneously be interpreted as indicative of lead toxicity. Therefore, biochemical iron deficiency in plumbism merits consideration in the proper interpretation of elevated FEP and FEP/Hb ratio in the absence of anemia. Transferrin saturation and/or ferritin level should be determined to help distinguish between the degree of associated biochemical iron deficiency and relative lead toxicity.

Acknowledgment. The authors thank Dr Julian Chisolm Jr for his comments and review of the manuscript.

REFERENCES

1. Watson JR, Decker E, Lichtman HC: Hematologic studies of children with lead poisoning. *Pediatrics* 1958; 21:40-46.
2. Watson CJ: Erythrocyte coproporphyrin; variation in respect to erythrocyte protoporphyrin and reticulocytes in certain anemias. *Arch Int Med* 1950; 86:797-809.
3. Braitman R: Marked elevation of FEP with iron deficiency anemia [letter]. *J Pediatr* 1978; 93:155-156.
4. Koenig HM: Classification of microcytic anemia by flurometric analysis of free erythrocyte porphyrins (FEP). *Ann Clin Res* 1976; 8(suppl 17):151-155.
5. Koenig HM, Lightley A, Schanberger J: The micromeasurement of free erythrocyte protoporphyrin as a means of differentiating alpha thalassemia trait from iron deficiency anemia. *J Pediatr* 1975; 86:539-541.
6. Centers for Disease Control: Surveillance of childhood lead poisoning. *MMWR* 1980; 29:170-171.
7. Cannan RK: Proposal for a certified standard for use in hemoglobinometry. *Amer J Clin Path* 1958; 30:211-215.
8. Mcgovern JJ, Jones AR, Steinberg AG: Hematocrit of capillary blood. *N Engl J Med* 1955; 253:308-312.
9. Fischer DS, Price DC: A simple serum iron method using new sensitive chromogen tripyridyl-s-thiazine. *Clin Chem* 1974; 10:21.
10. Hessel DW: A simple and rapid quantitative determination of lead in blood. *Atoms Abs Newsletter* 1968; 7:55.
11. Piomelli S, Davidow B, Guinee VF, et al: The FEP (free erythrocyte porphyrins) test: A screening micromethod for lead poisoning. *Pediatrics* 1973; 51:254-259.
12. American Academy of Pediatrics Committee on Nutrition: Iron balance and requirement in infancy. *Pediatrics* 1969; 43:134-142.
13. Pearson HA: Iron nutrition revisited—infancy, childhood, adolescence, in Oski FA, Pearson HA (eds): *Report of Eighty-Second Ross Conference on Pediatric Research.* Columbus, Ohio, Ross Laboratories, 1981, pp 8-9.
14. Klecka WR, Nie NH, Hull CH: *Statistical Package for the Social Sciences (SPSS) Primer.* New York, McGraw-Hill Book Co, 1975.
15. Centers for Disease Control: Preventing lead poisoning in young children. *J Pediatr* 1978; 93:709-720.
16. Centers for Disease Control: Increased lead absorption and lead poisoning in young children. *J Pediatr* 1975; 87:824-830.
17. Cohen AR, Trotzky MS, Pincus D: Reassessment of the microcytic anemia of lead poisoning. *Pediatrics* 1981; 67:904-906.
18. Chisolm JJ Jr, Barrett MB, Mellits ED: Dose-effect and dose-response relationships for lead in children. *J Pediatr* 1975; 87:1152-1160.
19. Chisolm JJ Jr, Hastings CW, Cheung DK: Microphoto-fluorometric assay for protoporphyrin in acidified acetone extracts of whole blood. *Biochem Med* 1974; 9:113-135.
20. Hannah TL, Dietzler DN, Smith CH, et al: Erythrocyte porphyrin analysis in the detection of lead poisoning in children: Evaluation of four micromethods. *Clin Chem* 1976; 22:161-168.
21. Piomelli S, Brickman A, Carlos E: Rapid diagnosis of iron deficiency by measurement of free erythrocyte protoporphyrin and hemoglobin: The FEP/ hemoglobin ratio. *Pediatrics* 1976; 57:136-141.

FROM THE LIBRARY

LEAD POISONING ASSOCIATED WITH ASCARIASIS

This incident was reported to the Poison Control Center as a case of lead poisoning. For about a year the mother had observed the two-year-old child eating bits of plaster but was unaware of the harmful effects which it may cause. The child was under supervision in a child health station, and because of a failure to gain weight and a history of pica, the patient was referred to the hospital for further investigation and possible treatment.

On admission to the hospital the only complaint was abdominal pains, and the presenting symptoms were anemia and underweight. The laboratory findings showed the lead blood level to be 0.07 mg. per 100 ml. and the urine lead level to be 0.06 mg. per liter. Spinal fluid and protein determinations were not done. The hemoglobin was 9.4 Gm. per 100 ml., and the red blood cell count was 3,300,000. After two weeks of hospitalization and therapy, the child was discharged.

This case was complicated by the finding in the stools of a parasitic infestation (ascariasis). In addition to ascariasis, a diagnosis of lead intoxication was made. Following her discharge from the hospital, the child was treated in the outpatient department with piperazine hydrochloride for the parasitic infestation and with iron therapy for the anemia.

Lead poisoning and parasitic infestation are not infrequent in Puerto Rican children, and their coexistence is not uncommon. A confirmed diagnosis of lead poisoning, particularly in a Puerto Rican child, should not exclude a stool examination for the determination of another condition which also may contribute to the anemia. . . .

(*NY State J Med* 1962; 62:2551-2553)

The role of physicians in the life insurance industry in the 19th century

JOHN S. HALLER, JR

Although life insurance in its most primitive forms can be traced to the ancient mutual and fraternal orders of the Greeks and Romans, its establishment on a more rational and scientific basis occurred in England during the reign of Queen Anne, where so-called "friendly" or "benevolent" societies paid out small sums for burial expenses and provided annuities to widows of clergymen. Many of these societies failed due to their lack of sound business principles, and the fact that they assessed premiums without a careful analysis of the risk. As long as societies accepted applicants on the basis of charity or fraternity and without a medical examination, their very survival was in jeopardy. Attributes such as good fellowship, mutual aid, and sociability had their place, but not in business.[1]

Later in the 18th century, applicants for life insurance were required to sign affidavits of good health before a policy could be issued. Later, applicants were required to appear before a board of directors who, as laymen, determined by inspection the value and classification of the risk, and set the amount to insure. Eventually associations recognized that their prosperity depended upon a more scientific understanding of life and health and called upon physician examiners to serve as their gatekeepers. However, the earliest medical examinations were superficial at best.[2] In 1770, the health certificate for life insurance simply required the signature of two individuals, one of whom was a physician. The directors of the company then conducted a secret vote on whether the applicant should be accepted or rejected. Although the first physician examiner was appointed in 1779, they did not become a regular part of the insurance industry until 1820. One of England's oldest insurance companies, founded in 1706, did not retain a physician examiner until 1855.[3,4]

THE MEDICAL EXAMINER

Obviously a business as vast as life insurance required substantial administration. "Like the temple of Gaza," noted C.C. Bombaugh, MD, at the 11th annual meeting of the American Academy of Medicine at Washington in 1887, "the structure of life insurance has two chief supporting pillars. The one is the mathematical, the other is the medical. The one we leave to the actuary; be it our care to see that no mischievous or antagonistic forces are applied to the other."[5] In the financial risk-taking of these institutions, the role of the physician examiner became critical to each company's program. Like a bad loan for a bank, bad risks for an insurance company weakened it; too many broke it. As one physician noted,

> A company's natural constitution is represented by its assets, its power of endurance by its surplus, its liability to premature death by the character of its risks; hence a company without assets has no natural constitution, one without surplus no powers of endurance, one loaded down with bad risks is ready to die at any time.[6]

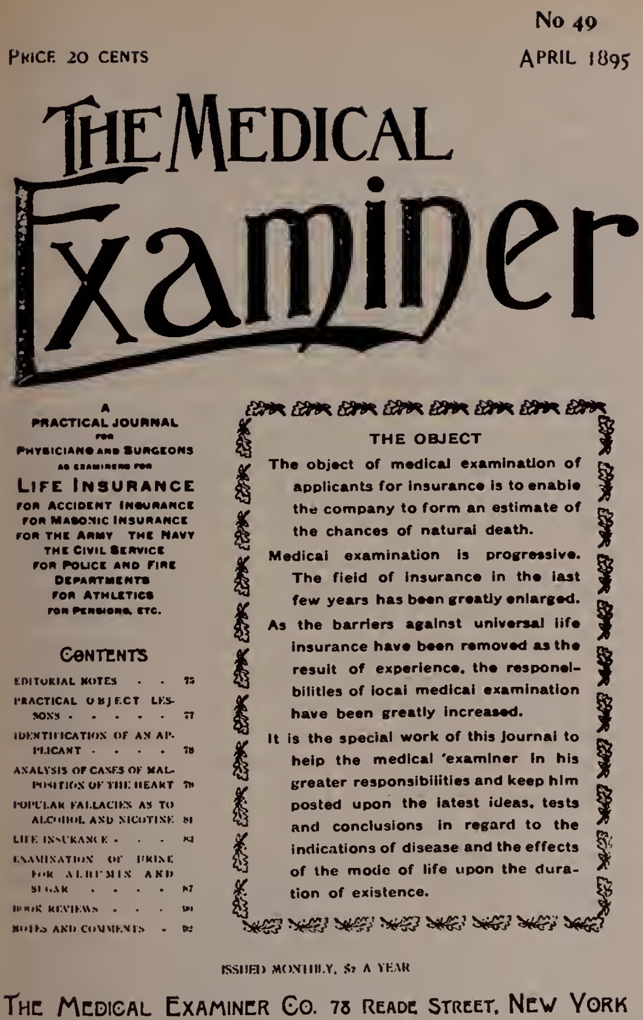
The physician examiner stood to the insurance company as the legal examiner did to a court; decisions by both the company and the court required the skillful assessment of facts. "No medical referee having a due regard for his own or the reputation of his profession," wrote J.B. Temple, president of the Southern Mutual Life Insurance Company of Kentucky, "will fail to remember that confidence of interested parties—not the company only, but the future orphans and widows whose trustee it is—has been reposed in him. He is not the employee of an agent or an applicant who may chance to call on him, but of a society wielding important and delicate trusts."[7]

From the point of view of the medical profession, the physician examiner was the most important officer on the roster of a life insurance company. Without his ability and integrity a company opened itself to financial disaster. For this reason, companies expected certain qualities from the physician examiner, including being a "believer in life insurance, not only theoretically but practically."[8] Unless an examiner accepted the utility and benefits of insurance, he failed in his responsibilities to both the insured and the company.[8,9]

Characteristics of the ideal physician examiner, as explained in numerous 19th century publications, included mental adaptability, good judgment, accurate powers of discrimination, and a keen eye trained to discover the laws of character and heredity. Although the final decision whether to accept or reject an applicant would lie with the company itself, the data obtained from the medical examination gave the only clear basis for the decision. For this reason, remarked William Detmold, MD, in the *Medical Record* of 1877, "strict honesty. . . is much more im-

---

Address correspondence to Mr Haller, Professor of History, University of Colorado at Denver, Denver, CO 80202.



No 49
PRICE 20 CENTS
APRIL 1895

THE MEDICAL
Examiner

A
PRACTICAL JOURNAL
FOR
PHYSICIANS AND SURGEONS
AS EXAMINERS FOR
LIFE INSURANCE
FOR ACCIDENT INSURANCE
FOR MASONIC INSURANCE
FOR THE ARMY THE NAVY
THE CIVIL SERVICE
FOR POLICE AND FIRE
DEPARTMENTS
FOR ATHLETICS
FOR PENSIONS, ETC.

CONTENTS

EDITORIAL NOTES . . . 75
PRACTICAL OBJECT LESSONS 77
IDENTIFICATION OF AN APPLICANT 78
ANALYSIS OF CASES OF MALPOSITION OF THE HEART 79
POPULAR FALLACIES AS TO ALCOHOL AND NICOTINE 81
LIFE INSURANCE 83
EXAMINATION OF URINE FOR ALBUMIN AND SUGAR 87
BOOK REVIEWS 91
NOTES AND COMMENTS . 92

THE OBJECT

The object of medical examination of applicants for insurance is to enable the company to form an estimate of the chances of natural death.

Medical examination is progressive. The field of insurance in the last few years has been greatly enlarged.

As the barriers against universal life insurance have been removed as the result of experience, the responsibilities of local medical examination have been greatly increased.

It is the special work of this Journal to help the medical examiner in his greater responsibilities and keep him posted upon the latest ideas, tests and conclusions in regard to the indications of disease and the effects of the mode of life upon the duration of existence.

ISSUED MONTHLY, $2 A YEAR

THE MEDICAL EXAMINER CO. 78 READE STREET, NEW YORK

FIGURE 1. Cover of an early journal addressing the issue of medical examination of applicants for insurance.

portant than scientific assumptions."[10,11]

Other characteristics as well were expected of the physician examiner. While he may have had a preference for certain companies or insurance plans, he could not discuss these with the applicant; nor could he work for a company he could not endorse as sound and legitimate. "A physician should no more lend the weight of his professional . . . standing as a citizen to the assistance of a quack life insurance company," wrote a physician in the *Indiana Medical Journal* in 1896, "than he would give certificates to quack remedies."[12] If he examined for more than one company he should avoid betraying confidences involving applicants or actions of the company. He could not reveal pending transactions and, acting in his capacity as examiner, should disregard any relationship to the individual as personal physician. Thus personal knowledge of a disease which the applicant preferred to remain secret could not be kept confidential. Company loyalty, avoiding personal relationships with applicants, maintaining precision in information, attending promptly to all appointments, showing discretion and industriousness, and never asking for business were other qualities noted as essential for every examiner.[13,14]

Physician examiners preferred to be responsible solely to the Medical Department of the company and believed that their reports should be private, with neither the agent nor the applicant having access to them. The final determination of acceptance or rejection would lie with the Medical Department, acting on the report of the examiner. However, this was not always the case. Often, physician examiners and agents divided their commissions, which, practically speaking, amounted to offering a bribe to the examiner for passing a risk. Cases where local agents and physician examiners pooled their commissions and examination fees resulted in devastating statistics that usually sent "cold chills over the home office."[8,15]

Agents, who usually appointed their own physician examiners, sometimes abused their power of appointment. By allowing themselves to be motivated by the number of policies written rather than the potential "risk" of a policy holder, they encouraged physician examiners who were less cautious for fear of losing a client. Clearly the unscrupulous agent could write policies only with the support of an unscrupulous doctor—or an incompetent one. Such results, noted the editor of the *Western Journal of Medicine*,[16] in 1868, were the inevitable product of a system which erected a "beggarly standard of professional fees." The outcome was a "system of cheap physicians." When this occurred, the editor went on to say, "the aggregate worthlessness of appointments, begotten of the niggardly principle on which services are judged of, and their price adjusted, must fearfully increase the natural liability of losses."[16] This wasteful situation, when compounded by similar wrong judgments—by both the agent in seeking the client and the physician in approving the policy—brought into question the savings to the company and the price of insurance.[16]

Articles in medical journals cautioned physicians to be on their guard with agents or solicitors whose personal interest was to have the physician examiner "pass" every potential client for life insurance, because some agents knowingly wrote life policies on unsound applicants, thinking only of the fee or commission attached to the policy. To protect themselves from pressure, doctors were urged never to allow the agent to be present at an examination.[17]

In 1874, a number of American insurance companies organized themselves into an association known as the Chamber of Life Insurance. Included in its mission of promoting life insurance interests was the need to maintain a list of accepted physician examiners. This suggested, by implication, that the examiner functioned as an expert witness for the company in the selection of lives and that he could be trusted to treat all individuals impartially. Since considerable patronage followed these appointments, the association expected a higher level of responsibility from its member physicians. Advocates of this change believed that while no physician was expected to make a specialty of examinations, he should be thoroughly qualified and knowledgeable on such matters as longevity, heredity, occupation, and habits—areas directly bearing upon insurance matters.[18]

By the late 1880s, the home offices had begun to select local examiners. Physician examiners thus became independent of agents, generally better paid for their services,

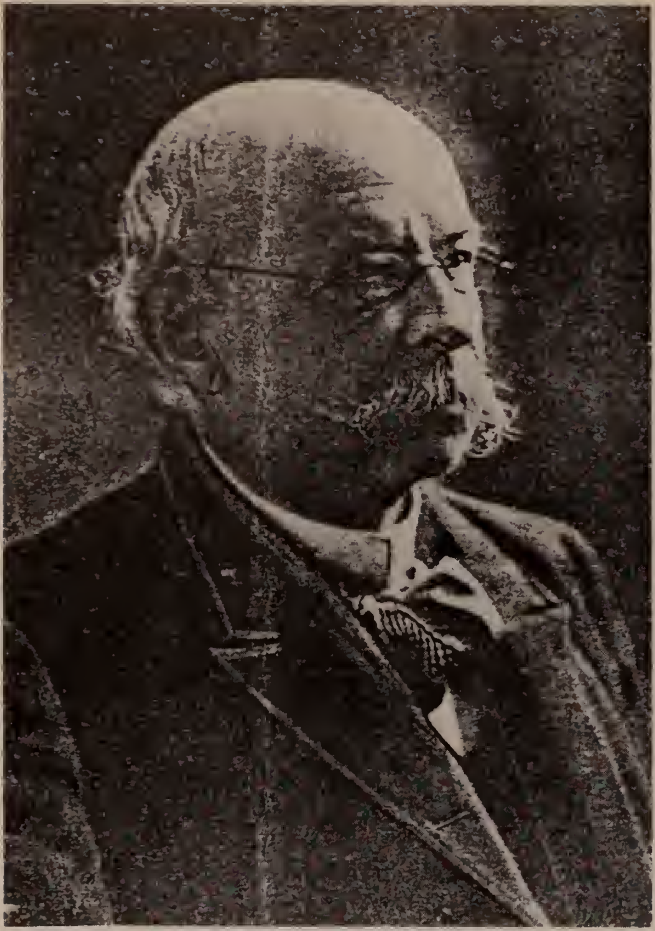


FIGURE 2. Francis Peyre Porcher, MD, President of the Medical Society of South Carolina in 1895, and an examiner and referee for the Mutual Life and the Equitable of New York insurance companies.

and more aware of their responsibilities. The physician had the legal duty to ascertain and report to the company on the health or physical condition of the applicant and whether he suffered any life-shortening disease or defect. Only the physician who acted "without fear or favor" was qualified to fill this role.[6]

Since most insurance companies did not recognize homeopaths as examiners, homeopathic physicians preferred to work for fraternal societies rather than accept what they considered the discriminatory attitude of insurance companies. In 1890, the Aetna Life Insurance Company of Hartford had 56 physician examiners in 20 cities, none of whom were homeopaths. The same held true of New York Life. Northwestern Mutual Life Insurance of Milwaukee had two homeopaths out of a total of 43 examiners, Berkshire Life Insurance Company had nine homeopaths out of 558 examiners, Equitable of New York had one out of 132 examiners, and Metropolitan Life Insurance had one out of 49 examiners in eight cities. Similar statistics existed for Travelers of Hartford and John Hancock Mutual Life Insurance Company of Boston, which had no homeopaths affiliated as examiners.[19]

MEDICAL EXAMINATION

American life insurance companies tended to use application forms that were generally similar throughout the industry. The application form requested the amount, character, and condition of policy desired by the client; the family or hereditary history of the applicant; the individual history of the applicant including past occupations, residence, diseases, and habits; and finally, an assessment of the present condition of the applicant. The key elements of any application included information on the age, habits, and personal and family history. Of these, age was the only provable element; the rest depended upon the honesty of the applicant and the ability of the physician examiner to discern health and family history.[17,20]

By the 1860s and 1870s, insurance companies required applicants to answer many detailed questions on their health and family history, and insisted upon the absolute truthfulness of each statement. The law as interpreted by the courts rendered a policy void if the applicant answered the questions dishonestly. This gave companies discretionary powers that allowed them to take advantage of mistakes made by the applicant, even those that were unintended and immaterial to the risk. Eventually, legislation forbade companies from voiding contracts on the basis of these mistakes unless they were material to the risk, willfully made, and "life terminated because of something of which a truthful answer would have given the company a warning note."[12] By the turn of the century, companies voluntarily adopted policies which made applications indisputable after three years regardless of misstatement. Because of the competitive nature of insurance companies in the United States, many eventually chose to reduce the limitation to one year. In effect, no misstatement in the application voided a policy after it had been in force for one year.[12]

Physician examiners faced a more difficult task in examining for life insurance than dealing with a typical office visit. The reasons were obvious. Patients calling upon physicians to find a cure for a particular ailment were anxious to acknowledge every pain and symptom of disease; on the other hand, those wanting life insurance seldom communicated the cause or history of any malady. Instead, they preferred to withhold all information and respond as indirectly as possible to questions that delved into the risk side of insurance. Many applicants feared that an examination would disclose some poor habit, bad health situation, disease, or hereditary trait that might void the application.[21]

Articles and handbooks written for the physician examiner urged caution in guarding against applicants who attempted to conceal defects of personal health or family history. Seldom did the physician know the patient before the examination; most often, the physician had to become an instant expert on the personal and family history of the applicant.[22-26] Through obtaining the family history, the physician examiner could learn facts concerning the family longevity, including any hereditary tendency to diseases such as consumption, insanity, and epilepsy. If the family history suggested anything that might make the applicant objectionable, the nature of the individual history became all the more important.

Of principal concern among the supposed hereditary diseases was consumption. Doctors were cautioned to watch for histories that included deaths from exposure to cold, shipwreck, journeys across the plains, or child-bed. Too often, these were thinly disguised examples of deaths from hereditary consumption. Although medical science gave "no rules or ratio of the frequency of this inheritance," doctors generally agreed that the disease occurred through a "hereditary taint" as well as by independent acquisition.[17] Increasingly, however, Felix von Niemeyer's (1820–1871) *Clinical Lectures on Pulmonary Consumption* (1870) inclined the medical world toward the idea of independent acquisition, with consumption originating in pneumonia, bronchitis, pulmonary congestion, and pulmonary hemorrhage rather than in a tubercular diathesis. Nevertheless, many physicians recommended rejecting an applicant if both parents had died of consumption or when even one parent had died of the disease "unless it can be clearly shown that the death was from an acquired consumption, dating years after the birth of the applicant, and that the latter shall have passed the age of forty-five in perfect health."[27] The same held true if two or more brothers or sisters had died of consumption.

Insanity was another supposed hereditary disease which physician examiners considered important in applicants; others included cancer and gout, and "hereditary intemperance." Physicians cautioned against approving applicants whose family was exposed to drunkenness and even teetotaling ("for in a large proportion of cases the teetotaler is a reformed drunkard"). The same held true for tobacco addiction which included symptoms of heart palpitation, irregularity of pulse, dyspepsia, emaciation, and general debility.[10]

FIGURE 3. Thomas Chappell Cook, AM, MD, medical examiner for the Manhattan Life Insurance Company and the Mutual Life Insurance Company, both of New York.

Insurance companies also relied to a certain degree on evidence of classical constitutional pathology.

> If the swarthy are bilious, the lymphatic indolent and intemperate; if short necks, florid faces and heavy frames are correlated to gouty change with fragile blood vessels disposing to apoplexy; if blue eyes, golden hair and velvety skins are indexes to a strenuous diathesis favoring consumption, glandular swellings, spinal caries and lumbar abscesses; if slender bones, taut muscles, compact abdomens and large fore brains mark the nervous temperament—logical subjects for neurotic maladies and drug habits; if these physical distinctions are imparted to offspring, who subsequently develop the pathological states foreshadowed by definite physical qualities, then is hereditary transmission of disease not to be gainsaid; then are tendency and heredity convertible terms.[28]

In the individual history of each patient, physician examiners sought to verify those significant diseases which had afflicted the patient, as well as his physical development and ability to resist disease. The applicant's actual condition proved equally important. This included proportion of height to weight, excessive obesity or emaciation, figure or form of the applicant (round shouldered, flat chested, etc), countenance or general expression, and appearance. Other factors included the physical examination of the applicant, in which the physician distinguished between normal and abnormal sounds elicited by auscultation and percussion, and inspected the thoracic and abdominal viscera, noting the existence or absence of hernias, varicose veins, tumors, ulcers, cicatrices, and glandular enlargements.

Obviously, occupation influenced both the health and longevity of the client. Applicants whose work environment contained dust were susceptible to lung problems. Thus, bakers, upholsterers, ax-grinders, and stone and marble cutters were always considered poor risks. The same was true of those who were exposed to intense heat or sudden cold. Members of the learned professions, on the other hand, had the greatest longevity (Table I).[10]

No longer was it sufficient for examiners simply to thump the chest, poke the belly, or guess at the pulse rate. Hasty examinations that made no note of heredity, occupation, habits, tendencies, and family history, and that made no effort to test the urine or blood, were useless. By the mid-1880s, even beneficiary associations were beginning to require examinations as well as tests of the urine, lungs, and heart as part of the application for membership.[29,30]

Although American physician examiners differed among themselves in the scope of their inquiries and the forms used in their examinations, the differences among countries were even greater. At the Second International Congress of Insurance Physicians in Amsterdam in 1902, a universal medical form was introduced. The commission had spent two years preparing the form but achieved no consensus among the member physicians. American physicians objected to the fact that the form lacked questions on the cause of death or state of health of grandparents. Moreover, the form contained 63 questions, exclusive of family history, and an appendix with 100 additional questions. American physicians preferred a form with a maxi-

TABLE I. Professions Ranked from Most Dangerous to Most Favorable*

Class I	Class II	Class III	Class IV
Brakeman on freight trains	Bridge builder, boatman	Barkeeper	Apothecary, architect
Buzz sawyer, circular sawyer	Barber on steamboat	Blacksmith (working)	Artist, painter, author, writer
Powder maker, seaman	Brakeman on mail train	Bricklayer	Attorney, lawyer
	Clerk on river steamboat	Captain on river steamer	Bank officer or clerk
	Car coupler	Car driver	Baker, barber
	Conductor on freight trains	Carman (drayman)	Boot and shoe maker
	Cooper	Carpenter and joiner	Builder (not laborer)
	Engineer on river steamer	Chief engineer	Civil engineer
	Fireman (locomotive)	Conductor on passenger trains	Clergyman (minister)
	Horse shoer	Distiller	Clerk (generally)
	Laborer (wharf, warehouse, elevator)	Foundry & furnace employee	Chemist, manufacturing
	Miner (underground)	Hod carrier	Dentist, dressmaker
	Sailor	Laborer, common	Editor, farmer
		Mason	Marble cutter
		Miner (surface)	Photographer
		Puddler	Physician, postmaster
		Stone cutter and dresser	Painter (house, ornamental)
		Teamster	Plumber, printer
		Wood chopper	Tanner, tailor
			Teacher, tool maker
			Vitriol manufacturer
			Wheelwright
			Watchman

* Abstracted from work cited in reference 17.

mum of 40 to 50 questions and which placed more emphasis on the actual physical examination. They also objected to the lack of questions on body temperature, condition of knee jerk reflexes, pupillary reflexes, and a determination of the applicant's environment.[31]

Dr Price, who was known as the "Father of correct life insurance," warned insurance companies of the dangers of inaccurate or uninformed medical examinations. "Those persons will be most for flying to these establishments," he remarked, "who have feeble constitutions or are subject to distempers which they know render their lives particularly precarious." The disposition to pass "bad lives" onto the lists of an insurance company offered short term profit to the agent but a substantial and long term loss for the company. Admittedly, agents lacked the skill to detect latent disease which the educated physician was presumed to possess; only when both agent and physician exercised good moral judgment was it reasonable to conclude that the decision to insure was both reasonable and fair.[7]

Information sharing among companies was common, especially on undesirable risks. Companies found it mutually convenient to share among themselves "interchange rejections." Thus, when a company declined a particular risk, it sent a notice of the decline to other companies in the interchange. Eventually, companies found it easier to send these notices to a distribution center. This plan continued in use into the 1890s.[32]

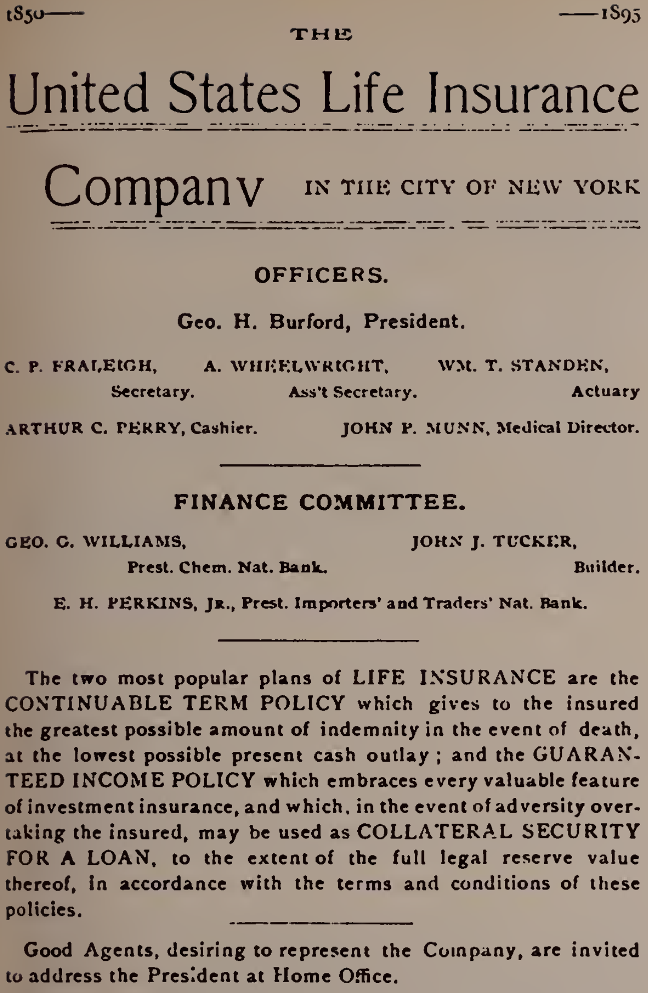
FEES

"Of physician examiners," commented the editor of the *Western Journal of Medicine* in 1868, "none will deny that many are ignorant, some are ductile, and that a few are probably corrupt."[16] Life insurance companies were to blame for this situation, argued the editor, because physicians were paid so poorly in spite of the importance placed on their examination. No lawyer, for the same pitiful sum of money, would hold a consultation to weigh circumstances, examine facts, and furnish a sound legal opinion. While the lawyer received $20 for such a function, the physician was grudgingly paid only $2. Were companies to take more seriously the services performed by the examiner, they would avoid the "broken-down, diseased, and dying incumbrances" which drew upon the resources of the companies.[16]

Because of low fees, some physicians refused outright to serve as examiners. S.H. Stout, MD, of Dallas, Texas, was so opposed to examiners' low salaries that he urged his fellow physicians to refuse to work for these "great moneyed corporations."[33] For too long, physicians had willingly accepted poor compensation on the mistaken belief that the companies were "purely benevolent institutions." If physicians would "study the subject of life insurance, . . . refuse to be no longer sponged upon by them or their agents, and [refuse] to lend medical skill for an inadequate compensation," then there would be reason to cooperate with them.[33]

RETROSPECT

Through most of the 19th century, medical schools made little mention in their curricula of the life insurance industry and the role of the medical profession in it. Medical journals across the nation, however, discussed it at length, and journals such as *The Medical Examiner*, New York (1891–1909) and *The Medical Examiner; A Semi-Monthly Journal of Medical Sciences*, Chicago (1860–1875), were founded expressly for the purpose of communicating to physicians the needs and purposes of the insurance industry as it applied to the profession. One of the earliest physicians to actually teach the subject of the physician's role in life insurance was J.T. Craig, MD, who lec-



1850— —1895

THE

United States Life Insurance Company IN THE CITY OF NEW YORK

OFFICERS.

Geo. H. Burford, President.

C. P. FRALEIGH, Secretary. A. WHEELWRIGHT, Ass't Secretary. WM. T. STANDEN, Actuary

ARTHUR C. PERRY, Cashier. JOHN P. MUNN, Medical Director.

FINANCE COMMITTEE.

GEO. G. WILLIAMS, Prest. Chem. Nat. Bank. JOHN J. TUCKER, Builder.

E. H. PERKINS, Jr., Prest. Importers' and Traders' Nat. Bank.

The two most popular plans of LIFE INSURANCE are the CONTINUABLE TERM POLICY which gives to the insured the greatest possible amount of indemnity in the event of death, at the lowest possible present cash outlay; and the GUARANTEED INCOME POLICY which embraces every valuable feature of investment insurance, and which, in the event of adversity overtaking the insured, may be used as COLLATERAL SECURITY FOR A LOAN, to the extent of the full legal reserve value thereof, in accordance with the terms and conditions of these policies.

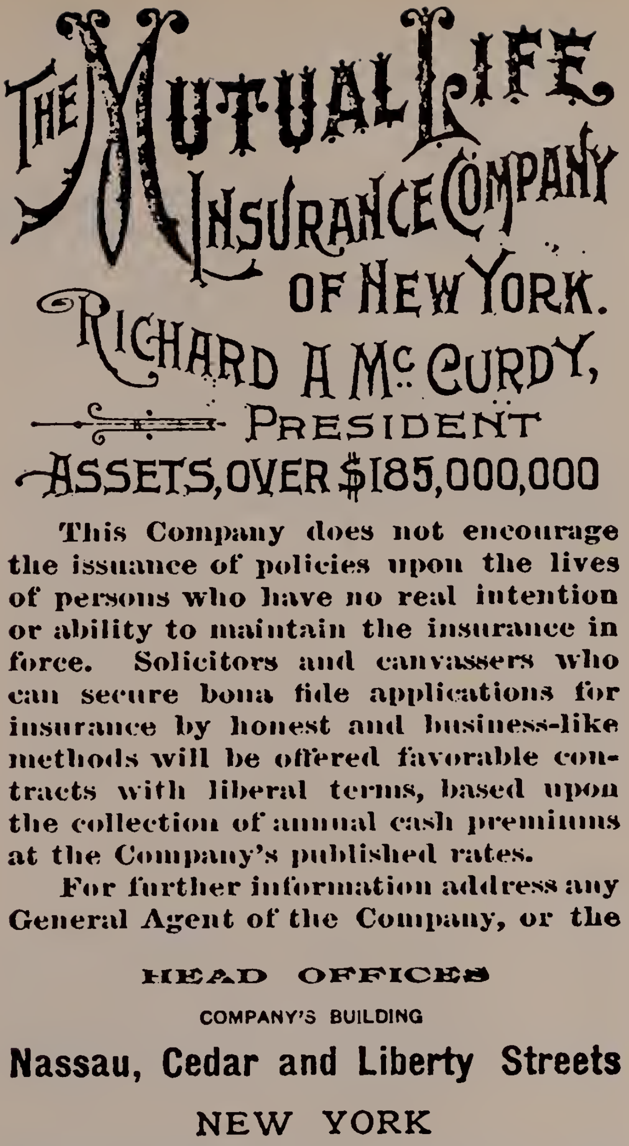
Good Agents, desiring to represent the Company, are invited to address the President at Home Office.

FIGURE 4. Advertisement in the April 1895 issue of *The Medical Examiner* soliciting life insurance agents for the United States Life Insurance Company.

tured to the class of 1898 of the College of Physicians and Surgeons in the Medical Department of Kansas City University. Craig believed it of sufficient importance to be part of the didactic education of every medical student. "I have the proud distinction," he noted, "of being the first lecturer to deliver a regular course on this all-important subject in this city or elsewhere in the West."[3]

Ultimately, medical examiners hoped that life insurance would carry the same level of esteem with the public as fire insurance. To achieve this end, they encouraged companies to investigate the hygiene of cities, make further inquiries into the hereditary tendencies and personal habits of the insured, and look more closely at appropriate rules of health.[29] Their recommendations carried with them the desire that medical examiners would receive respectability and remuneration equivalent to the seriousness of their decisions.

Although physician examiners oftentimes lamented their depressed socioeconomic status,[34] ironically, the meager payment given to them for performing insurance examinations was both a symptom of their economic plight as well as a safeguard against the competition caused by the fact that there were too many physicians and too few patients. The examination fee provided a modicum of financial security, though seldom did it reach a level of compensation sufficient to provide the physician examiner with an independent income base. But for the young practitioner seeking to establish a practice, or the retired or semiretired physician attempting to supplement an income, the fee from medical examinations offered a margin of financial security and served, in some measure, to reduce the need for physicians to cut their rates in an effort to compete successfully with more established physicians.



THE MUTUAL LIFE INSURANCE COMPANY OF NEW YORK.

RICHARD A. McCURDY, PRESIDENT

ASSETS, OVER $185,000,000

This Company does not encourage the issuance of policies upon the lives of persons who have no real intention or ability to maintain the insurance in force. Solicitors and canvassers who can secure bona fide applications for insurance by honest and business-like methods will be offered favorable contracts with liberal terms, based upon the collection of annual cash premiums at the Company's published rates.

For further information address any General Agent of the Company, or the

HEAD OFFICES

COMPANY'S BUILDING

Nassau, Cedar and Liberty Streets

NEW YORK

FIGURE 5. Mutual Life Insurance Company advertisement for "solicitors and canvassers" (published in the April 1895 issue of *The Medical Examiner*).

References

1. Brown GL: Duties of physicians as examiners for life insurance. *Buf Med Surg J* 1891-92; 31:69-74.
2. Brower DR: Some observations on medical examinations. *Med Exam* 1903; 13:83-84.
3. Craig JT: Lecture on medico-insurance. *Med Exam* 1899; 9:15-18.
4. VanMeter LM: Medical examination for life assurance. *Memphis Med Mon* 1902; 12:355-361.
5. Bombaugh CC: The later phases of medical examination for life insurance. *Chicago Med J Exam* 1887; 55:189-194.
6. Foster TA: The relation of life insurance to the medical profession, with

hints to medical examiners. *Trans Maine Med Assoc* 1886-88; 35/36:303-314.

7. Temple JB: Relations of the medical profession to life insurance. *Louisville Med News* 1876; 1:111-113, 269-271.
8. Danforth IN: Life insurance and the medical profession. *Chicago Med J Exam* 1884; 49:349-359.
9. On the relation of the medical examiner to the business of life insurance. *Med Rec* 1873; 8:245-248.
10. Detmold W: Examination for life insurance. *Med Rec* 1877; 12:81-84, 97-98, 113-115.
11. Morris M: Medical supervision in distinction from medical selection in life insurance. *Med Rec* 1875; 10:51-55.
12. Finch JA: Medical examinations for life insurance. *Indiana Med J* 1896; 15:447-450.
13. Nicholson WA: Life insurance examinations. *Med Exam* 1901; 11:12-15.
14. Grant WE: Lecture on medical life insurance. *Med Exam* 1898; 8:275-277.
15. Sholl EH: The practical relation of the physician to life insurance. *Trans Med Assoc State of Alabama* 1890, 276-285.
16. Editorial. *West J Med* 1868; 3:189-193.
17. Steedman JGW: The duties of physicians to life insurance companies and their policy-holders. *St Louis Med Surg J* 1871; 8:204-218.
18. Medical experts in the selection of lives. *Med Rec* 1874; 9:222-223.
19. Burlingame FW: The relation of homeopathy to life insurance. *Trans Homeop Med Soc State of Penn* 1900; 188-194.
20. Sieveking EH: The medico-legal aspects of life insurance. *Med Rec* 1874; 9:218-222.
21. Dodge CL: Some points in the examination of persons for life insurance. *New York Med J* 1890; 52:206-207.
22. Skinner CE: Medical examination for life insurance in the field. *New York Med J* 1898; 58:234-267.
23. Sieveking EH: *Medical Advisor in Life Insurance.* Hartford, Conn, NP Fletcher, 1886.
24. Keating JM: *How to Examine for Life Insurance.* Philadelphia, WB Saunders Co, 1890.
25. Allen JA: *Medical Examinations for Life Insurance.* New York, Spectator Co, 1880.
26. Brinton W: *On the Medical Selection of Lives for Assurance.* New York, The Johns Hopkins Press, 1863.
27. VonNiemeyer F: *Clinical Lectures on Pulmonary Consumption.* London, New Sydenham Society, 1870.
28. Smith FA: Heredity and its value as a factor in life insurance. *Med Exam* 1891; 11:463-466.
29. Himes IN: Remarks and cases connected with medical examinations for life insurance. *Trans Ohio State Med Soc* 1885; 93-101.
30. Cluness WR: The medical examiner's duties in connection with life insurance. *Pacific Med Surg J* 1876; 18:412-420.
31. Green CL: Modern life insurance—Does its medical selection reflect the scientific advance of the past century. *Med Exam* 1902; 12:87-93.
32. Albright CE: The medical man in life insurance. *Milwaukee Med J* 1893; 1:352-357.
33. Stout SH: Ethical relationships of the medical profession and life insurance companies. *Texas Med J* 1894; 9:493-497.
34. Haller JS: Business and ethics of medicine, in *American Medicine in Transition, 1840-1910.* Urbana, University of Illinois Press, 1981, pp 234-279.

FROM THE LIBRARY

THE MEDICAL EXAMINER FOR LIFE INSURANCE AND HIS RESPONSIBILITIES

. . . The medical examiner should have constantly in mind two important differences in mental attitude between the patient and the applicant for insurance. In the first place, many, especially at their first examination or among the younger applicants, are extremely nervous; the idea that the examination may reveal some hidden ailment so takes possession of their minds as to disturb considerably the nervous equilibrium—to induce, as it were, a mild degree of shock. In such cases the pulse may be found extremely rapid or intermittent; there is apt to be pallor or muscular tremor. The picture is one of nervous debility or of a want of normal bodily vigor. Whenever an examiner meets with this condition he should be able by tactful handling to reassure the applicant and to re-establish the normal nervous balance; at any rate, he should make due allowance in his report for the disturbed mental state of his subject.

As to the other differences to which we have referred, when he consults his physician the patient endeavors to describe every detail of the disease of which he complains. He conceals nothing. His mental attitude is one of unreserved co-operation. When he is a candidate for life insurance, the case is very different; his memory for details is less acute; his state of mind is one of antagonism. He believes himself to be a good risk, and his bias of mind in that direction is so strong as—no doubt, unconsciously—to color his entire history. On this account a medical history for life insurance is a very different matter from that which is obtained from a patient. The patient assists his physician—the applicant for insurance does not assist the examiner. It requires time for any physician to adjust himself to this difference in mental attitude. The skilled medical examiner has learned this lesson.

It is on this account particularly that the examiner for life insurance must bring to his task complete independence of character, absolute integrity, and the ability to detect attempts at fraud. He must constantly bear in mind that he is a guardian of the interests of the company, and that upon his vigilance, judgment, and integrity depends, largely, its success or failure. . . .

S. OAKLEY VANDER POEL, MD
(*Trans Med Soc St New York* 1900; 288-294)

The physician-patient relationship in interesting times

JOSEPH A. STANKAITIS, MD, MPH

There is an old Chinese curse that states, "May you live in interesting times." One could surmise that "interesting times" represents an era of great turbulence and flux, when events occur at a rapid pace and when familiar institutions that customarily grant stability and comfort seem to be disappearing. It would seem that the physician-patient relationship is the recipient of such a curse as it now finds itself in the midst of tremendous change and instability, creating great distress for both the physician and the patient. Indeed, things are no longer what they used to be.

Economic factors and the availability of resources influence this relationship in a major way. In years past, this was felt to be a land of opportunity, multitudinous possibilities, and unlimited resources. Now, however, there is a realization that economic resources are limited, and that some potentially painful choices will need to be made. Medical care costs are now in excess of 10% of the gross national product. The United States faces budget deficits of trillions of dollars. The federal government has understandably entered a cost-cutting phase, and health care expenditures, unlike those for defense, have and will continue to face reductions. Large corporations, which for years have allowed their health care costs to rise with apparent indifference, have suddenly awakened to this situation and realized that if they are to compete successfully with foreign corporations whose employee costs are lower, they too must pare down their health care expenses. Thus, previously "generous" or indifferent payers for health care in the United States now insist on stringent cost containment measures, and because they pay a significant portion of these expenses, they are in many ways influencing the physician-patient interaction.

Other factors contribute to this turbulence. The technology of medicine today far surpasses even the dreams of practitioners 50 years ago. The development of antibiotics, intensive care units (ICU) with respirators and vasopressors, computed tomography (CT), magnetic resonance imaging (MRI), cardiac surgery, transplant surgery, and dialysis, to list a few, has created multitudinous diagnostic and treatment options unavailable to physicians of days gone by. Many of these modalities we take for granted today; however, they raise many ethical and cost/benefit issues that previously did not have to be addressed.

Consumerism has also entered the physician-patient relationship. Whereas in the past the physician was given the upper hand in the relationship by virtue of his professional authority, this has generally been supplanted by more of a contractual relationship between equals. Most people feel that this change is highly appropriate. What this change has also produced, however, is the increased probability of conflict between these parties, especially when there is a greater realization of how inexact the practice of medicine can be.

An outgrowth of some of these events has been the ongoing malpractice crisis. Previously trusting partners in the physician-patient relationship have become adversaries. The expectations of successful treatment that modern-day medicine holds out to its patients are mitigated by the hard realities of human imperfection and inexactitude, leading the disappointed patient to seek solace in financial rewards offered by an ever-willing tort system.

Physicians also have not been immune to the influences of financial inducements. Daily, via journal advertisements, mailings, and salespersons, physicians are bombarded with information and enticements on how to increase their income by the purchase and use of various gadgets and procedures. Given the increasing expenses of medical practice, coupled with ever-decreasing revenues, such temptations are difficult to ignore.

THE PHYSICIAN-PATIENT RELATIONSHIP

In ancient Egyptian and Mesopotamian societies there was a division between the supernatural and natural treatment of illness. One sought help from a priest for the former, and from a medical "craftsman" for the latter. At least initially, the craftsmen had a much lower social status than their priest counterparts. However, with time and increased success, they acquired more respect from their patients. It was during this time that sanctions and rules to control the activities of physicians and surgeons were first issued.[1]

The Greeks were able to absorb much from these cultures, primarily via trade, and began to develop their own medical system. They developed the medical craft into a science. With the study of philosophy, Greek physicians added theoretical and philosophical knowledge to their

---

From the Department of Medicine, State University of New York at Buffalo School of Medicine, and Health Care Plan, Buffalo, NY.

Address correspondence to Dr Stankaitis, Health Care Plan, 2075 Sheridan Dr, Buffalo, NY 14223.

technical skills. The Greek study of order and philosophy naturally led to the development of codes of ethics and behavior with regard to the practice of medicine. These were traditionally attributed to Hippocrates (ca 460-377 BC), though it is unclear whether he actually authored such codes and an oath. Nevertheless, the various codes that were compiled during that time implied that the physician's responsibility to the individual patient was of paramount importance.[2]

The importance of this relationship has continued through the ages. Jewish law views the physician-patient relationship as a sacred one involving the patient, the physician, and God. It is the physician's responsibility to treat the patient, and the patient's to obtain the best medical care possible, all in order to "collaborate with God in the process of continuing creation and to mend the imperfections in the world and in men."[3] With the advent of Christianity, the treatment of ill individuals was motivated by compassion for the needy individual and by love of God.[1] A person afflicted by a malady sought out the advice and care of another person who was "empowered" with the knowledge and skill to cure or ameliorate the affliction. Thus, historically, the physician-patient relationship became the center of medicine, where the primary concern of the physician is the health and care of the patient.

Exactly what are the responsibilities of the physician? It might be said that the physician's central responsibility is to use his medical expertise to assist the patient in the preservation of health. This can be accomplished by diagnosing the patient's condition and then informing and educating the patient about it (including prognosis and treatment options); the physician can then recommend a course of action that in his opinion constitutes the best approach for the problem; and finally, the physician can carry out the alternative chosen by the patient.[4]

The most crucial aspect of this sequence of events is the physician-patient relationship, which should and must be more than just a business relationship. The physician is a member of a profession, and this distinguishes the physician's activities from those of the automobile mechanic, plumber, or other craftsman. The word *profession* originates from the Latin word *profiteri*, meaning to declare aloud, or to make a public avowal. Thus entry into the profession was a public declaration of belief and intent to practice certain ideals. Through the ages it also came to mean a declaration of the possession of special skills to be used in the service of others.[5] Thus, the profession of medicine signifies the commitment of the special skills of medical knowledge and expertise to the service of others.

What held true for Peabody 60 years ago still holds true for clinicians today: "The good physician knows his patients through and through, and his knowledge is bought dearly. Time, sympathy and understanding must be lavishly dispensed, but the reward is to be found in that personal bond which forms the greatest satisfaction of the practice of medicine. One of the essential qualities of the clinician is interest in humanity, for the secret of the care of the patient is in caring for the patient."[6] In order to properly care for any patient, a physician needs to make a tremendous investment of self in getting to know the patient. Furthermore, the patient-physician relationship demands that patients put themselves in a very vulnerable position. They must undergo intense scrutiny, of both their bodies and the most intimate details of their lives. There needs to be a feeling of mutual respect between these parties for this relationship to survive and be successful.

This intimacy forms the heart of the physician-patient relationship. Each relationship is unique. Each patient has his own particular needs, fears, standards, and moral background. Thus no matter how each physician-patient relationship is structured, as Pellegrino[5] states, "the right decision is the one that is good for this patient—not patients in general, not what is good for physicians, for science, or even for society as a whole." Thus, this is indeed a very special professional and human relationship, between a potentially vulnerable individual seeking assistance in healing, and an individual professing to help and heal. One can be a physician only if one satisfies the essence of the medical act: that is, from within the physician-patient relationship, to come to the conclusion that is best for this particular patient.

ROLES WITHIN THE PHYSICIAN-PATIENT RELATIONSHIP

The roles of the physician and the patient have evolved through the ages. At least until the past few decades, paternalism on the part of the physician traditionally was a key element of this relationship. The attitude on the part of both physician and patient was that "doctor knows best." This was based on trust in the physician's technical skills and moral stature, and on the attribution of "magical" powers to the healer, and was characterized by patient dependency and physician control. For those times, it probably was appropriate. Few complex technical modes of diagnosis and treatment were available, and any major impact on morbidity and mortality essentially took place not as a result of medical advances, but because of improvements in public health.[7] Patients found great comfort in the presence of their physician during trying times, and this presence was greatly appreciated for its therapeutic benefits. This all seemed most appropriate when viewed in the context of the ancient precept, "above all, do no harm."

As medicine realized more scientific advances, the physician was equipped with a wider variety of options that could help the patient. Trust in the physician became a more crucial part of the relationship; the physician had to serve as the patient's advocate and represent the patient's interests in medical matters. The patient was extremely vulnerable in the situation in which diagnostic and therapeutic decisions needed to be made. Since most patients had little knowledge or understanding of the medical situation, they had to put their trust (and lives) in the judgment of the one person who knew them intimately and had mastery of the field, the physician. Thus paternalism, in the form of blind trust, ruled this intimate relationship.

As technology became more complicated, lay people also became more educated and sophisticated. They no longer blindly followed the treatment regimens prescribed by physicians without a clear understanding of why such regimens were appropriate. A clear example of this is the history of the treatment of breast carcinoma. For years, women afflicted with this cancer were subjected to the disfigurement of the radical mastectomy. It was through the insistence of lay women and the courage of researchers such as George Crile that less disfiguring procedures were found to be equally effective. People realized that they could and should provide input into medical decisions that affected their lives. Thus paternalism began to give way to a contractual model for the physician-patient relationship.

In this type of relationship, the physician enters into a contract with the patient, each party being viewed as equal. The physician pledges to commit his medical knowledge and skills to the service of the patient. The physician is compensated for his role under this contract; the patient is seeking help and is willing to pay the physician for it.[8] Thus the patient has sought out the physician to serve as his adviser when crucial decisions have to be made, and as the patient's advocate in the development and implementation of these decisions in the medical world. In the role of advocate, the physician speaks for the patient. However, unlike in the previous paternalistic model, the physician does so with the knowledge and informed consent of the patient. This is a special role. The physician is not merely a mouthpiece or a robotic medical technician employed by the patient. The physician works as an aggressive partisan for the betterment of the patient's health, and since it is a contractual relationship, the physician brings his moral ethics into the relationship. He tries to honestly recommend what he thinks is medically in the patient's best interest, and not just what the patient wants. Although there has been a shift in decision-making power to a much more equal basis, the physician also has the added responsibility of educating the patient and of obtaining informed consent. This new autonomy has also brought into question issues of cost, convenience, quality, and choice in health care provided to the patient.

These changes naturally led to a consumer awareness on the part of patients regarding choices in health care plans, physicians, and procedures that might be performed on their bodies.[9] The previously close and trusting allegiances to individual physicians and institutions were fading. In an upstate New York community, for example, an employer offered its employees a choice between continuing the traditional health insurance, which had a sizable deductible, or entering a health maintenance system that required almost no out of pocket expenses for the subscriber. Overnight approximately 30,000 people switched to the new program, many giving up long-standing relationships with physicians who did not participate in the health maintenance organization. This age of consumerism in medicine highlights a greater distancing between the physician and the patient. Previously clear distinctions between a professional relationship and that of a craftsman and client seem at times to be less clear. This distancing certainly complicates the malpractice situation.

According to Siegler, the patient-physician relationship has now entered the Age of Bureaucracy.[10] This is a time of cost containment and cost efficiency; pressures have been placed upon the physician by employers, hospitals, insurance companies, the government, and even investor-owned hospital chains. The distancing between the physician and patient is becoming such that the physician-patient relationship is now regarded by many as the provider-consumer relationship. It seems that something of great value is in jeopardy of being lost.

MEDICAL ECONOMICS

Who is to blame for the rise in health care costs that has led to the implementation of various schemes to control them, schemes that also threaten the physician-patient relationship? We can blame many parties—physicians, patients, employers, insurance companies, lawyers, and government.[11]

The Medicare and Medicaid programs were implemented in good faith to assist those who needed quality health care: the elderly and the impoverished. Until recently, reimbursement for most of these services was cost based. Consequently, there was an incentive for hospitals and other institutions to expand and to acquire expensive, sophisticated equipment, since these costs would be readily reimbursed by governmental funds. There seemed to be a bottomless well from which to obtain funds for such expansion. This fostered a style of medical practice that encouraged the use (sometimes inappropriately) of these resources. During this time, the technology of medicine made great, though expensive, advances, with the development of CT scanners, magnetic resonance scanning, new pharmaceutical agents, and so on, all of which drove up the costs of medical care.

Commercial insurers found it to be to their benefit to encourage practice methods that would increase premiums paid to them. The greater the premiums paid, the more capital would be available to these companies for investment purposes. American corporations and unions negotiated contracts that offered extensive health care benefits for employees since these benefits were not viewed by the government as taxable income. Unlike most insurance products, most health insurance plans had low or nonexistent deductibles, which encouraged beneficiaries to increase their utilization of health services. Physicians certainly complied, since it satisfied the patient and provided them with revenues while someone else was paying the bill. With such seemingly never-ending resources, the application of many costly medical innovations was not fully examined for their costs/benefits to both patients and society.

A situation such as this could not continue forever, and indeed we presently are witnessing a rude awakening to this reality. Government is faced with huge deficits and is now scrambling to implement containment mechanisms for health care expenditures. Employers cannot effectively compete with foreign companies and are unwilling to fund expensive health benefits, and insurance companies are devising newer and different health care plans that will meet the needs of employers yet remain profitable. People who have been used to carte blanche medical programs now must choose between programs that restrict subscriber choices and those that require higher out-of-pocket expenditures. The physician is caught in the midst of this turbulence.

Many health economists believe that it is the physician, not the patient, who controls most of the health care costs. Patients do have control when it comes to the selection of the physician and the extent of their demand for health care. However, most medical decisions are prescribed and implemented by the physician. It is the physician who advises the patient which diagnostic or treatment options should be exercised; it is the physician who generally possesses the professional expertise and knowledge regarding these options; and it is the physician who ultimately orders the implementation of these options. Thus, physicians tend to be the primary decision makers, either directly or indirectly, in the health care system.[12] It therefore is not surprising that a substantial amount of pressure is being applied by insurers, government, employers, and society in general to contain the costs that are generated in the care of patients. Unfortunately the physician cannot escape these pressures.

Besides these external influences, the physician is also experiencing increased personal financial pressures that certainly affect his relationship with patients. The cost of a medical education has risen dramatically over the past 20 years. The tuition fees of many private medical schools are approaching $20,000 a year. If one adds on to this the costs of living while in school, it is not surprising that a substantial number of medical school graduates have educational debts of $80,000–$100,000. This, in the face of rising malpractice premiums and ever-increasing start-up costs, makes entry into the private practice of medicine difficult economically.

Reimbursement from sources such as Medicare and Medicaid has been essentially frozen for many physicians already in practice; some states (such as Massachusetts) do not allow "balance billing." With an increasing number of new physicians being produced and the exodus of many patients into managed care systems, many physicians are experiencing a gradual loss of income in comparison to prior years. Many (especially younger physicians) have sought employment with managed care systems as a means of obtaining a secure form of income. Given this state of affairs, it is not surprising that many physicians in private practice are looking for other ways to supplement their incomes. It is not uncommon to find increased use of office procedural and laboratory services, investment in larger capital items such as CT scanners, ultrasound, etc, and investment in for-profit medical systems.

One wonders what effect such actions might have on the physician in his relationship with the patient. If Dr Jones has invested in a CT scanner, might he not be influenced to recommend a CT scan of the head for his patient who complains of headaches? In pre-ownership days, he would have performed a thorough neurological examination, and probably would have declined to perform a CT scan, satisfied with the diagnosis of migraine, which indeed the patient had. What about the physician who insists on performing flexible sigmoidoscopy yearly on asymptomatic patients for screening purposes, and then bills for this procedure as a limited colonoscopy? Are these and other similar actions and procedures truly in the patient's best interest? From the patient's perspective, it might appear that these actions are occurring for the physician's benefit under the guise of providing the patient with "top quality care," when in reality there is little or no benefit to the patient. One must agree that the benefit of many procedures is unclear. However, one might question the integrity of those physicians who are proponents of various controversial—and expensive—procedures who also happen to be the owners/shareholders of the equipment required for these procedures. There is an incredible temptation for well-meaning physicians to place their own interests above those of their patients.

Employment of physicians by various health care systems poses another different influence. An organization whose goal is to minimize expenses in providing health care can pressure physician employees to cut corners in providing care. These pressures can take the form of performance bonuses, capitation agreements, bounties, and salary. These dangers do exist. However, unlike their counterparts in fee-for-service private practice settings, many managed care systems have voluntarily elected or been mandated by employer groups or governmental agencies to have quality assurance programs in place to monitor the care which they provide to their members. This of course would not eliminate all abuses of such a system. However, it is a safeguard that encourages the physician to provide the top quality care that the patient requires.

THE PHYSICIAN'S RESPONSIBILITY TO SOCIETY

The physician also has a relationship with society in general. Physicians are indebted to the community for the monies spent to support their education, for the patients who were kind enough to share themselves with them in mind, spirit, and body during their training for the profession, and for the generally high level of prestige and income they enjoy as a result of their work.[13] Physicians as individuals and as a group are privileged to hold an elevated station in American society. Much of this is derived from the services they perform for individuals and the aggregate public good. One might speculate that some of this regard could also be a vestige of the belief in the "magical," at times unexplainable, power over life and death that physicians may appear to have. In any event, it seems that physicians should play a special role in ensuring the welfare of society, especially within the realm of health. Society has provided them with many privileges, some of which are rarely granted to other members of society, and as one's stature within society grows, so does one's responsibilities. Under the ancient principle of *noblesse oblige*, physicians have a special duty to promote the welfare and good of others.[14] The American Medical Association has recognized such an obligation and suggests that "the honored ideals of the medical profession imply that the responsibilities of the physician extend not only to the individual, but to society."[15] Thus, in the words of Veatch, the physician has "the well-being of both the individual and the community as an essential responsibility."[2]

This point of view may substantiate and validate the pressure now being placed on physicians to respond to and participate directly in cost containment efforts. One result of this pressure is the development of the "gatekeeper" role for primary physicians. Physicians have actually played such a role previously, since by tradition and law many therapies have had to be prescribed and implemented by a physician. This gatekeeper role has now acquired a component of financial control. In addition to ensuring the quality and coordination of care for the patient, the primary care physician has now become the fulcrum for the curtailment of costs.[16] Gatekeepers determine the necessity for specialized care, be it hospital, subspecialty, or supportive care referral. Without such screening, reimbursement for services may be denied. Thus the primary physician has become not only the controller of entry to medical care, but also the controller of the use of health care resources and the guardian of society's health care coffers.

There have been dramatic changes in the attitudes of government and many other players in the health care drama. Government, which was previously generous, has cut back on health expenditures through such methods as diagnosis related groups and managed care systems. For-profit hospital chains, which previously encouraged their affiliated physicians to maximize hospital charges by increasing the use of ancillary services and procedures, now encourage physicians to decrease hospital length of stay and minimize the use of expensive procedures. In all likelihood there has been much inefficiency and "fat" within the health care system. However, it is unclear how far cost cutting can go before patients experience deleterious effects from these efforts. There is a tremendous risk that physicians might be persuaded that their primary responsibility is the implied obligation for societal good in terms of cost containment.

CONFLICT OF OBLIGATION TO THE PATIENT VS SOCIETY

Given the current state of affairs, it is easy to see that in terms of health care, the needs of the individual and the desires of society are headed on a collision course, with the physician being in the unenviable middle position. On the one hand, the individual patient has placed his confidence in the physician as his advocate, who is doing all that is reasonably possible to promote and maintain the patient's well-being. On the other hand, society, as government, employers, and insurers of health care, is exerting pressure on the physician to restrict health resource usage in order to reduce rising costs. This highlights an incongruence between the value of an identifiable life and that of a statistical life.

In the physician-patient relationship, physicians deal with the value of an identifiable life. As Peabody has stated,". . . the secret of the care of the patient is in caring for the patient."[6] If he is to provide excellent health care, the physician must develop a personal, caring, mutually respectful relationship with his patients, and must have a clear understanding of the patients' needs, fears, and concerns. He must know each patient as a unique individual, with his own individual history. Can physicians as individuals give a monetary value to a spouse's life, a child's life, a close friend's life? It would be almost impossible to do so. If physicians were faced with a life and death situation involving someone dear to them, money would be no object. Yet a similar emotional concern faces the physician who has a caring relationship with his patients. In each case the physician is dealing with an identifiable life.

Society, via government, insurers, employers, and politicians, in making global decisions, usually deals with statistical values for life. For example, is the installation of a traffic signal for a particular intersection worth its cost of $40,000 if perhaps one life would be saved? In all probability, this decision would be made based on budgetary criteria. If indeed a life would be saved by the presence of this particular signal, no one knows whose life would be saved. We cannot give that life a name or a face. If we knew ahead of time whose life it would be, in all likelihood increased efforts, probably spearheaded by the identified individual, would be made to guarantee the installation of that $40,000 traffic signal.

This is the situation facing physicians today. Budgetary decisions for resource allocation are being made by various parties dealing with faceless, statistical lives, while physicians are faced with applying the impact of some of these decisions to the identifiable lives of their patients. Society is hoping that aggregately physicians will accordingly respond to its decisions based upon statistical data. Physicians, in order to remain true to their patients, place a much higher value on the identifiable life, and must continue to make the welfare of their individual patients the foremost consideration in these decisions.

There must be a middle ground between these at times conflicting views of the value of life. On the one hand one can advocate arbitrary criteria in order to conserve resources, such as: "no person over the age of 75 will be cared for in the intensive care unit." At the other extreme, one could encourage cardiac transplantation for all patients with heart disease, no matter what age. Both of these proposals are not likely to be viewed as responsible, yet they do highlight several points. Because we are in an era of financial constraint in health care, we cannot afford the reckless application of health care technology simply because it exists. Heroic procedures, especially high-priced ones, should be implemented only after careful consideration of whether the patient will truly benefit, with an improved quality of life, and whether such an improvement is worth the cost to the patient and society in general. Heart transplants simply cannot be provided on demand; they are too costly. Furthermore, arbitrary exclusion from care without careful analysis and consideration is irresponsible. If the success rate of cardiac transplantation is markedly diminished after a certain age, this would support setting an age limit for transplantation. Use of health resources needs to be evaluated on an individual basis in the context of what society can afford to underwrite. If a patient would meaningfully benefit from the care provided and society can afford to provide this care, the patient should not be excluded from care. The physician's role is to determine with and for the individual patient what would constitute responsible use of available resources; what would constitute meaningful care for the patient; and how the patient's quality of life can be improved by the proposed therapy. There are no simple answers to these questions, but they are questions that need to be explored by the physician in the milieu of the physician-patient relationship.

All of these issues arise under the condition that health care resources are to be expended in the present economic situation. No one has yet determined what portion of our nation's resources ought to be used for the provision of health care. One can enter into the "guns versus butter" discussion, and such a debate needs to occur at a national level. Furthermore, equity issues need to be addressed. Since there apparently are no correct answers to these issues, they must be debated by all responsible sectors of society, with the realization that difficult choices will need to be made. However, to ignore the effects of current constraints on resources while awaiting the outcome of the debate will lead to disastrous consequences for physicians, their patients, and the health care system.

Choices for the Future

These are indeed interesting times for the physician-patient relationship. This should not be viewed as a curse, however, but as a challenge to physicians to take an active role in making the practice of medicine better than ever. As a unique profession, different from any other, medicine needs to retain the caring component of the physician-patient relationship if it is to survive. Despite the pressures placed on patients—by insurers, lawyers, and other groups—to view themselves as consumers, it is and will continue to be important for the physician to relate to his patients as patients, not as consumers. The primary concern of the physician should be the maintenance of a close relationship with the patient so that together they can choose the best diagnostic and treatment options.

In so doing, the physician can still execute his duties on behalf of the patient in harmony with the demands of society. One would hope that in response to the needs of society, and without compromise to the patient, the physician will educate himself as to what is efficient, appropriate, and effective medical practice. Such knowledge would not only help eliminate waste of medical resources, but also optimize the care given to the patient. This can only improve the relationship with the patient, and minimize some of the dangers of malpractice by enhancing the standard of practice.

We cannot assume that physicians entered medicine primarily for monetary gain. If this were the case, many would have entered other, more lucrative fields. Nevertheless, it is imperative that physicians recognize the problems that financial self-interest can bring to the physician-patient relationship. We need to examine carefully the reasons we entered this profession. Physicians, especially those in primary care, should honestly question whether their direct ownership of various expensive technologies and equipment is really in the patient's best interest rather than their own. A careful examination of one's motives and responsibilities should hopefully lead to an appropriate decision. Again, this should make the patient in the relationship more of a patient rather than a consumer.

A unionization movement for physicians has developed in New York State and other states. Although this may be an effective means of representing physicians' concerns regarding the economic issues they face, such an effort widens the gap between the physician and patient and further distorts the distinction of medicine as a profession rather than a craft or trade group. If such a movement were to grow significantly, patients would view physicians as only providers of care, further encouraging the view of a provider-consumer relationship. If quality of medical care is the major concern, then the county and state medical societies are legitimate and appropriate vehicles for representing physicians' concerns on these issues. They must strive to support efforts for technology assessment, for quality assurance of medical care rendered, for effective physician peer review, for the innovation and development of new and better ways of providing care, and for representation of the profession in the debate concerning the equity of the allocation of health care resources.

Economic issues have become a reality in health care. It is the responsibility of physicians to acquaint themselves with these issues in order that, either as individuals or through their medical societies, they may represent both the physician's and the patient's viewpoints in society's debate regarding the direction that health care is to take. To avoid this challenge might be construed as an abdication of the responsibilities bestowed on the profession by society for its benefit and that of the physician's patients. Unfortunately, the response to this challenge has tended to be reactive in nature, rather than proactive. It is understandable that many would like to see the return of the "good old days," however, the reality is that conditions will never be the same. Such resistance to change has allowed businesspeople, bureaucrats, and other nonphysicians to take the initiative in the development of various health care models and schemes, some of which are highly unpalatable for physicians and patients. It is critical that physicians take a proactive role in the design and development of health care delivery systems that will allow the preservation of the physician-patient relationship and the delivery of quality health care. Since no one group can succeed by itself, physicians will need to project a posture of cooperation and willingness to constructively approach problems that arise from the interests of the many diverse groups in the health care drama. Furthermore, physicians will need to spearhead a drive to educate patients as to what constitutes quality health care and thus to modify patient expectations and demands from the health care system.

Many models of health care delivery are presently under consideration or are already in operation. For example, one sector of the health care field, the health maintenance organizations (HMOs), has raised the possibility of providing cost-effective care comparable in quality to that of traditional fee-for-service care.[17] Preferred provider organizations (PPOs) are being considered as a possible means of allowing physicians in private practice the capability of continuing to provide high quality care for their patients in conjunction with cost containment. There are many other models worthy of consideration. If the development and implementation of a system of care that is cost effective yet does not compromise the patient's interests can be achieved, it behooves physicians to seriously consider its adoption in the practice of medicine.

In 1984 the American College of Physicians produced a position paper which stated:

> Society has conferred great authority on medicine in the belief that physicians will use such power for the benefit of patients . . . Like any other good citizen, the physician should strive for the well-being of the community and of society. He should support community health endeavors. In particular, he should seek to use all health related resources in a technically appropriate and effective manner and to husband limited resources. He should conduct himself so as to merit the respect of his community, both as a professional and as an individual citizen . . . Under the covenant of personal medical care the physician is ordinarily the advocate and the champion of his patient, upholding the patient's interests above all others.[18]

Thus the challenge to the survival of the physician-patient relationship—and for that matter, of medicine as a profession of science, ethics, and caring—has been made. Action or inaction on the part of physicians will result in repercussions that will last for many years to come. Only then will we know for certain whether these interesting times were truly a curse, or were the opportunity offered to raise medicine to a better and higher state.

Acknowledgments. The author thanks Howard H. Hiatt, MD, and Vickie Myers Stankaitis, RPA-C, for their encouragement and help in conceptualizing the issues addressed in this paper. The Kellogg Foundation provided generous support during the study of these issues.

REFERENCES

1. Amundsen DW, Ferngren GB: Evolution of the patient-physician relationship: Antiquity through the Renaissance, in Shelp EE (ed): *The Clinical Encounter.* Boston, D. Reidel Publishing Co, 1983, pp 3–46.
2. Veatch RM: *A Theory of Medical Ethics.* New York, Basic Books Inc, 1981.
3. Franck I: Jewish religious law as a model of the patient-physician relationship: A comment on Professor Brody's essay, in Shelp EE (ed): *The Clinical Encounter.* Boston, D. Reidel Publishing Co, 1983, pp 141–148.
4. Jonsen AR, Siegler M, Winslade WJ: *Clinical Ethics.* New York, Macmillan Publishing Co Inc, 1982.
5. Pellegrino ED: *Humanism and the Physician.* Knoxville, Tenn, University of Tennessee Press, 1979.
6. Peabody FW: The care of the patient. *JAMA* 1927; 88:877–882.
7. Powles J: On the limitations of modern medicine, in *Science, Medicine and Man.* Elmsford, NY, Pergamon Press, 1973, vol 1, pp 1–30.
8. Quill TE: Partnerships in patient care: A contractual approach. *Ann Intern Med* 1983; 98:228–234.
9. Haug M, Lavin B: *Consumerism in Medicine: Challenging Physician Authority.* Beverly Hills, Calif, Sage Publications Inc, 1983.
10. Siegler M: The progression of medicine. From physician paternalism to patient autonomy to bureaucratic parsimony. *Arch Intern Med* 1985; 145:713–715.
11. Feldstein M, Taylor A: *The Rapid Rise of Hospital Costs.* Council on Wage and Price Stability, 1977.
12. Enthoven AC: Consumer-choice health plan (first of two parts). Inflation and inequity in health care today. Alternatives for cost control and an analysis of proposals for national health insurance. *N Engl J Med* 1978; 298:650–658.
13. May WF: Code, covenant, contract, or philanthropy. *Hastings Cent Rep* 1975; 5:29–38.
14. Ballantine HT: Annual discourse—the crisis in ethics, anno Domini 1979. *N Engl J Med* 1979; 301:634–638.
15. American Medical Association: Principles of medical ethics, 1957.
16. Eisenberg JM: The internist as gatekeeper. Preparing the general internist for a new role. *Ann Intern Med* 1985; 102:537–543.
17. Sloss EM, Keeler EB, Brook RH, et al: Effect of a health maintenance organization on physiologic health. Results from a randomized trial. *Ann Intern Med* 1987; 106:130–138.
18. Ad Hoc Committee on Medical Ethics, American College of Physicians: American College of Physicians Ethics Manual. Part I: History of medical ethics, the physician and the patient, the physician's relationship to other physicians, the physician and society. *Ann Intern Med* 1984; 101:129–137.

FROM THE LIBRARY

THE PHYSICIAN AND THE PATIENT OF THE FUTURE

. . . We build on the experience of the past. The rapid strides in medical discoveries of the last two generations, together with the more practical employment of sanitation and preventive medicine, have already effected great changes in the actual duties of the practicing physician. The daily round of our immediate predecessors was made up largely of the treatment of infectious and contagious diseases. The day book of the family physician usually recorded visits to families for the treatment of measles, scarlet fever, diphtheria, typhoid fever, or malaria only. He was educated to treat disease. He knew little of preventive medicine, sanitation, or hygiene, nor did his medical education provide such knowledge. Older living practitioners know of the changes which mark the new era only by keeping abreast of the times.

National, state, and municipal departments of health have gradually become powerful and controlling factors in preventive medicine, sanitation, and hygiene. Health and welfare organizations, both national and local, have also invaded the field of preventive medicine. Great industrial organizations have introduced various health measures, believing it to be for their own best interests from an economical standpoint, as well as for the benefit of the hundreds of thousands of employees. Pay clinics and group practice have made some slight imprint on the modes of practice of to-day. Furthermore, the publicity work of all these agencies, together with that of our own Association, has gradually created a demand from the public for more information regarding personal health. The outcome of these measures has led to confusion regarding not only the present but also the future of medical practice. But the confusion of the present need never become chaos in the future, provided the medical profession prepares to meet the demands of the patients of the future. . . .

WENDELL C. PHILLIPS, MD
(*NY State J Med* 1926; 26:375–378)

Mandatory Medicare assignment

MARIO V. MENGHINI

Physicians throughout the country are currently expressing grave concern over varying state proposals mandating mandatory acceptance of Medicare assignment. It is ironic to note that the Congress, which enacted Medicare in 1965, was so concerned about the possibility of inadequate physician participation that it sought to give every assurance the new program would not be used as a vehicle to interfere with physician/patient relationships. This assurance finds expression in the first section of the Medicare Act, which remains unchanged to this day: "Nothing [in Medicare] shall be construed to authorize any federal officer or employee to exercise any supervision or control over. . . the manner in which medical services are provided. . . ."[1]

The congressional concern over avoiding government interference in traditional physician practices through the Medicare Act is especially apparent in the Act's provision governing the billing of program beneficiaries. Congress included a provision under which a physician could elect to accept assignment and limit fees to an amount determined by the Medicare carrier. At the same time it provided physicians with the option to decline assignment acceptance.

Congress specifically gave physicians the option to deal directly with their Medicare patients and to charge accordingly. This set of alternatives finds expression in 42 USC § 1395u(b)(3)(B), which specifies that payment under Medicare will be made either "(i) on the basis of an itemized bill, or (ii) on the basis of an assignment under the terms of which. . . the reasonable charge is the full charge for the service. . . ."

An assignment is an agreement wherein the Medicare beneficiary transfers to the physician the right to payment of covered services identified on a submitted Medicare claim form. The physician, in return, agrees to accept the reasonable charge determined by the carrier as the full fee (encompassing deductible and coinsurance amounts) for the services rendered. In essence, the physician who accepts assignment is precluded from billing the beneficiary more than the deductible and coinsurance amounts predicated upon the carrier's determination of a reasonable charge. Under this arrangement, it is the physician who elects to accept assignment, whereupon he or she will be paid directly by the carrier; or not to accept assignment, in which case the carrier will pay the beneficiary directly.

On April 12, 1984, the House of Representatives rejected a proposal calling for mandated assignment tied to a one-year freeze in the prevailing charge levels for services provided to hospital inpatients. Since that action, the Congress created the physician "participation" program under the Deficit Reduction Act of 1984, whereby physicians are provided the option to participate in the Medicare program and accept all Medicare claims on assignment. Furthermore, on April 8, 1987, a mandatory assignment amendment to a proposal to incorporate acute care catastrophic coverage into Medicare was offered and withdrawn in a meeting of the Health Subcommittee of the House Ways and Means Committee.

Despite the foregoing, a startling and ominous development occurred in 1985 when the Massachusetts legislature unanimously enacted a law requiring physicians, as a condition of licensure or license renewal, to agree not to charge or collect from Medicare beneficiaries any amount in excess of the reasonable charge for that service, as determined by the Medicare carrier in compliance with guidelines emanating from the Office of the Secretary of the Department of Health and Human Services.[2] That law directs the Massachusetts Board of Registration in Medicine to:

> . . . require as a condition of granting or renewing a physician's certificate of registration, that the physician, who if he agrees to treat [a Medicare beneficiary] shall also agree not to charge to or collect from such beneficiary any amount in excess of the reasonable charge for that service as determined by the United States Secretary of Health and Human Services.

Physicians would still be required to bill for deductible and coinsurance amounts, except in cases of financial hardship.

The Massachusetts Medical Society (MMS) and the American Medical Association (AMA) sued to challenge the constitutionality of the state statute on the grounds that the law:

- was inconsistent with the federal Medicare Act, and conflicted with federal Medicare statutes, ie, the US Constitution requires that a federal law always take precedence over a state law (the "Supremacy Clause");

Address correspondence to Mr Menghini, Director, Division of Socio-Medical Economics, Medical Society of the State of New York, 420 Lakeville Rd, Lake Success, NY 11042.

- was violative of due process;
- conflicted with the congressional intent to allow physicians an option in the acceptance of assignment;
- would diminish the access of Medicare beneficiaries in the state to medical care as physicians withdraw from involvement in the Medicare program;
- would have an adverse impact on medical innovation, with the likelihood that the best qualified physicians would refuse to treat Medicare patients; and
- would have a disparate impact on rural areas.[3]

Although the mandatory assignment concept has been rejected legislatively on the federal level, on June 5, 1986, the Federal District Court for the District of Massachusetts upheld the state's statute, ruling that it does not violate the Supremacy Clause of the Constitution (ie, Massachusetts state law was not found to be in conflict with any portion of the federal Medicare Act); encroach upon any field occupied by Congress; or violate due process.[4]

The case was appealed to the First Circuit Court of Appeals, which affirmed the lower court's decision in an opinion issued on March 30, 1987.[5] The opinion of the US Court of Appeals in Boston had a significant impact on the effort to protect a physician's right to decline acceptance of Medicare assignment when mandated to do so as a condition of licensure. Even though Congress had on various occasions voted to give physicians the option of taking or rejecting assignment, the court asserted that it is permissible for states to force physicians to accept assignment. It was the court's assessment that Congress, in enacting the Medicare law, was not clear enough in delineating physician freedom to accept or decline assignment. In *Massachusetts Medical Society v Dukakis*, the appeals court held that states traditionally have regulated physician fees, and when Congress gave physicians the option of taking Medicare assignment, it was not explicit enough in asserting that this option is actually a "right" preventing states from fee interference.[5]

AMA lawyers contested that the distinction the appeals court made between an "option" and a "right" to reject assignment seemed meaningless, in view of Congress' explicitly giving physicians the option of balance billing. Consequently, the question arose as to whether a state can nullify an option Congress established in a federal program. That is what the AMA will argue in applying for a review of the case by the US Supreme Court. Whether the court will decide to hear the case, however, is questionable since it generally hears only about 5% of the cases that come before it, and then usually because there is a difference of opinion among appeals courts in other similar cases.

As the law in Massachusetts stands now, physicians have no right to decline assignment, no matter what the market dictates or the individual patient can afford to pay. The state assistant attorney general has asserted that the appeals court decision will allow other states to pass similar laws. However, if the Supreme Court does hear the case, or if Congress further clarifies its intent that Medicare operates more effectively when physicians are not subject to restrictive government regulations, that option could be precluded.

Given the state's unique environment, it is not surprising that legislation of the foregoing nature was enacted in Massachusetts. Under state law regulating Massachusetts Blue Shield, physicians must sign a participation agreement in order to receive reimbursement from the plan, thereby precluding balance billing of patients; and Blue Shield payments are not permitted to be made to either the doctor or the patient for services performed by a nonparticipating physician.

The National Perspective

Although Massachusetts is the only state to date to have enacted a law conditioning physician licensure on acceptance of Medicare assignment, similar legislation was introduced in a number of other states in 1986 and new proposals have surfaced in 1987. While the predominant approach of these proposals has thus far been similar to that of Massachusetts, bills have also appeared which tie refusal to accept the Medicare reasonable amount to violations of a state's Consumer Protection Act, allegations of unprofessional conduct and subjection to medical discipline, the grant or denial of hospital staff privileges, and a misdemeanor violation of criminal law.

The introduction of such legislation in these states is of great concern to physicians, especially with respect to their potential impact on access to quality medical care for the nation's elderly population.

Following are specific examples of the differing types of bills introduced in other state legislatures since enactment of the Massachusetts law.[6]

Acceptance of Medicare Assignment Tied to Licensure. In 1986, bills similar to the Massachusetts law were introduced in other states which, as a condition of licensure to practice medicine, require physicians not to charge or collect from beneficiaries any amounts in excess of Medicare-determined allowable charges. While none of these bills attained passage last year, similar legislation is, or was, pending in the 1987 legislative sessions in Arkansas, Connecticut, Iowa, New Hampshire, New Jersey, New York, Ohio, Oregon, Rhode Island, West Virginia, and Vermont. In Arkansas, New Hampshire, and New Jersey, the 1987 legislative proposals were defeated. While the tie to licensure was eliminated from the bill passed in Vermont, compliance with its provisions will be monitored by the State Medical Board. Vermont physicians are now required to post a summary of the law's provisions in their offices, and to accept assignment on Medicare beneficiaries with annual incomes of $25,000 or less for a single person, or $32,000 or less for a married couple (home and office medical visits are excluded from the requirement for mandatory assignment acceptance).

In Connecticut, the state legislature passed a compromise mandatory assignment bill for physicians who treat Medicare patients, with the legislation establishing a Medicare assignment contract program in the state Department on Aging. The program currently calls for the Connecticut State Medical Society to issue medical courtesy cards to beneficiaries whose incomes are less than $19,950 for individuals or $24,000 for couples, with physicians participating on a voluntary basis, accepting assignment for care given to cardholders.

After July 1, 1988, the program requires that at least:

- 85% of Medicare claims in the state be taken on assignment;
- 85% of the physicians in each county who treat Medicare patients participate in the courtesy card program;
- 80% of physicians statewide in each specialty who treat Medicare patients participate in the courtesy card program;
- 50,000 of income-eligible beneficiaries be enrolled.

If these levels are not met, the Department on Aging is to adopt regulations requiring that all physicians limit their charges to Medicare's reasonable amount for income-eligible patients. Physicians who violate the regulations would be subject to civil penalties of up to $1,000 per charge.

The Connecticut law also requires physicians with Medicare provider numbers to post their policies on Medicare payments and processing in a prominent office location. Despite the fact that the Connecticut State Medical Society's medical courtesy card program is in place for the needy elderly, and that about 75% of the state's physicians voluntarily participate, the Connecticut state legislature deemed it necessary to pass the foregoing legislation.

Acceptance of Assignment and Consumer Protection. In 1986 the Washington state legislature considered a bill which would have redefined "unprofessional conduct" to include the failure to accept the Medicare-determined reasonable charge as payment in full for patients whose income is at or below 200% of the poverty level. In addition, a coalition of senior citizen groups formulated an initiative to be placed on the 1987 ballot which would have made it a violation of the state's Consumer Protection Act for any physician to "charge, collect or attempt to collect for medical services provided to any Medicare-eligible patient, any amount in excess of the Medicare-determined charge." Ultimately, the bill did not pass, primarily because of effective interaction between the Washington State Medical Association and the state's senior citizen groups.

In Montana, a bill was introduced in a recent legislative session which would have made it a violation of the state's Consumer Protection Act for a health care provider to charge Medicare patients more than approved Medicare rates for treatment. The bill, which would have also required posting of its provisions in the place of business of a health care provider, failed to pass.

Acceptance of Assignment and Medical Discipline. In 1986, a proposal in California would have made it an element of "unprofessional conduct" for a physician to refuse to treat anyone over the age of 65 on the grounds that the person's only health insurance is Medicare or Medi-Cal. Although hearings were held on this proposal, a formal bill was not introduced last year in the California legislature. Legislation has been introduced, however, for the 1987 session.

An Ohio bill formulated at the end of 1986 and reintroduced in 1987 would also make it an element of "unprofessional conduct" to knowingly charge or collect from an individual an amount in excess of the Medicare reasonable amount "for a health service the practitioner agreed to provide. . .under Title XVIII of the Social Security Act."

Legislation recently passed by the Rhode Island state legislature and signed by the governor expands the definition of "unprofessional conduct" to include charging more than the Medicare reasonable amount for individuals and couples with income levels of $12,000 and $15,000, respectively.

An Illinois bill calling for the state's Department of Registration and Education to discipline physicians for failure to accept assignment of all Medicare claims has been introduced in the Illinois General Assembly. The bill would subject physicians who balance bill a Medicare beneficiary to disciplinary proceedings that could result in license revocation, suspension, probation, or other disciplinary action as deemed proper by the Department through the Medical Disciplinary Board. Proponents of the measure include the Coalition for Consumer Rights and the Illinois Public Action Council.

Acceptance of Assignment and Hospital Staff Privileges. A 1985 Nevada bill, which failed to pass, would have directed every licensed hospital to "require as a condition of employment or extension of privileges that a practitioner of a healing art accept as full payment for any services provided in the hospital the assignment of benefits payable pursuant to 42 U.S.C. § 1395d and 1395l."

Failure to Accept the Medicare-Determined Amount as a Criminal Violation. A bill introduced in Maryland in 1987 would have made it a criminal violation for a physician to charge a Medicare beneficiary any more than the Medicare-determined charge for that service. Violation of this provision would have subjected a physician to a fine of up to $5,000 or imprisonment of up to five years, but the Maryland bill was defeated.

It is noted that proponents of mandatory assignment include a group calling itself the National Health Care Campaign, a grassroots effort to improve health care headed by Dr Arthur Fleming, former Secretary of the US Department of Health, Education and Welfare. This group has identified mandatory Medicare assignment as among the various issues it will be pursuing in approximately 20 states, including California, Colorado, Connecticut, Florida, Idaho, Illinois, Indiana, Iowa, Maryland, Minnesota, Montana, New Jersey, New York, North Carolina, Ohio, Oregon, Pennsylvania, Rhode Island, and Washington. In some states, the state chapters of the American Association of Retired Persons (AARP) have either opposed state legislation or have taken a neutral position on state mandatory assignment because of the deleterious effect it could have on access to quality medical care for the elderly. However, it should be noted that senior citizen organizations and activist consumer groups have been promoting mandatory assignment at state levels.

Interestingly, the current rate of physician assignment acceptance on a national level is significant. For the last quarter of 1986, 68.2% of all Part B claims and 70% of charges comprising these claims were accepted on an assigned basis. This shows the continuation of a trend toward increased acceptance of assignment for Medicare Part B claims, from 51.4% in 1980 to 67.7% in 1985.

Although most physicians do not formally participate in the Medicare program, nonparticipating physicians nationwide submitted 61% of charges for covered services during the third quarter of 1986, with 46% of these services being accepted on an assigned basis.

Furthermore, it has been determined that acceptance of assignment on a claim-by-claim basis has not resulted in undue hardship to beneficiaries. Based on 1982 Health Care Financing Administration data (when the assignment rate was only 52.8%), only 1,032,600 of 25,363,000 aged enrollees (or a little over 4%) had Part B financial liability in excess of $1,000. In addition, 13,392,600 of the more than 16 million beneficiaries who used Part B services (or a little over 83%) had out-of-pocket expenses of less than $500. These expenses included the statutorily mandated deductible ($75) and coinsurance amounts.

It seems evident that mandated assignment will result in subsidizing the cost of care for a population apparently able to fulfill its financial commitments. In fact, recent statistics point out that many of the nation's elderly are in a position to meet physicians' regular charges, with elderly Americans as a group reported to have the lowest poverty rates and more discretionary dollars to spend, after taxes and expenses, than any other age group in the nation. It is also important to emphasize that mandated assignment would force a shift in costs to other segments of the population and across generations. Requiring physicians to accept assignment for patients who are not in need of such preferential treatment would turn the Medicare program into a welfare-type program for the many individuals who are not in need of subsidy. Consequently, it is logical to conclude that attaining a specific age, as opposed to actual need, should not be the criterion for preferential treatment and governmental presumption of economic need. Most noteworthy, mandated assignment would result in reduced access to services if physicians should choose not to accede to governmental encroachment and refuse to treat Medicare beneficiaries. In fact, a report prepared pursuant to an HCFA grant and published in the Summer 1983 issue of *Health Care Financing Review*[7] indicates that if physicians were placed in the position of having to decide whether to accept assignment for all of their Medicare patients or for none of them, more than two-thirds of physicians would choose to take no assignments.

THE NEW YORK STATE PERSPECTIVE

Concurrent with the foregoing activities on a national level concerning mandatory assignment, Assembly Bill No. A.6801 was introduced in the New York State legislature in May 1987 (significant action on this bill is not anticipated until late 1987 or early 1988). The bill reads as follows:

> An ACT to amend the education law, in relation to requirements for a professional license for a physician.
> *The People of the State of New York, represented in Senate and Assembly,* do enact as follows:
>
> Section 1. Section sixty-five hundred twenty-four of the education law is amended by adding a new subdivision eleven to read as follows:
> (*11*) *No license or registration shall be issued to any applicant under this section unless the applicant shall agree in writing that if in the course of the practice of his profession, such applicant agrees to treat a beneficiary of health insurance under Title XVIII of the Social Security Act, he shall also agree not to charge to or collect from such beneficiary any amount in excess of the reasonable charge for that service as determined by the United States Secretary of Health and Human Services.*
> § 2. This act shall take effect immediately.

The following information is included in the New York State Assembly memorandum in support of the proposed legislation:

> TITLE OF BILL: AN ACT to amend the education law, in relation to requirements for a professional license for a physician.
> PURPOSE OR GENERAL IDEA OF BILL: The purpose of this bill is to help contain health care costs for older New Yorkers.
> SUMMARY OF SPECIFIC PROVISIONS: This bill would require all physicians who agree to treat Medicare patients, as a condition of licensure, to charge Medicare patients no more than the Medicare determined reasonable fee.
> EFFECTS OF PRESENT LAW WHICH THIS BILL WOULD ALTER: Adds a new subdivision 11 to section 6524 of the Education Law.
> JUSTIFICATION: Established in 1965, Medicare is a federal health insurance program for people 65 and over and some of the disabled. Medicare premiums are paid by the elderly and out of revenues from social security taxes. Medicare beneficiaries are required to pay a deductible and copayment for the various services provided. Under the Medicare program there are two methods of payment. In the first method, the physician agrees to accept "assignment". This means that he/she agrees to accept the reasonable fee set by the Department of Health and Human Services (HHS) as payment in full for the service provided. The physician then directly receives 80% of this reasonable charge from Medicare and collects the remaining 20% from the patient. Under the second method the physician bills the patient directly for the service. The patient then collects reimbursement from Medicare that equals 80% of the charge for that service. The patient is then responsible for the remaining 20% plus 100% of any overcharge above the reasonable charge levied by the physician. The practice by physicians, of charging more than Medicare's reasonable charge, is commonly called "balance billing." In spite of the protection provided by Medicare, health care costs for the elderly remain unpredictable and continue to pose an ominous threat to their financial security and independence. This legislation will limit what physicians can charge their Medicare patients, insure that the rates they are charged are reasonable and predictable and will help to alleviate the burden of rising health care costs for older persons.
> PRIOR LEGISLATIVE HISTORY: New bill.
> FISCAL IMPLICATION FOR STATE AND LOCAL GOVERNMENTS: None to the State.
> EFFECTIVE DATE: Immediate.

THE POSITION OF THE MEDICAL SOCIETY OF THE STATE OF NEW YORK

In opposition to mandatory assignment, the Medical Society of the State of New York (MSSNY) affirms that:

- Physician licensure should only be related to education, training, and ethical criteria, rather than to acceptance of Medicare assignment.
- Mandatory assignment has the potential to compound the existing two-tiered system of medical care created by the Deficit Reduction Act of 1984, which established the Medicare participating and nonparticipating physician categories.
- Mandatory assignment will have an adverse impact

on accessibility to providers and on quality of care, particularly in already underserved rural areas of the state.

- Incentives for physicians to adopt innovative technologies in a state with mandatory assignment could be discouraged, as their income is significantly reduced and spending is affected accordingly.
- Younger physicians will likely not choose to set up practice in a mandated-assignment state, and older physicians will be discouraged from continuing practice and will retire earlier.
- In view of the present 72% rate of acceptance of assignment on Medicare claims by participating and nonparticipating physicians in New York State, mandatory assignment legislation is unnecessary.
- In accordance with AMA assessments, reform of the entire Medicare program should be undertaken rather than addressing its inequities through such approaches as state mandatory assignment legislation.
- The spirit and intent of the Medicare law as originally enacted, which gave physicians the option to bill beneficiaries directly or accept assignment, should be reaffirmed under all circumstances.

The MSSNY believes that the current reimbursement system in which assignment is accepted by nonparticipating physicians on a claim-by-claim basis, and in which Medicare patients have access to nearly all physicians in the state, has not resulted in undue beneficiary liability and has not been abused by the medical profession. The MSSNY continues to urge physicians to consider each patient's financial needs when setting charges, and to accept Medicare assignment, reduce fees, or charge no fee at all in financial-hardship cases.

On behalf of both the state's elderly population and the physician community, the MSSNY:

- has been conducting ongoing liaison activities with the Statewide Seniors Action Council—a leading senior citizen group in New York State—concerning the overall negative ramifications of mandatory assignment legislation;
- plans to sponsor educational senior citizen workshops conducted by volunteer physicians throughout the state concerning the pitfalls of mandatory assignment vis-a-vis accessibility and quality of medical care;
- is preparing testimony stressing the negative impact that mandatory assignment would have on accessibility and quality of care in anticipation of state legislative hearings;
- is studying the feasibility of patient advocacy activities to assist beneficiaries with claims problems experienced with Medicare carriers;
- is assessing the potential of county medical society sponsored voluntary physician assignment acceptance programs;
- reaffirms its encouragement of fee discussions between physicians and patients, with recommendations for assignment acceptance, and appropriate fee considerations for cases of financial hardship.

REFERENCES

1. 42 USC § 1395.
2. Massachusetts Acts of 1985, ch 475.
3. Brief of the Massachusetts Medical Society and the American Medical Association to the United States Court of Appeals for the First Circuit, Boston, 1987.
4. *Massachusetts Medical Society v Dukakis*, No. 85-4312-K, slip op (D Mass, June 5, 1986).
5. *Massachusetts Medical Society v Dukakis*, No. 86-1575, US Court of Appeals, 1st Cir.
6. Division of Legislative Activities: *Mandatory Assignment: A Legislative Guide*. Chicago, Ill, American Medical Association, 1987.
7. Mitchell JB, Cromwell J: Impact of an all-or-nothing assignment requirement under Medicare. *Health Care Finan Rev* 1983; 4(4):59–78.

Bile peritonitis following expectant management of liver fracture

STEPHEN L. BARKER, MD, DAVID FROMM, MD

The liver is the second most commonly injured abdominal organ in cases of trauma, exceeded only by the small bowel in penetrating injuries and the spleen in blunt trauma. Since World War I, when the mortality rate was 66%, there has been a steady decrease in the mortality rate associated with hepatic injuries. Current civilian series report mortality rates of 10–20%, with exsanguination as the leading cause of death.[1] Hemorrhage from these injuries can be massive, reflecting 1,500 mL/min of splanchnic/hepatic blood flow, as well as a potential 1,200 mL/min of renal/pelvic/leg venous return in cases of injury to the retrohepatic vena cava.[2] Despite this, at least half of all liver injuries are not actively bleeding at the time of laparotomy.[3]

Recent reviews of liver injury have stressed surgical control of hemorrhage by various means, ranging from temporary packing to suture, electrocautery, microfibrillar collagen, selective hepatic artery ligation, and hepatic resection.[1-8] While hemorrhage from hepatic injury is the most dramatic and immediately life-threatening consequence of blunt trauma to this organ, delayed and long-term complications relate instead to infectious and biliary tract problems.[9] These insidious processes may become more apparent as a result of recent trends toward nonsurgical management of some cases, as illustrated in this report.

CASE REPORT

An 18-year-old man was brought to the emergency room after a motorcycle accident. His only complaint was vague abdominal pain in the right upper quadrant. He was not in acute distress. His pulse was 54 beats/min, and his blood pressure was 120/86 mm Hg. Mild right upper quadrant tenderness was present. Pertinent laboratory findings included a hematocrit of 37%, white blood cell count of 10,100/mm^3, and a serum glutamic-oxaloacetic transaminase (SGOT) level of 283 U/L (normal range, 0–41 U/L). Plain radiographs of the chest and abdomen were normal.

Over the next 24 hours, the patient's hematocrit fell to 31%. His abdominal discomfort increased, and he noted pain in the right shoulder. A computed tomographic (CT) scan showed a fracture of the right lobe of the liver with fluid in both paracolic gutters (Fig 1). Although many physicians would have operated at this point, it was elected to continue a period of cautious observation. The patient's hematocrit slowly drifted downward, and he received two units of blood. Thereafter he seemed to improve, his hematocrit stabilized, and he was placed on a solid-foods diet.

The hepatic enzyme levels reflected liver injury. Peak values were as follows: bilirubin, 5.1 mg/dL; alkaline phosphatase, 108 U/L (normal range, 20-90 U/L); lactate dehydrogenase, 1,275 U/L (normal range, 100–225 U/L); SGOT, 665 U/L, and serum glutamic-pyruvic transaminase (SGPT), 286 U/L (normal range, 3-36 U/L). These values all slowly declined to less elevated levels.

The patient's temperature rose to 103°F 14 days after the injury. He began to complain of more generalized abdominal discomfort, and ileus was evident on abdominal radiographs. His white blood cell count was 13,300/mm^3, with 85% neutrophils and no band forms. Parenteral nutrition was begun. Paracentesis delivered dark serous fluid, with a bilirubin level of 40.7 mg/dL and a white blood cell count of 3000/mm^3 with 50% neutrophils and 50% mononuclear cells. Antibiotic therapy was begun, but the patient continued to have more diffuse abdominal pain and spiking temperatures. A repeat CT scan showed a decrease in the size of the liver fracture but an increase in peritoneal fluid (Fig 2). Abdominal distention and peritonitis were clinically evident, and the patient was taken to the operating room with a preoperative diagnosis of probable intra-abdominal abscess.

Bilious fluid, 5,200 cc, was evacuated from the peritoneal cavity. The peritoneal surfaces were grossly inflamed and were covered with shaggy fibrinous exudate. The gallbladder appeared somewhat collapsed. Adhesions to the dome of the liver were taken down, revealing a capsular defect corresponding to the recent fracture. There was no bile staining of the retroperitoneal fat behind the duodenum. Because of the uncertain source of the bile leak, a cholecystocholangiogram was performed and disclosed a transected intrahepatic tributary of the right hepatic duct with extravasation of dye through the right lobe (Fig 3). A cholecystostomy was performed, and drains were placed in the right upper abdomen. Aerobic and anaerobic cultures of the peritoneal fluid showed no growth. Bilious fluid drained from the drain sites, decreasing in volume over a ten-day period. The drains were re-

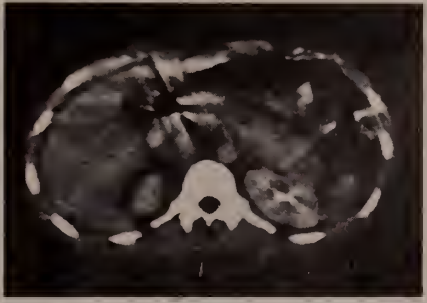


FIGURE 1. CT scan showing a large laceration of the right lobe of the liver with hemoperitoneum.

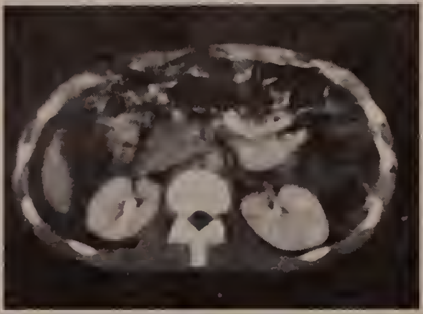


FIGURE 2. CT scan showing increasing intraperitoneal fluid.

From the Department of Surgery, State University of New York Health Science Center at Syracuse, NY.

Address correspondence to Dr Fromm, Department of Surgery, University Hospital, 750 East Adams St, Syracuse, NY 13210.

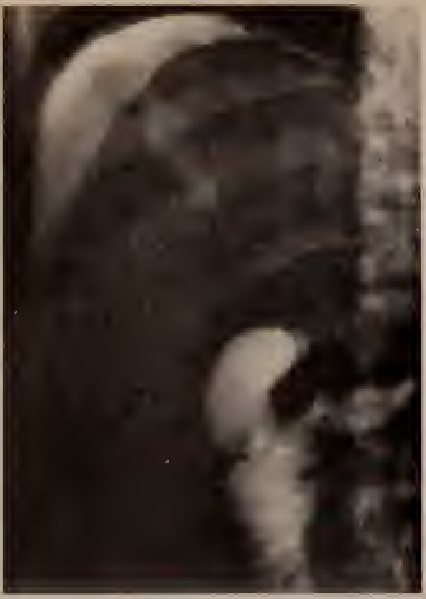


FIGURE 3. Operative cholecystocholangiogram showing the right hepatic duct with contrast extravasating into a laceration of the liver and the subdiaphragmatic space.

moved on the 16th day after surgery. The patient is doing well one year after injury.

Discussion

The true incidence of bilious leak from intrahepatic ductal injury following trauma is not known, although the incidence of biliary peritonitis and fistula has been reported to vary from 2% to 4% in many large series of hepatic trauma.[10] When intraperitoneal bile is found following a blow to the abdomen, the most likely source is a laceration or tear of the liver due to the size and relative fixation of the organ. Rupture of the extrahepatic bile ducts should be considered next, with the gallbladder being the least likely source of choleperitoneum.[11] There are reports of at least 36 cases of avulsion of the common bile duct and 101 cases of gallbladder injuries.[12,13] Conversely, reports of bile leakage from intrahepatic ductal disruption are rare.

Although the risk of postoperative bile drainage from a liver laceration is roughly proportional to the size of the wound, copious flow may occur in small wounds if a major biliary radical is disrupted. There are no reliable criteria by which such cases can be selected prior to or during surgery,[14] a time when there normally appears to be a diminution of bile flow. This has been the rationale for advocating drainage for liver wounds since Madding's[15] World War II series of 829 liver injuries. He expressed the opinion that the establishment of adequate external drainage for both bile and products of tissue injury is the most important feature in the surgical care of liver wounds.

Others also endorse the concept of drainage for all liver wounds, claiming that complications relating to sepsis and the biliary tree are almost universally related to inadequate drainage.[14] According to still others, the sole exception to mandatory drainage for all liver injuries is the laceration which is cleanly incised and superficial, similar to a hepatic biopsy wound, which may be treated by suture only.[16] The scarcity of reports of complications from intraperitoneal accumulation of bile as a result of intrahepatic rupture may be due to the widespread use of drains in cases of parenchymal laceration.

The current trend of nonoperative management in selected instances of hepatic injury leads to the speculation that the incidence of biliary complications may increase. However, complications have been few when strict criteria[17-20] are applied to such management which requires accurate definition of the degree of injury afforded by such modalities as radionuclide scanning, ultrasonography, and, particularly, CT scanning. For example, Meyer et al[19] advocate nonoperative management (in the absence of other clinical indications for laparotomy) in the presence of simple parenchymal laceration or intrahepatic hematoma that does not become enhanced following intravenous administration of contrast material, an estimated amount of free intraperitoneal blood of less than 250 mL (by its restriction to Morison's pouch), and the absence of other intraperitoneal injury. Other authors extend these criteria to include more extensive parenchymal lacerations and regard the amount of intraperitoneal fluid to be the more critical factor in electing a nonoperative course.[20] In this regard, neither hepatic scintigraphy nor diagnostic peritoneal lavage are of much value, because they do not provide data relative to blood volume lost into the peritoneal cavity. Furthermore, lavage has repeatedly been shown to be positive in the presence of small amounts of intraperitoneal blood (as little as 25 mL) and gives little information about the source of bleeding.[20]

Biliary complications have not been reported in four pediatric series (total of 59 patients) and one adult series (24 patients) of blunt hepatic trauma treated without surgery. However, bile drained in one child for 20 days after exploration for delayed rupture of a subcapsular hematoma, six days after injury.[17-19,21,22]

There is still controversy about the effect of bile in the peritoneum. Earlier in this century, much work was done on the mechanism of bile peritonitis, concentrating on bile salts, combination of bacteria and bile, as well as circulatory disturbances caused by inflammatory transudation of fluid into the peritoneal cavity.[11,23,24] There is no unanimity of opinion about the cause of peritonitis when bile leaks into the peritoneal cavity. There also exists the phenomenon of bile ascites, in which patients are without acute symptoms in spite of the presence of up to eight liters of bile in the peritoneal cavity.[25]

A recent review concludes that no factors can be identified that permit prediction of the development of bile ascites as opposed to bile peritonitis.[25] The development of bilious ascites most often follows elective biliary surgery, whereas the most common cause of bile peritonitis is spontaneous perforation. Trauma was the cause of bile ascites in seven of 24 patients and of bile peritonitis in five of 34 patients. Intraperitoneal bile cultures were uniformly sterile in patients with bile ascites, while five of 11 cultures were positive in the peritonitis group. The mortality rate for treatment of bile ascites was 8%, and this increased two and a half fold in the presence of bile peritonitis.

A biliary complication is easily diagnosed in patients with overt peritonitis, either before or during surgery. The patient who manifests vague symptoms of persistent, moderate, right upper abdominal tenderness or minimal generalized tenderness, in the appropriate clinical setting up to 28 days after injury, should be evaluated for the presence of bile ascites. This is an important diagnosis to make, as all 24 patients in a series reported from this institution required surgery for definitive treatment.[25] Paracentesis with the finding of an elevated bilirubin in the aspirate can be diagnostic.

Computed tomography or ultrasound can confirm fluid in the abdomen, but hepatobiliary scanning is the least invasive method necessary to establish the presence of a biliary fistula. Historically, this consisted of measuring the radioactivity of ascitic fluid after the intravenous administration of ^{131}I rose bengal.[26] More recently, Tc-99m IDA scintigraphy has been used successfully with high sensitivity in cases of bile leak into the hepatic parenchyma,[27,28] the peritoneal cavity, and abdominal incisions following biliary surgery and spontaneous perforation.[26,29] Delayed images should be obtained in patients suspected of having a bile leak no matter how normal the study appears within the first hour. It may take time for enough activity to build up in a collection or tract to be detectable. Weissmann et al[26] note that 14 of 23 leaks would have been missed in the absence of delayed views.

There is a subset of patients with blunt trauma to the liver that may be managed without surgery provided there is hemodynamic stability, no other operative indication, and strict CT criteria are applied. This would most often involve a contained subcapsular hematoma, although capsular tears, as in the patient described here, have been observed. Patients with a palpable liver or who are receiving anticoagulant therapy have been shown to have a higher incidence of complications and should therefore be treated more aggressively.[30] Documentation of resolution of the hematoma is necessary with serial radionuclide scanning, ultrasonography, or computed tomography.[31]

CT scanning may be useful in selecting patients who will not have hemorrhagic complications resulting from nonoperative management. Unfortunately, CT scanning cannot be used to predict septic or bilious complications. Increasing abdominal fluid on serial CT or ultrasound as well as heightened clinical suspicion should lead to more liberal use of hepatobiliary scanning and early diagnosis of bile leaks.

In this patient as well as in the majority of the cases reviewed, prompt exploration in the event of a complication, with debridement, lavage, and abundant drainage, has led to a successful outcome. There are considerable time and cost burdens associated with nonoperative management, as well as a small but finite incidence of morbidity. Mortality with this type of approach has been unusual but can be greater than 20% if bile peritonitis supervenes.

REFERENCES

1. Dickerman RM, Dunn EL: Splenic, pancreatic, and hepatic injuries. *Surg Clin North Amer* 1981; 61:3-16.
2. Witte CL, Zukoski CF: A rational approach to serious blunt hepatic injury. *Am Surg* 1983; 49:446-453.
3. Moore EE: Critical decisions in the management of hepatic trauma. *Am J Surg* 1984; 148:712-716.
4. Brotman S, Oliver G, Oster-Granite ML, et al: The treatment of 179 blunt trauma-induced liver injuries in a statewide trauma center. *Am Surg* 1984; 50:603-608.
5. Carmona RH, Lim RC Jr, Clark GC: Morbidity and mortality in hepatic trauma. A 5-year study. *Am J Surg* 1982; 144:88-94.
6. Dunham CM, Militello P: Surgical management of liver trauma. *Am Surg* 1982; 48:435-439.
7. Hasselgren PO, Almersjo O, Gustavsson B, et al: Trauma to the liver during a ten-year period. With special references to morbidity and mortality after blunt trauma and stab wounds. *Acta Chir Scand* 1981; 147:387-393.
8. Levin A, Grover P, Nance FC: Surgical restraint in the management of hepatic injury: A review of Charity Hospital experience. *J Trauma* 1978; 18:399-404.
9. Madding GF: Wounds of the liver. *Surg Clin N Amer* 1958; 38:1619-1629.
10. Samlouri F, Gruenberg JC, Brown RS: Intrahepatic cutaneous biliary fistulas secondary to trauma. *Henry Ford Hospital Med J* 1980; 28:63-66.
11. Norgore M: Traumatic rupture of the gallbladder. *Ann Surg* 1946; 123:127-134.
12. Carmichael DH: Avulsion of the common bile duct by blunt trauma. *South Med J* 1980; 73:166-168.
13. Soderstrom CA, Maekawa K, DuPriest RW Jr, et al: Gallbladder injuries resulting from blunt abdominal trauma. An experience and review. *Ann Surg* 1981; 193:60-66.
14. Sparkman RS, Fogelman MJ: Wounds of the liver. Review of 100 cases. *Ann Surg* 1954; 139:690-719.
15. Madding GF: Injuries of liver. *Arch Surg* 1955; 70:748-756.
16. Shires GT: Trauma, in Schwartz SI, Shires GT, Spencer FC, et al (eds): *Principles of Surgery*, ed 4. New York, McGraw-Hill, 1984, pp 242-247.
17. Grisoni ER, Gauderer MW, Ferron J, et al: Nonoperative management of liver injuries following blunt abdominal trauma in children. *J Pediatr Surg* 1984; 19:515-518.
18. Karp MP, Cooney DR, Pros GA, et al: The nonoperative management of pediatric hepatic trauma. *J Pediatr Surg* 1983; 18:512-518.
19. Meyer AA, Crass RA, Lim RC Jr, et al: Selective nonoperative management of blunt liver injury using computed tomography. *Arch Surg* 1985; 120:550-554.
20. Moon KL Jr, Federle MP: Computed tomography in hepatic trauma. *AJR* 1983; 141:309-314.
21. Cywes S, Rode H, Millar AJ: Blunt liver trauma in children. Nonoperative management. *J Pediatr Surg* 1985; 20:14-18.
22. Giacomantonio M, Filler RM, Rich RH: Blunt hepatic trauma in children. Experience with operative and nonoperative management. *J Pediatr Surg* 1984; 19:519-522.
23. Cohn I Jr, Cotlar AM, Atik M, et al: Bile peritonitis. *Ann Surg* 1960; 152:827-835.
24. Mentzer SH: Bile peritonitis. *Arch Surg* 1934; 29:227-241.
25. Ackerman NB, Sillin LF, Suresh K: Consequences of intraperitoneal bile: Bile ascites versus bile peritonitis. *Am J Surg* 1985; 149:244-246.
26. Weissmann HS, Byan KJC, Freeman LM: Role of Tc-99m IDA scintigraphy in the evaluation of hepatobiliary trauma. *Seminars Nuc Med* 1983; 13:199-222.
27. Esensten M, Ralls PW, Colletti P, et al: Posttraumatic intrahepatic biloma: Sonographic diagnosis. *AJR* 1983; 140:303-305.
28. Stegman CJ, Levy JM, Serlin S: Case of the month No. 60—laceration of the liver. Bile leakage into the laceration on hepatobiliary scan. *Ariz Med* 1982; 39:640-653.
29. Weissmann HS, Chun KJ, Frank M, et al: Demonstration of traumatic bile leakage with cholescintigraphy and ultrasonography. *AJR* 1979; 133:843-847.
30. Geis WP, Schulz KA, Giacchino JL, et al: The fate of unruptured intrahepatic hematomas. *Surgery* 1981; 90:689-697.
31. Lambeth W, Rubin BE: Nonoperative management of intrahepatic hemorrhage and hematoma following blunt trauma. *Surg Gynecol Obstet* 1979; 148:507-511.

See also pp 534-536 and 536-537.

Modified radical neck dissections necessitated by a retropharyngeal abscess

JAY H. KLARSFELD, MD; STEVEN H. SACKS, MD; ROBERT P. GREEN, MD

Since the advent of antibiotics, the incidence of retropharyngeal abscess has decreased significantly. Only recently has the topic received attention in the literature, after many years of relative obscurity. The classic presentation in the young child is to be considered in juxtaposition to that of patients of variable ages who are now presenting due to an underlying immunocompromised status. Occasionally, a patient presents in such an advanced condition that life-threatening complications ensue and require extraordinary therapeutic measures, as in the following example.

CASE REPORT

A 14-month-old girl was transferred to

From the Department of Otolaryngology, Mount Sinai School of Medicine, New York, NY.

Address correspondence to Dr Green, Associate Clinical Professor, Mount Sinai School of Medicine, Department of Otolaryngology, 1 Gustave L. Levy Place, New York, NY 10029.

Mount Sinai Hospital with a diagnosis of retropharyngeal abscess. Two weeks prior to admission, nausea and vomiting developed, and she had a temperature of 103°F. Her pediatrician made the diagnosis of gastroenteritis and sent the child home on a diet of clear fluids. At that time, a mild degree of cervical adenopathy was noted. Three days later, she was seen with a persistent fever of 104°F; white blood cell count, 21,000/μL; erythrocyte sedimentation rate, 102 mm/h; and there were some bacteria in her urine. Her cervical adenopathy had persisted, but the remainder of her head and neck examination was normal. The child was sent home on oral trimethoprim and sulfamethoxazole for treatment of an apparent urinary tract infection.

During the next few days, the child remained febrile, the adenopathy progressed, and she became unable to tolerate anything by mouth. She was admitted to a local hospital for intravenous hydration. Following an otolaryngologic consultation and a lateral radiograph of the neck (Fig 1), the diagnosis of retropharyngeal abscess was made, and the patient was transferred to our institution.

At the time of transfer, the child was slightly lethargic, febrile (102°F), and breathing without compromise. The pharynx was erythematous and convex in appearance. Multiple, bilateral cervical nodes were palpable. The lateral neck films confirmed a bulging, grossly enlarged, retropharyngeal space. The medical history was unremarkable for recurrent infection or exposure to tuberculosis, and the patient had undergone routine immunization. She was taken to the operating room and, under general endotracheal anesthesia and via a peroral approach, a large right-sided retropharyngeal abscess was drained of frank pus, and a smaller left-sided retropharyngeal mass was opened, yielding a lesser amount of pus and containing obviously inflamed and hypertrophic lymphoid tissue. Following surgery, the endotracheal tube was removed, and the antibiotic coverage was switched from a combination of ampicillin and oxacillin to oxacillin alone, as the Gram stain and culture yielded only *Staphylococcus aureus*.

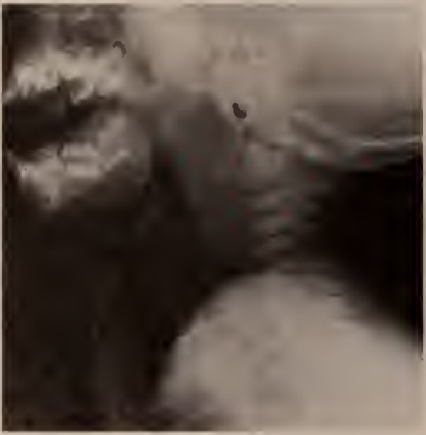


FIGURE 1. Lateral neck film demonstrating obvious retropharyngeal bulge.

During the following week, there was distinct clinical improvement, and oral intake was resumed. Daily temperature elevations in excess of 102°F, however, persisted. In addition, the patient's cervical adenopathy remained unchanged. She was returned to the operating room, where the retropharyngeal space was reopened transorally and large, necrotic nodes with scant pus were found.

On histologic analysis, the tissue was unremarkable. On the recommendation of the infectious disease consultant the antibiotic regimen was changed to cefotaxime and clindamycin. In addition, viral titers and immune response tests were performed, including immunoglobulin assays, lymphocyte counts, skin tests, phagocyte studies, and complement levels, all of which were unremarkable.

After a week of this new antibiotic regimen, it was apparent that the cervical adenopathy had not resolved and the temperature spikes persisted. A computed tomographic (CT) scan, performed with sedation and constant observation, revealed bilateral, abscessed, necrotic lymph nodes throughout both sides of the neck (Fig 2). Bilateral, modified radical neck dissections with preservation of all structures but vigorous removal of the major lymphatic chains was performed, yielding numerous frankly abscessed and necrotic nodes. The procedure consisted of raising subplatysmal flaps; dissecting and preserving the carotid sheath contents, sternocleidomastoid muscles, accessory nerves, phrenic nerves, brachial plexuses, and submaxillary glands; and removing the matted and abscessed nodal chains and infected fatty tissues, as is performed in the most conservative form of modified radical neck dissection. Suction drains were placed bilaterally and gradually advanced over six days. No growth was obtained on aerobic and anaerobic cultures. Gram stain revealed only inflammatory cells.

The child rapidly defervesced, maintained a stable airway, and was discharged after ten days. At one year's follow-up, she has no deficits and is without evidence of the above events, other than a well-camouflaged, long, horizontal cervical scar.

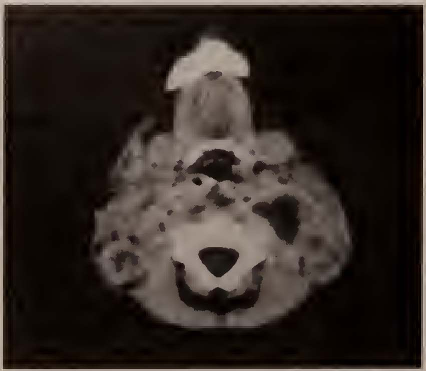


FIGURE 2. CT scan: axial section through midneck revealing multiple abscessed lymph nodes.

DISCUSSION

To our knowledge, this is the only reported case of a retropharyngeal abscess that necessitated modified radical neck dissections. This is even more remarkable considering that reports of retropharyngeal abscesses date back to Galen in the second century AD.[1] Most retropharyngeal abscesses are initiated in children by an upper respiratory tract infection of the oral and nasopharyngeal lymphoid tissue. In adults, dental caries, tuberculosis, and trauma also play a role.[2,3]

The space between the visceral fascia (which is just posterior to the superior constrictor muscle and pharyngeal mucosa) and the alar fascia (which is just anterior to the prevertebral fascia) is anatomically the retropharyngeal space. The prevertebral space is a potential space posterior to the prevertebral fascia and anterior to the vertebral bodies. The "danger space" lies between these two spaces and is demarcated anteriorly by the alar fascia and posteriorly by the prevertebral fascia.[4] Extending from the base of the skull into the mediastinum, this space is the route by which both retropharyngeal and prevertebral abscesses spread to produce mediastinitis. The convergence of all fascial layers into the carotid sheath also provides a potential pathway for extension.[5,6]

The retropharyngeal lymph nodes are located in two groups on each side of the midline and undergo atrophy as maturation ensues.[7] The commonly noted sequestration of retropharyngeal abscesses to one side of the midline is probably secondary to an attachment of the superior constrictor muscle to the prevertebral fascia and is enhanced by the lateral placement of the relevant lymph nodes.[8]

Most of the lymphatic drainage from the nose, throat, and ears is carried directly to the deep cervical nodes, and only a small percentage is directed toward the retropharyngeal spaces, even in children.[9] Efferent lymphatics from the retropharyngeal spaces drain into the internal jugular nodes of the deep cervical chain, with cross anastamosis into other nodes of the cervical lymph system, hence, the frequently occurring cervical adenopathy.

In the preantibiotic era, retropharyngeal abscess was clearly a disease of early childhood.[10] Since the advent of antibiotics and the increased prevalence of immunocompromise in patients, the ratio of adult patients to children has dramatically increased.[11]

In adults who complain of fever, dysphagia, and possibly some difficulty breathing, the diagnosis may easily be suspected and possibly confirmed on pharyngeal or indirect examination. In young children, the symptom complex will be less clear, with fever, malaise, and poor appetite being quite nonspecific. With progression, cervical adenopathy; refusal to eat, with or without drooling; and airway compromise make the localization of the problem more apparent. Direct examination will be difficult, and a bulge viewed face-on can be missed. Neck stiffness due to prevertebral muscle spasm can cause obvious confusion with meningitis in a febrile child.[12] Rupture can result in immediate asphyxiation or serious life-threatening aspiration pneumonia. Spread to the great vessels in the neck or to the mediastinum can result in vessel rupture, septic thrombosis, or mediastinitis.[13–15] As in this case, spread of infection to other nodal chains can result in suppuration and disseminated abscess formation.

The lateral neck film characteristically demonstrates marked soft tissue enlargement in the retropharyngeal region, often associated with straightening of the cervical spine. The soft tissue shadow should not exceed one and a half times the width of the fourth cervical vertebral body before the age of one, and this lessens to less than one half the width of the fourth vertebral body after the age of one.[16] A true lateral position must be achieved, and it must be kept in mind that expiration in an infant can cause a thickened appearance imitating the presence of a retropharyngeal process.[17] CT scanning enhances one's diagnostic capability with regard to the primary abscess as well as associated complications, but with an infant who may require sedation and in the setting of a compromised airway, CT scanning should be used judiciously.[18] In this case, the CT scan was invaluable in fully delineating the extent of suppuration throughout the neck and in indicating the need for modified neck dissection.

After endotrachael intubation, in the infant or child, transoral drainage via a direct vertical incision is easily accomplished and is usually adequate. With inferior progression, or in the adult, where the extent of the process is often less well defined, an external approach is preferable. Cultures for aerobic and anaerobic organisms are mandatory. Diverse organisms, often of a resistant nature, require broad spectrum antibiotic coverage at least until culture results are available. At a minimum, coverage of the usual aerobic and anaerobic upper respiratory pathogens is essential.[11]

The case presented here is obviously quite unusual with regard to the massive cervical suppuration that required such extensive surgery. This report should not be interpreted as advocating modified radical neck dissections unless conventional therapy has clearly failed, as there is obvious risk to cranial nerves and major vascular structures, heightened by the disturbed anatomy and inflammatory response. Simple drainage of just the grossly abscessed nodes was thought to be inadequate, as we had observed mildly infected nodes progress to abscesses in spite of drainage of the primary site of infection and ongoing intravenous antibiotic therapy. Suppuration of cervical nodes requiring external drainage has been reported in conjunction with a retropharyngeal abscess, but not of this magnitude.[12] The underlying reasons for this progression are not clear. The child was, based on all of our sophisticated testing, immunologically normal, and the organisms involved are aggressive but common pathogens. In spite of adequate drainage of the primary retropharyngeal abscess and vigorous antibiotic therapy, expanding cervical sepsis proved difficult to contain. The amount of time from the true onset of the illness to the initiation of aggressive and directed management is obscure but clearly is an important consideration. How this time span compares to that which occurs in other cases of retropharyngeal abscess is difficult to determine.

Finally, it should be remembered that all swelling in the retropharyngeal space may not be caused by infection. Neoplasms such as cystic hygroma, hemangioma, lymphoma, and a variety of other tumors can occupy this space.[19] Direct trauma, in addition to possibly initiating an infection, can also cause an isolated hematoma. Such conditions should be readily distinguishable from a frank retropharyngeal abscess.

REFERENCES

1. Davidson M: Abscesses of retropharyngeal spaces in adults. *Laryngoscope* 1949; 59:1146–1170.
2. Paparella MM, Shumrick DA: *Otolaryngology*, ed 2. Philadelphia, WB Saunders & Co, 1980, vol 3.
3. Husaru AD, Nedzelski JM: Retropharyngeal abscess and upper airway obstruction. *J Otolaryngol* 1979; 8:443–447.
4. Grodinsky M: Retropharyngeal and lateral pharyngeal abscesses; anatomic and clinical study. *Ann Surg* 1939; 110:177–199.
5. Frank I: Retropharyngeal abscess. *JAMA* 1921; 77:517–522.
6. Wong Y, Nootry G: Retropharyngeal space—a review of anatomy, pathology and clinical presentation. *J Otolaryngol* 1978; 7:528–536.
7. Seid AB, Dunbar JS, Cotton RT: Retropharyngeal abscesses in children revisited. *Laryngoscope* 1979; 89:1717–1724.
8. Johnson JT, Tucker HM: Recognizing and treating deep neck infection. *Postgrad Med* 1976; 59:95–100.
9. *Gray's Anatomy*, ed 35 (British). Philadelphia, WB Saunders & Co, 1973.
10. Brown J: Acute retropharyngeal abscess in children. *Laryngoscope* 1919; 29:9–12.
11. Barratt GE, Koopermann CF Jr, Coulthard SW: Retropharyngeal abscess—a ten-year experience. *Laryngoscope* 1984; 94:455–463.
12. Richards L: Retropharyngeal abscess. *N Engl J Med* 1936; 215:1120–1130.
13. Ramllo J, Harris U, White H: Empyema as a complication of retropharyngeal and neck abscess in children. *Ped Radiol* 1978; 126:843–846.
14. Mosher HP: Deep cervical abscess and thrombosis of internal jugular vein. *Laryngoscope* 1920; 30:365–375.
15. Wishart D: Retro-pharyngeal abscess; and erosion of internal carotid artery with pathological specimen. *Can Med Assoc J* 1923; 13:635–641.
16. Brenner GH: Variations in the depth of the cervical prevertebral tissue in normal infants studied by cinefluorography. *Am J Roentgenol* 1964; 91:573–577.
17. Gwinn JL, Lee FA, Canby JP: Retropharyngeal abscess. *Am J Dis Child* 1971; 121:237–238.
18. Endicott JN, Nelson RJ, Saraceno CA: Diagnosis and management decisions in infections of the deep fascial spaces of the head and neck utilizing computerized tomography. *Laryngoscope* 1982; 92:630–633.
19. McCook TA, Felman AH: Retropharyngeal masses in infants and young children. *Am J Dis Child* 1979; 133:41–43.

Fulminant lymphocyte-depleted Hodgkin disease in a homosexual man with HIV infection

DONALD COLBOURN, MD; HARRY STASZEWSKI, MD; STEVEN GOLDENBERG, MD;
VIRGINIA DONOVAN, MD; FRANK RAIO, MD

Recent reports have suggested a possible association of Hodgkin disease in patients at risk for acquired immunodeficiency syndrome (AIDS).[1-9] Usually, the pathologic subtypes of Hodgkin disease reported have been the mixed cellularity or nodular sclerosis types. In addition, most of the patients presented did not have human immunodeficiency virus (HIV) infections that had been confirmed with serologic or viral studies. We report on a patient with serologic evidence of HIV infection who had fulminant lymphocyte-depleted Hodgkin disease, which proved fatal.

CASE REPORT

A 43-year-old homosexual man was admitted to the Winthrop-University Hospital emergency room with a four-day history of fever, jaundice, and increasing mental confusion. He had been well until several months prior to admission, when he complained of mild anorexia and weight loss. No lymphadenopathy or organomegaly was detected at that time. T cell subset analysis at that time revealed an OKT4:OKT8 ratio of 1.06 (normal range, 1.2–3.8).

On admission, physical examination findings included a temperature of 103.2°F and a pulse rate of 100/min. The patient appeared acutely ill and was deeply jaundiced. Disorientation was present, but the patient remained alert. There was posterior cervical, bilateral supraclavicular, and axillary adenopathy, but no individual node was greater than 1 cm in diameter. Ascites and massive hepatosplenomegaly were demonstrable. Asterixis was present, but no focal neurologic abnormalities were detected.

Laboratory studies were remarkable for a severe pancytopenia. The blood urea nitrogen level was 92 mg/dL, and the creatinine level was 4.1 mg/dL. Total bilirubin was elevated at 11.8 g/dL, and hepatic enzymes were elevated to three times normal values.

A computerized axial tomographic (CAT) study of the abdomen revealed the presence of ascites and massive hepatosplenomegaly. There was no evidence of biliary tract obstruction or focal defects. The kidneys appeared normal, and there was a distinct absence of retroperitoneal adenopathy. A bone marrow aspiration and biopsy revealed a hypercellular marrow with megakaryocytic and histiocytic hyperplasia with occasional erythrophagocytosis. Stains and cultures for mycobacteria and fungi were negative. All stains and cultures of blood, urine, stool, and sputum for significant bacterial, fungal, mycobacterial, or protozoan organisms were negative. Antibody titers for Epstein-Barr virus, cytomegalovirus, and hepatitis A and B were not supportive of active infection. ELISA assay for HIV infection was positive in high titer and was confirmed by Western blot study.

The patient's condition deteriorated rapidly, with the development of renal and hepatic failure as well as severe metabolic acidosis. The bilirubin level continued to increase beyond 50 mg/dL. A diagnosis of a lymphoproliferative disorder, possibly malignant histiocytosis, was made. Heroic therapy with cyclophosphamide, vincristine, and prednisolone was administered intravenously. Eighteen hours later, a cervical lymph node biopsy was obtained (Fig 1). Progressive organ failure continued despite hemodialysis and blood product support, and the patient died nine days after admission. Postmortem examination demonstrated lymphocyte-depleted Hodgkin disease involving the liver, spleen, and bone marrow as well as the perisplenic and peripancreatic lymph nodes. Analysis of lymphocyte surface markers in lymph node sections by the immunoperoxidase technique demonstrated a lack of significant positivity, consistent with the diagnosis of Hodgkin disease.

DISCUSSION

It is apparent from recent reports that Hodgkin disease is yet another neoplastic disorder that occurs in patients at risk for AIDS.[1-9] Unusual presentations of Hodgkin disease with extranodal involvement and advanced stage at diagnosis have been characteristic of these reports. Most reports have been of the mixed cellularity subtype, though several patients with features of both mixed cellularity and lymphocyte-depleted Hodgkin disease have been presented.[8] While the occurrence of opportunistic infections characteristic of AIDS has suggested HIV infection in these patients, few cases with actual confirmation by serologic or viral studies have actually been published. It remains possible that some of these cases are the coincidental occurrence of Hodgkin disease in homosexual men.

Though the course of Hodgkin dis-

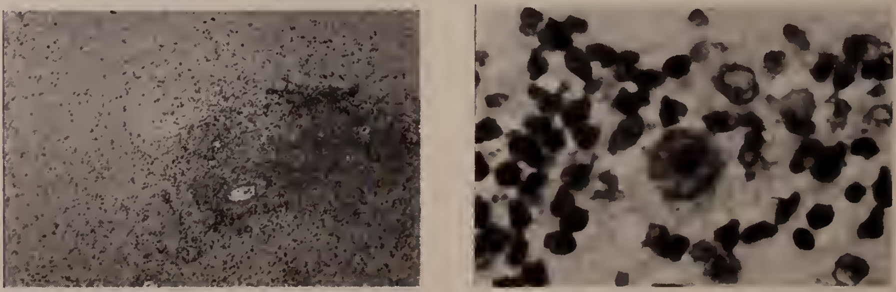


FIGURE 1. **Left,** Antemortem cervical lymph node biopsy showing marked lymphoid depletion and fibrosis (hematoxylin-eosin stain; original magnification × 40); **Right,** At higher magnification, a mononuclear Hodgkin cell admixed with benign lymphocytes (hematoxylin-eosin stain; original magnification × 1,000).

From the Oncology-Hematology Division, Department of Medicine (Drs Colbourn and Staszewski) and the Department of Pathology (Dr Donovan), Winthrop-University Hospital, Mineola, NY, and the Department of Medicine, Health Sciences Center, SUNY at Stony Brook, Stony Brook, NY (Drs Colbourn, Staszewski, Goldenberg, and Raio).

Address correspondence to Dr Staszewski, Director, Hematology Service, Oncology-Hematology Division, Winthrop-University Hospital, Winthrop-University Hospital Professional Building, 222 Station Plaza North, Mineola, NY 11501.

ease has been reported to be aggressive in most patients who are also at risk for AIDS, the disease usually responds to therapy. In most cases, the greatest morbidity and mortality have resulted from opportunistic infections or Kaposi's sarcoma, rather than from progressive Hodgkin disease. The role of the additional immunosuppression that inevitably occurs in the treatment of advanced Hodgkin disease is unclear in terms of the risk of activating opportunistic pathogens.

At least one group has cautioned against the use of steroids or combined modality therapy in male homosexuals with Hodgkin disease because of the sensitivity of the disease to treatment and the subsequent occurrence of overwhelming opportunistic infections in these patients.[2] Unfortunately, the fulminant nature of Hodgkin disease in patients such as the one described in this report may make such a course of action impossible to follow.

The present case has several unique features. No opportunistic infections either prior to the appearance of Hodgkin disease or during the hospital course appear to have contributed to the patient's demise. Fatal, fulminant lymphocyte-depleted Hodgkin disease presenting with pancytopenia and massive splenomegaly is an unusual occurrence in the more recent reports of this histologic subtype.[10] Unlike previous patients at risk for AIDS who had aggressive Hodgkin disease, this patient probably did not survive his malignancy long enough to develop opportunistic infectious complications.

Acknowledgment. The authors thank Dr Elaine S. Jaffe for reviewing the biopsy and autopsy reports.

REFERENCES

1. Unger PD, Strauchen JA: Hodgkin's disease in AIDS complex patients. Report of four cases and tissue immunologic marker studies. *Cancer* 1986; 58:821-825.
2. Baer DM, Anderson ET, Wilkinson LS: Acquired immune deficiency syndrome in homosexual men with Hodgkin's disease. Three case reports. *Am J Med* 1986; 80:738-740.
3. Schoeppel SL, Hoppe RT, Dorfman RF, et al: Hodgkin's disease in homosexual men with generalized lymphadenopathy. *Ann Intern Med* 1985; 102:68-70.
4. Scheib RG, Siegel RS: Atypical Hodgkin's disease and the acquired immunodeficiency syndrome [letter]. *Ann Intern Med* 1985; 102:554.
5. Ioachim HL, Cooper MC, Hellman GC: Hodgkin's disease and the acquired immunodeficiency syndrome [letter]. *Ann Intern Med* 1984; 101:876-877.
6. Robert NJ, Schneiderman H: Hodgkin's disease and the acquired immunodeficiency syndrome [letter]. *Ann Intern Med* 1984; 101:142-143.
7. Subar M, Chamulak G, Dalla-Favera R, et al: Clinical and pathological characteristics of AIDS associated Hodgkin's disease. *Blood* 1986; 68(suppl): 135a.
8. Prior E, Goldberg AF, Conjalka MS, et al: Hodgkin's disease in homosexual men. An AIDS-related phenomenon? *Am J Med* 1986; 81:1085-1088.
9. Mitsuyasu RT, Colman MF, Sun NC: Simultaneous occurrence of Hodgkin's disease and Kaposi's sarcoma in a patient with the acquired immune deficiency syndrome. *Am J Med* 1986; 80:954-958.
10. Kant JA, Hubbard SM, Longo DL, et al: The pathologic and clinical heterogeneity of lymphocyte-depleted Hodgkin's disease. *J Clin Oncol* 1986; 4:284-294.

Hypersensitivity reaction to anticonvulsants following head injury

JAMES M. HASSETT, JR, MD; THOMAS DOOLITTLE, MD; MICHAEL MOLLOY, BS; DAVID LALKA, PhD

Trauma is a complex state of bodily injury associated with physiologic changes that have the potential to alter hepatic drug clearance mechanisms. Responses to trauma include alteration in hepatic blood flow, reduction in hepatic microsomal activity, reduction in hepatic excretion processes, and changes in protein binding.[1,2] Moderate to severe brain injury commonly occurs in a variety of trauma mechanisms. Anticonvulsant prophylaxis after concussive head injury is indicated in patients who experience a single epileptic event that is not temporally associated with impact, or in patients with a significant risk of seizure. A patient's response to medication can be affected by the pre-existing, but clinically quiescent, hypersensitivity states. In this complex post-injury state, a patient's reaction to the most commonly used agents, phenytoin and phenobarbital, can have serious consequences. We describe a patient who experienced a hypersensitivity reaction to both agents.

CASE REPORT

A 17-year-old man was involved in an accident while riding a bicycle. The patient was transported to a regional hospital, where he was unresponsive to all but very painful stimuli. After the initial assessment, he was transferred to the Trauma Service of the Erie County Medical Center (ECMC). The patient experienced three episodes of emesis during transport. The initial examination revealed a Glascow Coma Scale of 12 with normal vital signs. Physical examination was essentially normal, except for a 4-cm laceration over the left temple, an abrasion of the left knee, a semiconscious state, and slowly reactive pupillary responses. Radiologic evaluations of the skull, cervical spine, chest, and abdomen were unremarkable. A computed tomographic (CT) scan of the brain demonstrated a right frontal contusion. The patient received both dexamethasone and phenytoin. The CT scan was repeated on the fourth day of hospitalization and again demonstrated the right frontal contusion. After an uneventful hospital stay, the patient was discharged on the sixth hospital day on continued therapy with phenytoin, 300 mg orally at bedtime.

On the eighth post-injury day, the patient returned with a 24-hour history of a maculopapular pruritic rash that involved his entire body. Physical examination revealed a temperature of 103°F (oral); blood pressure, 120/60 mm Hg; heart rate, 80/min; respiratory rate, 16/min, with diffuse, nonpitting edema of the extremities. He was readmitted, the phenytoin was discontinued, and he was given diphenylhydramine, 25 mg intramuscularly. The serum phenytoin level was 16.5 mg/dL. He was started on phenobarbital, 60 mg orally twice a day. After significant symptomatic improvement, he was discharged on the fourth hospital day (post-injury day 12).

On the 19th post-injury day, the patient returned to the hospital with a generalized maculopapular eruption that involved his

---

From the Departments of Surgery (Dr Hassett), Neurosurgery (Dr Doolittle), Pharmacy (Mr Molloy), and Pharmaceutics (Dr Lalka), State University of New York at Buffalo School of Medicine.

Address correspondence to Dr Hassett, Department of Surgery, Erie County Medical Center, 462 Grider St, Buffalo, NY 14215.

entire body. The new eruption was more severe than the previous one and occurred acutely on the day prior to admission. Physical evaluation revealed a temperature of 102.4°F; blood pressure, 100/60 mm Hg; heart rate, 82/min; respiratory rate 16/min, with a diffuse morbilliform rash. Dermatologic consultation confirmed a "drug eruption rash." The patient was started on hydroxyzine, 25 mg orally four times a day, and 0.1% triamcinolone acetonide, three times a day. The phenobarbital was discontinued, and the patient was discharged at the request of his family on the second hospital day. The cutaneous eruptions eventually cleared with symptomatic treatment. No further anticonvulsant therapy was administered, and the patient remained symptom free.

DISCUSSION

In a patient who has had a concussive head injury, the occurrence of a seizure further depresses the level of consciousness, increases the intracranial pressure, and may cause unnecessary death. The precise indications for and the dosage or duration of antiseizure chemotherapy are poorly defined.[3] The question of whether anticonvulsant medication should be given for prophylaxis against early, post-traumatic seizures is unanswered.[4] Prophylactic administration of phenytoin reduces the rate of late, post-traumatic seizures[4,5] and may decrease the frequency of early seizures.[4–6] Phenytoin and phenobarbital are the most frequently utilized agents.[7] Both are almost completely absorbed after oral administration in individuals with normal gastrointestinal physiologic responses.

From 85% to 90% of phenytoin is bound to serum albumin.[7] It is metabolized in the liver to p-(hydroxyphenyl-phenyl)-hydantoin (HPPH) via a pathway well described by Michaelis-Menten kinetics.[8] This metabolite has virtually no pharmacologic activity. It is rapidly conjugated with glucuronic acid and excreted in the urine[7,9,10] or through the gastrointestinal tract. Serum levels will peak four to eight hours after ingestion.

Phenytoin is oxidized by cytochrome P-450 mono-oxygenases to several oxidized products.[11,12] Arene oxides are reactive, electrophilic compounds, which are intermediates to such oxidative reactions. If they are not detoxified, they can covalently bind to cell macromolecules, resulting in cell death or mutation. They can also act as haptens, leading to secondary immune phenomena.

Ten percent of the patients who receive phenytoin will develop a rash that requires a change to another agent.[13] The eruption is usually accompanied by fever and occurs on the ninth or tenth day of treatment. The generalized skin reaction seen after phenytoin administration may be due to a hypersensitivity reaction[14] or a high body burden of phenytoin.[15]

Among patients in whom the rash develops, there is a lower capacity for metabolizing the phenytoin to p-hydroxyphenytoin without a change in the plasma protein binding. The appearance of the rash might be due to a slow rate of para-hydroxylation of the phenytoin, which forces the metabolic process toward an epoxide pathway.

Severe idiosyncratic reaction to phenytoin was first described as "phenytoin hypersensitivity" in 1936.[14] It can include lymphadenopathy, exfoliative dermatitis, eosinophilia, and hepatic necrosis.[13,16] The reaction appears to be a hypersensitivity that may also include hemolytic anemia, leukopenia, thrombocytopenia, diffuse vasculitis, splenomegaly, and severe hepatorenal failure.[17,18]

The frequency of phenytoin hypersensitivity is unknown.[14] It generally involves adolescents or young adults.[14,19] It has an antigenic component, with one of the metabolites acting as a hapten.[20,21] It would appear that the cytotoxicity of the phenytoin hypersensitivity reaction may be due to an inherent difficulty with epoxide hydrolase.[11,12] The genetic pattern of a possibly defective epoxide hydrolase pathway detoxification mechanism suggests an autosomal, inherited trait.[11]

The metabolism of phenobarbital is predominantly by hepatic microsomal enzymes.[7] The primary metabolite is p-hydroxyphenylbarbital, which has no known anticonvulsant properties and is excreted in the urine. An adverse reaction to phenobarbital occurs with a 4% maximum reported incidence of cutaneous reaction.[22] Phenobarbital administration is occasionally associated with a syndrome of hyperpyrexia, delirium, exfoliative dermatitis, and parenchymatous organ damage, which is frequently fatal.[23] The anecdotal clinical reports are remarkably similar to the phenytoin hypersensitivity reaction.[23,24] In vitro lymphocyte stimulation studies with anticonvulsant drugs suggest a cell-mediated hypersensitivity reaction to phenobarbital similar to that seen with phenytoin.[22]

In the majority of hypersensitivity reactions in which both phenytoin and phenobarbital have been implicated together, the interrelationships have generally been confused by the simultaneous administration of the two agents.[19,25–28] The structural similarities between phenytoin and phenobarbital have been considered to be the cause of the cross reaction.[22] In the patient presented here, there was a clear separation of the administration of the two drugs, with symptomatic improvement after discontinuation of the phenytoin and return of symptoms after administration of the phenobarbital.

A hypersensitivity reaction is independent of the traumatic event. However, traumatic injury has the potential to significantly alter drug pharmacokinetics through a variety of mechanisms. Studies in a rat trauma model indicate that hepatic microsomal activity (in vitro) is depressed during the post-traumatic period.[2] The normal hepatic response to injury results in an increase in the normal binding protein alpha-1-acid glycoprotein.[1,29] These alterations may increase the potential for idiosyncratic or hypersensitivity reactions. Environmental alteration may not be the only mechanism of altering pharmacogenetic conditions.[30] The trauma-induced alterations in hepatic metabolism, without a clinical change in liver function, can result in the creation of hypersensitivity-inducing ligands. This case clearly establishes the ability of both phenytoin and phenobarbital to induce similar hypersensitivity responses when administered separately to the same patient.

REFERENCES

1. Edwards DJ, Lalka D, Cerra F, et al: Alpha1-acid glycoprotein concentration and protein binding in trauma. *Clin Pharmacol Ther* 1982; 31:62–67.
2. Slaughter R, Hassett JM: Hepatic drug clearance following traumatic injury. *Drug Intell Clin Pharm* 1985; 19:799–806.
3. Rapport RL 2d, Penry JK: A survey of attitudes toward the pharmacologic prophylaxis of posttraumatic epilepsy. *J Neurosurg* 1973; 38:159–166.
4. Wyler AR, Ray MW: Anticonvulsant prophylaxis against posttraumatic seizures. *Contemp Neurosurg* 1985; 7:1–6.
5. Young B, Rapp RP, Norton JA, et al: Failure of prophylactically administered phenytoin to prevent early posttraumatic seizures. *J Neurosurg* 1983; 58:231–235.
6. Young B, Rapp RP, Norton JA, et al: Failure of prophylactically administered phenytoin to prevent late posttraumatic seizures. *J Neurosurg* 1983; 58:236–241.
7. Berman PH: Management of seizure disorders with anticonvulsant drugs: Current concepts. *Pediatr Clin North Am* 1976; 23:443–459.
8. Ludden TM, Allen JP, Valutsky WA, et al: Individualization of phenytoin dosage regimens. *Clin Pharmacol Ther* 1977; 21:287–293.
9. Olanow CW, Finn AL: Phenytoin: Pharmacokinetics and clinical therapeutics. *Neurosurg* 1981; 8:112–117.
10. Plaa GL: Acute toxicity of antiepileptic drugs. *Epilepsia* 1975; 16:183–191.
11. Spielberg SP, Gordon GB, Blake DA, et al:

Predisposition to phenytoin hepatoxicity assessed in vitro. *N Engl J Med* 1981; 305:722-727.

12. Spielberg SP, Gordon GB, Blake DA, et al: Anticonvulsant toxicity in vitro: Possible role of arene oxides. *J Pharmacol Exp Ther* 1981; 217:386-389.

13. Almeyda J, Levantine A: Drug reactions. XX. Cutaneous reactions to anticonvulsants. *Br J Dermatol* 1972; 87:646-649.

14. Haruda F: Phenytoin hypersensitivity: 38 cases. *Neurology* 1979; 29:1480-1485.

15. Wilson JT, Höjer B, Tomson G, et al: High incidence of a concentration-dependent skin reaction in children treated with phenytoin. *Br Med J* 1978; 1:1583-1586.

16. Van Oss CJ, Bronson PM, Border JR: Changes in the serum alpha glycoprotein distribution in trauma patients. *J Trauma* 1975; 15:451-455.

17. Hyman LR, Ballow M, Knieser MR: Diphenylhydantoin interstitial nephritis. Roles of cellular and humoral immunologic injury. *J Pediatr* 1978; 92:915-920.

18. Josephs SH, Rothman SJ, Buckley RH: Phenytoin hypersensitivity. *J Allergy Clin Immunol* 1980; 66:166-172.

19. Parker WA, Shearer CA: Phenytoin hepatotoxicity: A case report and review. *Neurology* 1979; 29:175-178.

20. Holland P, Mauer AM: Diphenylhydantoin-induced hypersensitivity. *J Pediatr* 1963; 66:322-332.

21. Robinson DS, MacDonald MG, et al: Sodium diphenylhydantoin reaction with evidence of circulating antibodies. *JAMA* 1965; 192:171-172.

22. Knutsen AP, Anderson J, Satayaviboon S, et al: Immunologic aspects of phenobarbital hypersensitivity. *J Pediatr* 1984; 105:558-563.

23. McGeachy TE, Bloomer WE: Phenobarbital sensitivity syndrome. *Am J Med* 1953; 14:600-604.

24. Shapiro PA, Antonioli DA, Peppercorn MA: Barbiturate-induced submassive hepatic necrosis. Report of a case and review of the literature. *Am J Gastroenterol* 1980; 74:270-273.

25. Crawford SE, Jones CK: Fatal liver necrosis and diphenylhydantoin sensitivity. *Pediatrics* 1962; 30:595-600.

26. Dubois EL: Toxic hepatic necrosis associated with taking dilantin, tridione and phenobarbital. *Am J Clin Path* 1950; 80:153-158.

27. Kahn HD, Faguet GB, Agee JF, et al: Drug-induced liver injury. In vitro demonstration of hypersensitivity to both phenytoin and phenobarbital. *Arch Intern Med* 1984; 144:1677-1679.

28. Pollack MA, Burk PG, Nathanson G: Mucocutaneous eruptions due to antiepileptic drug therapy in children. *Ann Neurol* 1979; 5:262-267.

29. Ting S, Dunsky EH: Diphenylhydantoin-induced hepatitis. *Ann Allergy* 1982; 48:331-332.

30. Vesell ES: Genetic host factors: Determinants of drug response [editorial]. *N Engl J Med* 1985; 313:261-262.

FROM THE LIBRARY

THE DIAGNOSIS AND TREATMENT OF LACERATIONS OF THE LIVER

Dr. G. Dencks gives the details of seven cases which were brought to the hospital at Friedrichshain, three of which recovered. In these three cases one resulted from a gunshot wound, one from a kick by a horse, while the third patient was run over. Of the four fatal cases, in two there were other severe accompanying lacerations. In the four fatal cases, the ruptures were subcutaneous, two having resulted from falls, and two having been crushed under wheels.

The conclusions reached from the observation of these cases do not very materially differ from those to be drawn from the study of a larger group of similar injuries. Thus it was observed that the right lobe is the part of the liver most often injured in subcutaneous ruptures, and that the tears are usually on the convex surface. Excluding the case of gunshot wound the left lobe was injured but once. In no case were isolated tears found on the concave surface.

As to the signs, tension of the abdomen and abdominal tendencies were constant manifestations. Neither was absent in a single case. The tension and rigidity were always general and never limited to one side. Tenderness, though likewise general, was found to be more marked over the right hypochondrium or epigastrium. In three cases there was neither hiccough eructation of gas nor vomiting.

These cases were all extremely severe, and in each large quantities of blood were found in the abdominal cavity. Dullness over the dependent parts of the abdomen was observed four times, and twice there was pain radiating towards the right shoulder blade.

The pulse could not be depended upon to indicate the degree of internal hemorrhage. Occasionally, in the most rapidly fatal cases, it was found to be of relatively good character.

The only therapeutic measure to be considered is laparotomy. In two cases the lacerations were sutured with catgut carried deeply into the liver substance. In the other cases, and this group includes the three which recovered, the bleeding was controlled by tampon. No ill effect ever attended this method of treatment. In one case the gauze strips were serviceable in draining an abscess which had formed between liver and diaphragm. The gauze tampon is to be regarded as the best means of controlling liver hemorrhage. It is the most reliable and at the same time the quickest of application. Suture is to be reserved for such wounds as have smooth, clean cut edges, easy of apposition.

Of greatest importance in cases of laceration of the liver is prompt diagnosis followed by immediate laparotomy. Even when the diagnosis cannot be absolutely made, if there is good ground to fear that this injury has occurred, exploratory laparotomy is indicated.—*Deutsche Zeitschrift f. Chirurgie*, 82 Band 4-6 Heft Mai, 1906.

(*NY State J Med* 1906; 6:379-380)

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Responses to "Death of a dentist"

TO THE EDITOR: Dr Bluestone's "Death of a dentist"[1] is a fine and well-written essay. We have lots of good mechanics who are dentists and physicians who are not doctors. They take care of diseases, not people with diseases. Of course, there are a few in each generation who, for whatever reasons, are sensitive to the needs of people. The applicants I have interviewed have often been broadly read and educated, not schooled, and my colleagues have rated them low "because their interests are too broad"! But I have persisted in swimming upstream at our meetings, and will die fighting for the principle of taking care of people with diseases.

MAURICE TULIN, MD
39 Hicks Lane
Great Neck, NY 11024

[1] Bluestone NR: Death of a dentist. *NY State J Med* 1987; 87:356–357.

TO THE EDITOR: What can I say? Dr Bluestone's "Death of a dentist"[1] brought tears to my eyes, too.

ALFRED P. INGEGNO, MD
27 Eighth Ave
Brooklyn, NY 11217

[1] Bluestone NR: Death of a dentist. *NY State J Med* 1987; 87:356–357.

TO THE EDITOR: "Death of a dentist"[1] is a masterpiece. I am enriched by having read it, and I thank Dr Bluestone for the time and effort that must have gone into writing it.

CHARLES S. HIRSCH, MD
Chief Medical Examiner
Suffolk County
Suffolk County Office Building
Hauppauge, NY 11788

[1] Bluestone NR: Death of a dentist. *NY State J Med* 1987; 87:356–357.

TO THE EDITOR: I was touched by Dr Bluestone's[1] beautiful, warm story of the death of a dentist in the June issue of the *New York State Journal of Medicine*. It is a loving tribute to one who served her and his other patients with his skill and himself.

CHARLES A. PERERA, MD
Kendal at Longwood #17
Kennett Square, PA 19348

[1] Bluestone NR: Death of a dentist. *NY State J Med* 1987; 87:356–357.

TO THE EDITOR: Dentists like Dr Bluestone's pal Jack are only rarely found. When I left dentistry for plastic surgery, it was because I felt I might not be appreciated. Dr Bluestone's essay[1] made me realize that, had I stayed, I might have been lucky enough to find a patient like her.

BARRY M. ZIDE, DMD, MD
New York University
Medical Center
333 East 34th St
New York, NY 10016

[1] Bluestone NR: Death of a dentist. *NY State J Med* 1987; 87:356–357.

Moses Maimonides and air pollution

TO THE EDITOR: I enjoyed the review article by McCunney[1] and the accompanying editorial by Hinkle[2] on indoor air pollution. The medieval giant of Judiasm, Moses Maimonides (1135–1204), in his *Treatise on Asthma*,[3] one of his ten authentic medical works,[4] describes air pollution. The last chapter of the *Treatise on Asthma* deals with concise admonitions and aphorisms which Maimonides considered "useful to any man desirous of preserving his health and administering to the sick." The chapter begins as follows: "The first thing to consider . . . is the provision of fresh air, clean water and a healthy diet." Fresh air is described in some detail:

> . . . City air is stagnant, turbid and thick, the natural result of its big buildings, narrow streets, the refuse of its inhabitants . . . one should at least choose for a residence a wide-open site . . . living quarters are best located on an upper floor . . . and ample sunshine . . . Toilets should be located as far as possible from living rooms. The air should be kept dry at all times by sweet scents, fumigation and drying agents. The concern for clean air is the foremost rule in preserving the health of one's body and soul.

Let our air pollution control experts take cognizance of Maimonides' prophetic statements made nearly 800 years ago.

FRED ROSNER, MD
Department of Medicine
Queens Hospital Center Affiliation
of the Long Island Jewish
Medical Center
Jamaica, NY 11432

[1] McCunney RJ: The role of building construction and ventilation in indoor air pollution: Review of a recurring problem. *NY State J Med* 1987; 87:203–209.
[2] Hinkle LE Jr: Indoor air pollution. *NY State J Med* 1987; 87:196–197.
[3] Rosner F: Moses Maimonides' *Treatise on Asthma. J Asthma* 1984; 21:119–129.
[4] Rosner F: The medical writings of Moses Maimonides. *Clio Medica* 1981; 16:1–11.

Rare *Streptococcus pneumoniae* soft tissue abscess

TO THE EDITOR: *Streptococcus pneumoniae* usually produces infection in the respiratory tract. Major secondary complications are principally central nervous system infections, endocarditis, and osteomyelitis. Soft tissue abscess without evidence of systemic or contiguous infection is a rare event. The patient presented here demonstrates a chronic, solitary abscess in the soft tissue space of the buttock and thigh, of undefined origin.

Case Report. A 50-year-old man was admitted to Harlem Hospital Center on March 1, 1982, with a history of inability to walk since December 1981. The onset of this disorder had been sudden. After a visit to the emergency room on December 12, 1981, he was sent home with a diagnosis of muscle spasms.

In February 1982 swelling of the left posterior flank developed. Pus was periodically drained on an outpatient basis. There was no previous history of trauma, fever, diabetes, sickle cell disease, hypertension, tuberculosis, or drug or alcohol abuse. The patient worked as a security guard and had not recently travelled.

The physical examination was normal, except for a fluctuant abscess extending from the level of the third lumbar vertebra to the sacral area in the left flank. There was edema in the right thigh, with diminished power in the iliopsoas, hamstring, and extensor hallucis longus muscles. There was a surgical scar from a prior partial colectomy and anastomosis.

Incision and drainage of the left flank yielded 1,000 cc of pus on the fifth day of admission. A Gram stain revealed only neutrophils and no organisms. Some improvement in leg movement and function of the gluteal and thigh muscles was noted with the initiation of cefoxitin, 1 g intravenously every four hours, and oxacillin, 2 g intravenously every four hours. Spinal (lumbosacral) radiographs and a bone scan were normal and showed no evidence of osteomyelitis. A computed tomographic (CT) scan showed a large, soft tissue abscess that dissected along tissue planes overlying the paraspinal muscle groups, gluteal, and upper thigh muscles, with some extension into the obturator canal. Two thousand cc of pus was drained from this abscess on the sixth hospital day. All cultures grew *Streptococcus pneumoniae* type 15. Therapy was promptly changed to penicillin, two million units every four hours, and the patient steadily recovered, with no residual neurologic deficit.

Discussion. This patient had an extensive soft tissue abscess which grew a pure culture of *S pneumoniae* type 15. Despite clear evidence of motor weakness at the time of admission, no signs of epidural or subdural infection or of vertebral osteomyelitis could be found. The most likely explanation for the neurologic findings would appear to be compression of muscle groups by the abscess mass and, perhaps, compression of nerves of the obturator canal.

Soft tissue abscess caused by *S pneumoniae* is rare. The two cases reported previously from this institution occurred in drug addicts.[1] It was presumed that these individuals had injected the pneumococcus through contaminated drug paraphernalia. This patient was clearly not a drug addict and had not received any injections. The pathogenesis of the lesion is therefore obscure. In the absence of evidence of bone involvement, it must be assumed that a temporarily remote bacteremia had evolved into a chronic, indolent, deep tissue infection.

LAKSHMANAN SATHYAVAGISWARAN, MD
P.C.T. DICKINSON, MD
JAY DOBKIN, MD
Division of Infectious Diseases
Harlem Hospital Center
of Columbia University
506 Lenox Ave
New York, NY 10037

1. Lewis RJ, Richmond AS, McGrory JP: *Diplococcus pneumoniae* cellulitis in drug addicts. *JAMA* 1975; 232:54–55.

Isolated endometriosis in a Bartholin gland

TO THE EDITOR: Endometriosis of the vulva and perineum has been reported.[1-3] However, endometriosis of the Bartholin gland with recurrent cystic swelling of the gland has not been reported. We describe a patient with endometriosis in the area of the Bartholin gland, in whom exploratory laparotomy for a pelvic mass revealed leiomyomata uterii and no evidence of pelvic endometriosis.

Case Report. A 29-year-old woman, para 0-0-4-0, underwent excision of a cyst in the left Bartholin gland on April 8, 1986; prior symptoms included current swelling of the gland. Her history is pertinent for incision and drainage of the left Bartholin gland in 1978 and 1982, to reduce swelling. The swelling appeared to be more prominent during menses. She had had episodes of menorrhagia for several months prior to admission, associated with leiomyomata uterii.

A chest film and intravenous pyelogram performed before admission to the hospital were within normal limits. A physical examination was unremarkable. Pelvic examination revealed a 4 × 3-cm, rubbery, firm mass in the left Bartholin gland. The uterus was irregularly enlarged to the size of 14 weeks gestation by leiomyomata uterii.

Pathologic examination of the mass yielded a diagnosis of endometriosis. The formalin-fixed specimen consisted of four irregular pieces of rubbery tissue, the largest of which measured 4 × 2.5 × 1 cm. On cut sections, the larger pieces revealed gray to brown smooth surfaces. The microscopic examination revealed dense, collagenous fibrous tissue (Fig 1), with scattered nodular areas of endometriosis composed of variable admixtures of endometrial glands, stroma, inflammatory cells, extravasated red cells, and focal collections of hemosiderin-laden macrophages. Occasional glands were dilated and showed a flattened atrophic epithelium (Fig 2). In other glands there was evidence of active epithelial proliferation. Some nodules contained no recognizable epithelial components.

On May 26, 1986, an exploratory laparotomy and myomectomy were performed. There was no evidence of abdominal or pelvic endometriosis. All peritoneal reflections were normal. Both ovaries appeared normal. There were no nodules in the posterior cul-de-sac or uterosacral ligaments.

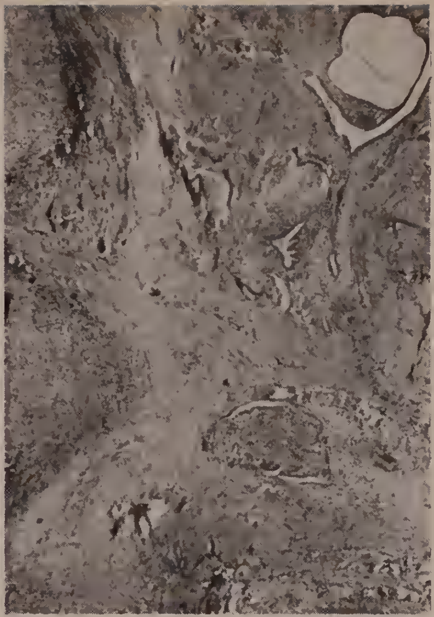


FIGURE 1. Low-power view of foci of endometriosis. Cystically dilated gland (bottom right) associated with extensive fibrous scarring and areas of hemorrhage and inflammatory cells (hematoxylin-eosin stain; original magnification × 30).

Discussion. Endometriosis of the Bartholin gland is a rare disease that may be responsible for recurrent enlargement of the gland, even in the absence of pelvic endometriosis. Although the absolute criteria for diagnosis of endometriosis are histologically demonstrable endometrial glands surrounded

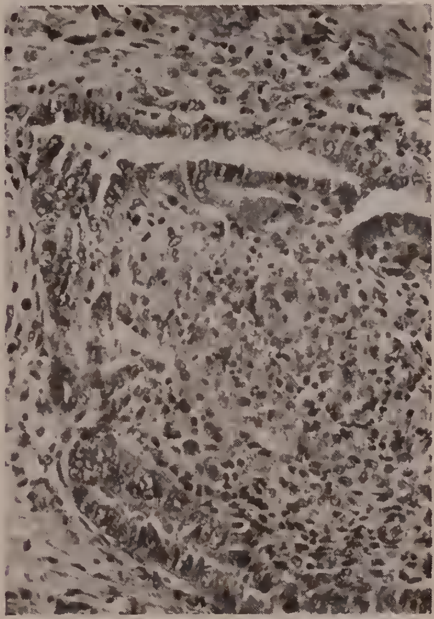


FIGURE 2. Close-up view showing proliferation of endometrial gland epithelium, inflammatory cell infiltrates, and extravasated red cells (hematoxylin-eosin stain; original magnification × 200).

by stroma, in longstanding cases extensive fibrosis may obliterate glands and stroma. Hemosiderin-laden macrophages and old blood pigment fibrosis may be the only evidence of endometriosis (Fig 1).

The origin of endometriosis in extra-abdominal pelvic sites is not fully understood. Ranney[4] states that hypotheses abound, but facts concerning the etiology of endometriosis are few. The popular theories include ceolomic metaplasia, lymphatic and vascular spread, and retrograde menstruation. Direct extension of endometriosis along the round ligament from preexisting pelvic endometriosis can explain endometriosis of the labia majora. An isolated focus of endometriosis in the Bartholin gland can, theoretically, be the result of ceolomic metaplasia, lymphatic and/or vascular spread. Hormonal stimulation can result in the growth of endometrial fragments that have reached the Bartholin gland via menstrual discharge. Endometrial fragments have the capacity to implant and grow on compatible tissue as demonstrated by the experiments of Ridley and Edward.[5] Because of this possibility, elective surgery of the cervix, vagina, or vulva should be avoided ten days prior to menstruation. Operative procedures on the lower genital tract can leave raw surfaces which can promote implantation endometriosis during subsequent menstrual bleeding.

STEPHEN MATSEOANE, MD
TOMMY HARRIS, MD
Department of Obstetrics and Gynecology

EDWARD MOSCOWITZ, MD
Department of Pathology
Harlem Hospital Center
College of Physicians and Surgeons of Columbia University
506 Lenox Ave
New York, NY 10037

1. Binder SS: Endometriosis of the vulva and perineum; report of a case. *Pacif Med Surg* 1965; 73:294–296.
2. Parmley TH: Screening techniques for vulvar and vaginal lesions. *Clin Obstet Gynecol* 1979; 22:683–688.
3. Ranney B: Endometriosis: Pathogenesis, symptoms, and findings. *Clin Obstet Gynecol* 1980; 23:865–874.
4. Ranney B: Etiology, prevention and inhibition of endometriosis. *Clin Obstet Gynecol* 1980; 23:875–882.
5. Ridley JH, Edward IK: Experimental endometriosis in the human. *Am J Obstet Gynecol* 1958; 76:783–789.

Tetracycline reduces the need for insulin

TO THE EDITOR: There have been scattered unsubstantiated reports that tetracycline reduces the need for insulin. Poltorak and Grinchenko and colleagues[1,2] have documented in animal studies that tetracycline binds insulin and influences its activity in some undetermined manner. Liver glycogen stores are also affected. Hiatt and Bonorris[3] postulated that oxytetracycline enhances the activity of insulin by interfering with epinephrine-induced glycemia, or increases the half-life of insulin. Those experiments were performed in pancreatectomized dogs.

Case Report. A 91-year-old woman with a long history of congestive heart failure and no history of diabetes was admitted to the hospital with a right pleural effusion involving almost 90% of the right lung volume. Clinical examination revealed a frail, pale woman in acute respiratory distress. Her heart rate was 100/min; blood pressure, 138/80 mm Hg; respiratory rate, 46/min. No breath sounds were heard over the right chest. Rales were heard in the lower half of the left lung field. Po_2 was 48 mm Hg on room air.

The patient initially improved with therapy that included diuretics, oxygen, and thoracenteses. Pleural fluid examination failed to reveal any malignant cells, and pleural biopsy was negative. Because the patient's systolic blood pressure (after diuresis) remained below 100, high doses of furosemide and captopril were not tolerated.

Because of continuous pleural drainage, one gram of tetracycline hydrochloride was instilled into the right pleural space. Two hours later, the patient became lethargic and then comatose. The blood glucose level at the time was 6 mg/dL. The patient was aroused after two intravenous boluses of 50 g of glucose. Approximately six hours later the patient again became stuporous. Blood glucose at this time was 23 mg/dL. Again she responded to intravenous glucose. For the next 48 hours, intravenous glucose solution was needed to stabilize the patient. She was eventually discharged with no recurrence of the pleural effusion. Her blood sugar level remained within normal limits.

Discussion. It is apparent that tetracycline has some direct hypoglycemic effect and probably also prolongs the insulin effect. Miller[4] reported a case of a patient with diabetes who became hypoglycemic after treatment with oral oxytetracycline. The profound hypoglycemia seen in the patient described here seems to support a hypothesis that tetracycline is either directly hypoglycemic or somehow prolongs the half-life of insulin. This case also demonstrates the need to be aware of adverse and unusual drug reactions. It is conceivable that tetracycline could be used in the future as adjunctive therapy in the treatment of diabetic patients.

VINCENT P. GARBITELLI, MD
71 East Williston Ave
East Williston, NY 11596

1. Poltorak VV, Grinchenko TS: Vliianie antibiotikov tetratsiklinovogo riada na uglevodnyĭ obmen (Effect of tetracycline series antiobiotics on carbohydrate metabolism). *Antibiotiki* 1978; 23:276–282.
2. Grinchenko TS, Poltorak VV, Rubinshtein ML: Vliianie antibiotikov tetratsiklinovogo riada na funktisiiu beta-kletok insuliarnogo apparata podzheludochnoi zhelezy (Effect of tetracycline series antiobiotics on beta cell function of pancreatic islets). *Antibiotiki* 1978; 23:1097–1102.
3. Hiatt N, Bonorris G: Insulin response in pancreatectomized dogs treated with oxytetracycline. *Diabetes* 1970; 19:307–311.
4. Miller JB: Hypoglycaemic effect of oxytetracycline [letter]. *Brit Med J* 1966; 2:1007.

BIOSTATISTICS IN CLINICAL MEDICINE

Second Edition. By Joseph A. Ingelfinger, MD, Frederick Mosteller, PhD, Lawrence A. Thibodeau, PhD, James H. Ware, PhD. 339 pp, illustrated. New York, Macmillan Publishing Co Inc, 1987. $29.95 (softcover)

This group of authors, distinguished in both medicine and biostatistics, has presented an introduction to statistical concepts and methods, with physicians as their intended audience. The book purports to prepare "physicians to use and understand applications of probability and statistics for the care of the individual patient." That goal is effectively pursued with a richness of clinical examples, used to illustrate the diverse statistical procedures discussed.

The emphasis throughout is placed upon the intuitive aspects of statistical concepts, rather than the mathematical aspects, but quantitative rigor is not sacrificed. Cogent discussions of the statistical concepts being illustrated are, however, sometimes sacrificed to lengthy discussions of the clinical problems presented as examples. Highlighting with boxes and prudent repetition of critical points are judiciously applied for emphasis.

A lucid presentation of the rules of probability as applied to problems of diagnosis should make these notions more palatable to motivated physicians, who may have been frustrated with lengthy discussions of coin tosses in other texts. Effective presentations of odds and likelihood ratios and decision trees are welcome additions to an introductory text. Requisite chapters on the basic descriptive statistics of central tendency and variability are freshly presented with the promised relevance to clinical care. The role of the common distributions in statistical inference, including the normal, Student's t, binomial, and Poisson, is also well handled. Omission of the exponential distribution in this setting is unfortunate. There is a good chapter on chi-squared analysis, and the discussion of regression analysis is enhanced with examples of common computer results of such analyses.

Hypothesis testing is afforded only cursory attention as a topic; however, one of the highpoints of the book is a chapter devoted to P values. Issues surrounding the application of P values to clinical questions are seriously discussed, including the several ambiguities of the word "significance," and the choice of the 0.05 level. The role of power in the interpretation of P values receives admirable treatment. Another highpoint is a chapter on clinical trials, in which problems associated with the design and execution of clinical trials are discussed, as are problems associated with interpretation of clinical trials. An accompanying chapter on the application of clinical trial results is less effective.

A new chapter on life table analysis represents the only important addition to this second edition. It is perhaps the most disappointing chapter in the book, providing a barely adequate introduction to this important and rapidly developing statistical technique. The contrast between the elementary treatment of life tables and the scholarly exposition of odds illustrates an unevenness of presentation, which can be a minor distraction. This is not an easy text to use, and the extent to which it has the potential to influence the delivery of patient care is problematic. However, for the physician who is seriously desirous of gaining a better understanding of statistical procedures, but whose mathematical grasp does not exceed algebra, this book is a superb introduction to probabilistic thinking. The relevance of these biostatistical concepts to a broad spectrum of clinical problems is presented both literately and authoritatively. Most of the material is complete and current, and some discussions cannot be found elsewhere.

OLIVER GLIDEWELL
Montoursville, PA 17754

THE DEVELOPMENT OF AMERICAN PHYSIOLOGY. SCIENTIFIC MEDICINE IN THE NINETEENTH CENTURY

By W. Bruce Fye, MD. 308 pp. Baltimore, Md, The Johns Hopkins University Press, 1987. $35.00 (hardcover)

This book is a timely discussion of the role of physiology in the maturation of American science and medicine. It is timely not only because 1987 marks the hundredth anniversary of the founding of the American Physiological Society, but because at present there has been a loss of the perception of physiology as an essential foundation of medicine and because major concern with reductionist thought is fragmenting the discipline and causing a loss of integrative thought so characteristic of physiology.

Following the account of the development of American physiology and the parallel revolution in American medical education given in the introduction, the author describes the careers of the men considered most responsible for these developments. Four were chosen: First, John Call Dalton, Jr: pioneer vivisector (founder of experimental teaching) and America's "First Professional Physiologist." He was founder of the Departments of Physiology at Buffalo, Vermont, what became the Long Island College of Medicine in Brooklyn, what is now the State University of New York Health Science Center at Brooklyn, and finally what is now Columbia University College of Physicians and Surgeons. A second was S. Weir Mitchell, Philadelphia's often frustrated physiologist and triumphant reformer. He provided much of the energy and drive required for organizing the American Physiological Society. The third was Henry P. Bowditch, a man of the highest prestige carrying the heritage of Carl Ludwig to America; Dalton carried Claude Bernard's. The author refers to Bowditch as the prototypical full-time physiologist and educational reformer. A thesis of the book is that American physiology attained stature when the country's institutions became able and willing to support full-time professional physiologists. The fourth leader described by the author was H. Newell Martin, who was involved in establishing physiology at the Johns Hopkins University and in developing the initial research ethic required for modern medicine.

The fifth chapter is entitled, "the American Physiological Society: a Profession Defining Itself." Although the men previously mentioned were the leaders, others were also involved. The first meeting was held in 1887 in the new physiological laboratory of the College of Physicians and Surgeons; Professor John G. Curtis was host. There were 28 original members. Some of the younger members, with a few others who were elected within the next few years, really brought American physiology to a position of preeminence after the first World War and by the 1930s. This book, however, treats only the earlier years, a quarter of the 19th century, and an early decade or two of the 20th. This chapter, like the preceding four, contains many details of the concerns, the struggles, and the attainments of the early days.

The sixth and final chapter deals with the influence of physiology on American medicine; the title is "A Scientific Prescription for American Medicine." Obviously the author deals with more than the history of the early development. There is an account of the 1910 Flexner report and its effects. The principles essential to high attainment are delineated but as the subtitle indicates, it is an account of physiology and "Scientific Medicine in the Nineteenth Century." The reader is left with the job of applying the teaching of history to today's problems.

The book concludes with appendices listing the institutions involved in the early meetings of the American Physiological Society. There is a bibiography of 28 manuscript collections and 286 individual references plus an index. The author has contributed a valuable reference work and a most interesting account of our history.

CHANDLER McC. BROOKS, PhD
State University of New York
Health Science Center
at Brooklyn
Brooklyn, NY 11203

MODERN MEDICINE AND JEWISH ETHICS

By Fred Rosner, MD. 405 pp. Hoboken, NJ, KTAV Publishing House Inc, 1986. $22.50 (hardcover)

The burgeoning developments in interventional therapeutic capabilities during the past three decades have spawned an impressive, though often less definitive, growth in general medical ethics. An early landmark in the development of Jewish medical ethics as a discrete discipline was the appearance in 1959 of a book by that name written by the present reviewer's father, Sir Immanuel Jakobovits, whose brief foreword graces the current work. By now the subject has taken off and has attracted many able writers and thinkers, medical and rabbinical, whose continuing contributions have assured the interested reader of numerous literary resources.

Fred Rosner, MD, has achieved well-earned preeminence in the secular and religious communities as a remarkably prolific and gifted exponent of the teachings an ancient religion brings to bear on many of today's medical dilemmas. As Chief of Medicine at a leading New York hospital and Professor of Medicine at the State University of New York Health Science Center at Stony Brook, he has direct contact with many of the medico-moral issues of which he writes. In addition, as a notable figure in numerous prominent regional and national medical ethics "think tanks," he is uniquely qualified; indeed, his is a virtual household name among the evergrowing cadre of religiously concerned physicians.

Modern Medicine and Jewish Ethics presents 26 topics in as many chapters culled in large part from previous essays by Dr Rosner. Virtually all the major areas of concern are touched upon. The discussions manifestly demonstrate how Jewish religious values and mores may be intelligently applied to even the most technically advanced situations. Analyses range from fetal surgery and sex-organ transplants to mechanical hearts and the appropriate use of artificial life supports. Fascinating questions regarding the allocation of scarce medical resources, smoking in public places, the definition of death, and animal experimentation are among the many thought-provoking essays which will appeal to a wide variety of readers. Conversely, several topics, such as mental health issues, are mentioned only parenthetically, and the Jewish attitudes to homosexual and other "alternate" sexual behaviors—so important in these days of the AIDS epidemic—are omitted altogether.

In recent years there has been a surge of interest at the grass roots level of the Jewish community in Tay-Sachs disease and in its prevention by mass screening. The implications of such screening and the potentially detrimental concerns which it may foster in the minds of poorly informed young men and women are sympathetically reviewed. Although the author thinks that, on balance, the risk-benefit ratio warrants such screening, the pros and cons are fairly presented. Indeed, an appealing feature of the book in general is its penchant for presenting all sides of an argument fairly and fully, and yet not leaving the reader so unguided as to be unsure of the author's position.

The division between ethics and ritual is intentionally vague in the Jewish tradition, so the inclusion of chapters on priestly defilement, embalming and cremation, and the therapeutic efficacy of prayer should engender no surprise, though they may be of less universal application than the remaining 23 sections. Otherwise, little reference is made to several important ritualistic Jewish concepts relevant to medical practice, such as the Sabbath and kosher food regulations.

The author notes that "Today, many physicians and patients do not feel firmly bound by religious precepts. Religious guidance is available, however, to those who seek it" (page 217). This book is not designed to draw readers to a religious theory of medical ethics. Those who seek, however, an authentically Jewish viewpoint on a kaleidoscope of issues will be richly rewarded by the breadth, erudition, and documentation contained within its pages.

JULIAN JAKOBOVITS, MD
Johns Hopkins University
School of Medicine
Baltimore, MD 21205

CARDIAC ARRHYTHMIAS: SELF-LEARNING

By Edward K. Chung, MD, 200 pp, illustrated. New York, Yorke Medical Books, 1986. $40.00 (hardcover)

This publication, a self-learning companion volume to the *Manual of Cardiac Arrhythmias*, should prove useful for primary care physicians, internists, medical residents, and cardiac care nurses. However, because of a lack of sufficient depth and thoroughness in certain areas, and because of some questionable interpretations in the text, it would not be suitable for cardiologists or cardiology trainees.

The book consists of nine chapters covering A-V block, intraventricular block, sinus arrhythmias, A-V junctional arrhythmias, ventricular arrhythmias, differential diagnosis of cardiac arrhythmias; Wolff-Parkinson-White Syndrome; cardiac arrhythmias related to artificial pacemakers, and miscellaneous ECG findings.

The omission of examples of some important and not uncommon arrhythmias is regrettable. These include Mobitz II type A-V block in Chapter 1, bradycardia (phase 4) related to bundle branch block in Chapter 2, Wenckebach type S-A block in Chapter 3, concealed junctional premature beats in Chapter 5, and torsades de pointes in Chapter 6. A number of cases are described wherein the author's interpretation of the arrhythmia can be questioned. For example, the diagnosis of "variant" of Wenckebach A-V block or "variant" of Mobitz II A-V block is questionable in patients with 2:1 A-V block and normal QRS complexes since the level of A-V block cannot be ascertained by surface electrocardiography (case #16). In case #43, the left anterior hemiblock is clearly rate related, and the QRS complexes without LAH do not suggest the presence of LVH. In case #54, S-A block of 4:3, 3:2, and 2:1 is unquestionably present, and the diagnosis of Mobitz II S-A block is very doubtful.

"Chung's Phenomenon," referred to in cases #138 and #191, inappropriately ascribes to Dr Chung credit for first recognizing the finding of "alteration of the P wave configuration of the sinus beat (the 4th and the 9th beats) immediately following APCs. This finding has been termed 'aberrant atrial conduction.' " Actually, this phenomenon was first reported by Sir Thomas Lewis in 1921 and again described in detail by Scherf and Schott in *Extrasystoles and Allied Arrhythmias* (1953).

Despite some of the shortcomings mentioned above, this is the first book of which the reviewer is aware that combines electrocardiographic diagnosis, an assessment of clinical significance of the arrhythmias, and a suggested therapeutic approach.

YUAN KUANG KU, MD
Long Island College Hospital
Brooklyn, NY 11201

BEAT THE ODDS

By Harold S. Solomon, MD. 311 pp, illustrated. New York, Villard Books, 1986. $19.95 (hardcover)

This book for the lay public is a comprehensive approach to awareness and the need for behavior change. It specifically addresses many aspects of personal health, including safety, macroenvironmental factors, and lifestyle factors such as substance abuse which contribute to the nation's leading causes of disability and death. In addition to educating the reader regarding the need to reduce personal health risks, it offers recommendations and methods to achieve this goal.

The author's background in internal medicine and hypertension supports this fine effort to reach people with an approach to wellness. The text has been written in terminology that the average layperson should comprehend with little difficulty. The book is an excellent resource for the physician who believes that patient education and knowledge is the key to compliance. The physical quality of the book is impressive, the print size allowing for ease of reading by all ages. A few typographical errors were noted. Lay readers may feel overwhelmed with the amount of information presented and annoyed with frequent repetition. A few terms were poorly defined (eg, ischemia, maximum heart rate). The organization of the text is simple and easy to follow. The chapter on a physician's perspective on exercise appears out of place and would seem more appropriately placed within the section offering strategies for change. Greater emphasis could have been placed on precautions concerning exercise and additional information given on the theory behind it.

The book's comprehensive perspective relates lifestyle to many disease processes. All recommendations made are consistent with the guidelines established by recognized professional organizations, such as the American Dietetic Association, the American College of Sports Medicine, and the American Heart Association. The author's objectives to educate the lay population regarding the need to modify one's life if the "odds are to be beat" are clear. The strategies offered and the opportunity for feedback on one's personal progress are what distinguishes this book from others available. *Beat the Odds* should be a part of any health educator's library.

MARY CARDAMONE
Geisinger Medical Center
Danville, PA 17822

TAKING THE WORLD IN FOR REPAIRS

By Richard Selzer, MD. 239 pp, New York, William Morrow & Co, Inc, 1986. $15.95 (cloth)

Unfortunately, I have not read any of the four books written by Dr Selzer prior to this one. His writing technique is superb but the subjects he selected for the 12 short stories in this work are odd and may be distasteful to some people. The first story is dull. It describes the daily life in a monastery he visits. While there, he finds that one of the monks has Grave's disease, and the abbot develops a carbuncle which is surgically treated by the author. The other stories involve, for example, a slaughterhouse, a laundry, a bee stuck on a tart, and a man who becomes a part of the life cycle of a guinea worm.

The last story bears the same title as the book and I found it to be the most interesting. It tells of a trip to Peru by a health care team sponsored by an organization called "International Plastic Surgeons Inc." It was founded in 1969 and serves indigent patients in third-world countries. Dr Selzer is at his best in depicting the emotional impact on the children whose deformities are corrected by this group. This book will depress those readers who are seeking pleasure and relaxation. However, those interested in creative thinking and good writing will find this book worth reading.

JULIUS E. STOLFI, MD
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

THE MEDICARE SYSTEM OF PROSPECTIVE PAYMENT. IMPLICATIONS FOR MEDICAL EDUCATION AND PRACTICE

Edited by Mohan L. Garg, ScD, and Barbara A. Barzansky, PhD. 229 pp. New York, Praeger Publishers Inc, 1986. $38.95 (hardcover)

Basically this well-referenced book argues the age-old battle between taking an action for the collective good of society versus acting for the benefit of the individual. Medicare's prospective payment system (PPS), utilizing the diagnosis related groups (DRGs) for reimbursement, is the decision that is the crux of the 15 chapters in this book that consider the implications of PPS/DRG on medical practice and education.

People in the health care industry do not have to be told that the changes ensuing from the implementation of the PPS/DRG scheme are complex, fundamental, and somewhat unpredictable. With solid historical and political background data tied into supporting evidence from the literature, the authors present their perspectives with reasoned prose and speculative predictions. Everyone may not agree with some of the optimistic projections, since the disaster trends appear more frequently in professional journals. However, the foreword does note that "not infrequently out of turmoil comes opportunity."

Topics covered include items on health care cost inflation and regulatory efforts to curtail the upward spiral, changing the practice behavior patterns of physicians, how to improve clinical decision making by physicians, new roles for physicians in hospital decision making, the impact on teaching hospitals and graduate medical education, whether or not technology development will be retarded, the role of professional review organizations, what will happen to quality assurance, utilization review strategies, legal issues including constitutionality and regulations, consideration of the morality or immorality of PPS/DRG, and the need to tighten the horizontal and vertical organization of teaching hospitals.

While a good deal of the material does seem to dwell on the academic locus of the teaching hospital and graduate medical education, the importance to the physician in his/her private office should not be negated by the titles of the chapters. For example, Schmitt and Berner discuss strategies to improve clinical decision making by physicians. They believe that PPS/DRG makes it necessary to upgrade the manner in which a physician decides to choose one form of therapy over another. Using illustrations of branching logic, analysis styles, and clinical algorithms, the authors make a case for cost containment forcing judgments into behavior practices in medical care. In a related chapter, Garg, Coulson, and Barzansky review the multiple variables of a physician's practice that influence the cost of health care. Citing research, they reiterate the fact that physicians are usually unaware of what medication costs or how much the hospital charges for lab tests or other services. Since PPS/DRG calls for an examination of the utilization of resources, the difficult task of altering medical practice patterns to contain cost expenditures must be undertaken if hospitals are to remain financially solvent. In addition to making better cost conscious decisions and using limited resources, Schroeder forecasts that new equipment in the future will only be technology that saves money while it flashes, blinks, whistles, and prints out fancy diagrams. Komaroff raises the critical issue of whether high quality care can be achieved if one has to have an accountant at one's side tabulating the cost as one sutures a wound. When does quality assurance take precedence? He does posit that the PPS/DRG system must do more to include severity of illness gradations in its payment mechanism to account for variances in clinical care within the same DRG. Loudly and clearly, the implications of PPS/DRG on the US health care delivery system appear to be punctuated with dollar signs. Although the PPS/DRG plan currently applies only to inpatients covered under Medicare, other third party payers are jumping on the bandwagon. Furthermore, the federal legislation opens the door for similar cost containment measures for other facilities, ambulatory care, psychiatric care, home care, long term care, and so on.

Callahan considers the ethical morality of the PPS/DRG plan and decides that it is not immoral to try to curtail costs. However, he does point out that conflicting loyalties arise for the physician who owes his/her patient the best care but also has a concern to keep the hospital from going bankrupt.

Freymann feels that the public will insist on good medicine rather than "Mac Medicine" even though it may cost a bit more. He sums up the situation strongly: "Beyond the balance sheet, beyond the quarterly dividends, the doctor's authority is inviolable in the end—if they exert it. Socrates said it 24 centuries ago: 'For the true physician is also a ruler having the human body as a subject, and is not a mere moneymaker.' "

To provide insight into the many facets and implications of the PPS/DRG cost containment scheme, this book is a sound investment of your time and energy.

ALLEN D. SPIEGEL, PhD
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

A DIABETIC DOCTOR LOOKS AT DIABETES: HIS AND YOURS

By Peter Lodewick, MD. 232 pp, illustrated. New York, Bantam Books, 1984. $3.95 (paperback)

Family physicians are always on the lookout for new patient education aids. Most commonly these take the form of materials supplied by the pharmaceutical houses or national medical societies. The lay press and major medical journals are also a good source for these materials. Occasionally, a book such as *A Diabetic Doctor Looks at Diabetes: His and Yours* comes along which succeeds in achieving its stated goal of providing patients with a better understanding of this multisystemic disease. Written by Peter A. Lodewick, MD, an endocrinologist and an insulin-dependent diabetic, this book gives its readers a detailed, precise, comprehensive overview of diabetes mellitus. Gearing his book toward diabetic patients and their families, the author makes a conscious effort to provide emotional encouragement to the nearly six million Americans suffering from this chronic disease.

The book is broad-ranging in content, and is written in a clear, concise style that is very readable. Dr Lodewick, president of the American Diabetic Association, New Jersey affiliate, guides the reader through this complex disease. By beginning with the basic definitions of diabetes mellitus and continuing through the different types of the disease and its natural course, the author makes readers aware of why therapeutic compliance is so important. Symptoms and signs are well described, and the diagnostic tools and latest treatment modalities available to physicians are covered. To help clarify his information, Dr Lodewick makes use of numerous case histories, many of them reflecting his own experiences as a patient with diabetes. I was particularly pleased to see chapters on glycohemoglobin and foot care. Other chapters in this book discuss diabetes and pregnancy, impotence, alcohol, and travel. The author concludes his book by answering the ten questions he hears most often, and by providing a list of recommended readings.

A Diabetic Doctor Looks at Diabetes: His and Yours comes across as a compassionate and upbeat guide to living an active and successful life with diabetes. While technically correct and contemporary in its tone, the book strives to provide an emotional lift to the reader. The author is successful in achieving that goal, mainly due to his insights from the dual perspective of physician and patient. As such he appears unafraid to discuss the emotional issues faced by newly diagnosed diabetics.

Although many instructional texts on diabetes are available, *A Diabetic Doctor Looks at Diabetes: His and Yours* is a valuable asset to its readers. It is written for the nonprofessional and offers both medical and emotional advice. Its price is not prohibitive, and both patients and primary care physicians will find this a helpful and enjoyable book to read.

DAVID BRECHER, MD
Mease Clinic Countryside
Safety Harbor, FL 33572

AMA's socioeconomic survey

The American Medical Association's Center for Health Policy Research released its most recent Socioeconomic Monitoring Survey Report. According to the report, the major problems facing physicians are the increasing cost of liability insurance and the growing number of claims (*JAMA* 1987; 258:455).

More specifically, the report states that annual liability insurance premiums averaged $10,000 in 1985. Paying $23,000, on average, obstetricians/gynecologists claimed the highest rates. During 1985, the average annual rate of claims rose to 10.1/100 physicians, compared to 8.2/100 for the previous four years. In addition, close to 9% of physicians faced at least one claim in 1985.

Increasing power of medical malpractice insurers

Malpractice insurers are setting guidelines and rules that will govern how physicians practice medicine. Failure to abide by these guidelines can result in the loss of liability coverage. What are the medical, legal, and economic ramifications of such a system? Mark Crane examined these issues in a special report for *Medical Economics* (1987; 64:23–29).

The arrangement is as follows: In exchange for supplying malpractice insurance, the insurers want to dictate terms regarding certain medical practices, including drug prescription, the decision to perform specific procedures, referral to specialists, and record-keeping practices. The insurers' goal is to reduce the risk associated with patient care, particularly involving those specialties and procedures associated with high risk and, ultimately, higher insurance losses. The "insurability guidelines," which have been adopted by almost half of the companies included in a recent survey by the Physician Insurers Association of America, focus on the practice of obstetrics and anesthesia, the diagnosis of breast cancer, and the issue of informed consent.

Penalties for defying the guidelines can range from surcharges of up to $25,000 to loss of coverage. Insurers may also refuse to renew coverage if physicians perform a procedure for which they are not appropriately trained. Compliance with the rules is generally determined after a claim is filed. Although companies will defend a client even if his or her practices were found to deviate from the rules, loss of coverage commonly results.

As liability insurance premiums skyrocket for obstetricians, as well as for family practitioners who deliver babies, one company has instituted the following guidelines: High-risk patients should be referred to an obstetrician; referral is required for patients with insulin-dependent diabetes, severe eclampsia or pre-eclampsia, multiple gestation, history of previous cerclage, hydatiform mole, serious cardiac or hematologic diseases, or confirmed intrauterine growth retardation. Furthermore, physicians must use "a prenatal scoring system for every patient to determine risk and appropriate management in six areas: cesarean sections, use of instruments, management of hypertension, monitoring of fetal distress, management of pre- and postmature deliveries, and administration of oxytocin." For example, the guidelines state that only a physician can administer oxytocin, the rate of delivery of which is set in the guidelines, and patients receiving oxytocin must have electronic fetal monitoring.

Another insurance company has applied stringent requirements to anesthesiologists and has achieved a dramatic reduction in paid claims. As a result, the company was able to reduce premiums for anesthesiologists. The guidelines included requiring the presence of an anesthesiologist or a certified nurse-anesthetist in the operating room at all times, and requiring the use of two simultaneous methods of pulse and blood pressure monitoring, as well as predetermined standards governing the use of regional and general anesthesia.

These insurance company-sponsored programs have been in effect for, at most, two years; their impact on malpractice claims, liability premiums, and medical practice and patient care is not yet known. Notes author Crane, "The financial impetus for the insurers is compelling, and the legal impact on physicians could be far-reaching."

Health effects of radon exposure

Increasing awareness of the sometimes high levels of radon in homes has led to widespread media attention and numerous studies on the health effects of radon exposure. Despite the limited data available on radon levels in North American homes, the results of preliminary studies suggest that exposure to excessive radon levels may be responsible for several thousand cases of lung cancer each year. The Council on Scientific Affairs of the American Medical Association (AMA) has reviewed the data on radon exposure, evaluated the resulting health effects, and developed three basic recommendations (*JAMA* 1987; 258:668-672).

Radon 222 is a radioactive gas that forms from the decay of radium 226. Radium is a mineral irregularly distributed throughout the earth's crust and present in earth-based materials such as soil and rock. As this radium decays, the radon gas rises through the soil to enter the atmosphere. If allowed to enter a home through joints or cracks in the foundation, radon can accumulate to hazardous levels. When inhaled, the radon continues to decay, thus irradiating pulmonary tissues.

The US Environmental Protection Agency (EPA) has recommended the following: concentrations in homes should not exceed 0.02 WL (working level) of radon decay products ("equivalent to 4pCi of radon per liter of air assuming 50% equilibrium"). Investigators have concluded that the average radon level in US homes is 1.5 pCi/L, although large variations have been documented. One to three percent of homes had levels that exceeded 8 pCi/L.

"A primary determinant of the level of radon in a house is the makeup of the earth under and around the house, especially the pressure of radon gas in the soil and pressure differentials between the soil and the house." Ventilation is another major determinant of radon levels; ex-

---

NEWS BRIEFS are compiled and written by Vicki Glaser.

hausting indoor air with fans or using a heat exchanger can reduce radon levels. Energy-saving techniques, including insulating, weatherstripping windows and doors, and caulking, virtually seal a house and can increase the level of radon gas.

Another important source of radon is the water supply. Water may account for as much as 35% of the increased level of radon inside a house as compared to outside. Radon escapes from the water during routine activities such as washing, drinking, and cleaning. Regions with the highest water levels of radon are the Appalachian-Piedmont area and New England.

The health effects associated with exposure to radioactive decay products are not well understood. Previous studies have focused on the risks to uranium miners. "A conservative position of protection against adverse effects of radiation assumes that the risk of developing radiogenic malignant neoplasms is proportional to radiation exposure." However, the risks of exposure to low-level radiation are not clear. One investigator determined that the average person suffers exposures of about 0.2 WLM (working-level-month)/year. This would lead to somewhere between 1,000 and 20,000 cases of lung cancer each year in the US. Other estimates suggest that 10,000 cases of cancer per year may be due to radon in homes.

The Council on Scientific Affairs has recommended that the AMA play an active role in the education of physicians, other health care professionals, and the public as to the potential risks of high radon levels in homes and other buildings. In addition, the Council recommended that the AMA should catalyze the organization of a conference to address the issue of radon. Other participants would include the Environmental Protection Agency, the Department of Energy, and the National Institute of Environmental Health Sciences. Finally, according to the Council, it is imperative that accurate measures be obtained of ambient radon levels in a sample of homes, workplaces, and commercial buildings.

Carpal tunnel syndrome: cause and treatment

Two recent reports have focused on carpal tunnel syndrome, which is characterized by numbness and/or burning pain on the palmar surface of the first three digits of the middle hand. The disorder results from compression of the median nerve at the carpal tunnel. Risk factors for carpal tunnel syndrome include hypothyroidism, rheumatoid arthritis, pregnancy, diabetes mellitus, acromegaly, amyloidosis, neuromas, and stress on the nerve caused by repeated flexion and extension at the wrist.

Barnhart and Rosenstock reported a cluster of seven patients with carpal tunnel syndrome, all of whom were grocery checkers (*West J Med* 1987; 147:37–40). As grocery checking requires highly repetitive motions involving the wrist, and no other risk factors were apparent, the authors concluded that in this patient group, carpal tunnel syndrome was probably a result of job-related activity. A review of the literature supported this conclusion.

Surgical decompression of the median nerve at the carpal tunnel is the definitive mode of treatment. Conservative therapeutic alternatives include splinting of the wrist, injection of hydrocortisone into the carpal canal, and the use of nonsteroidal anti-inflammatory agents.

Ellis and colleagues have demonstrated a correlation between the clinical symptoms of carpal tunnel syndrome (CTS) and the specific activity of erythrocyte glutamic oxaloacetic transaminase (EGOT), an enzyme that is released into the serum following tissue injury (*South Med J* 1987; 80:882–884). The authors reported that "the lower the EGOT activity the more severe and more numerous were the signs and symptoms of CTS." This finding is based on a study of 35 selected patients with carpal tunnel syndrome, who had the more common, idiopathic, nonspecific form of the syndrome, with no known predisposing disorders.

Pyridoxamine, an active form of vitamin B_6, acts as a coenzyme with EGOT; EGOT cannot function without the help of vitamin B_6. Thus, the EGOT test can be used to determine a person's vitamin B_6 status. Ellis treated the 35 patients with 100–200 mg/day of vitamin B_6. Following four weeks of this therapy, the vitamin deficiency was corrected. Symptoms of carpal tunnel syndrome improved or resolved following 12 weeks of therapy. Improvements included reduced swelling of hands, improved flexion of finger joints, increased pinch pressure, and diminished paresthesia in the fingers. Ellis noted improved results on electromyographic tests following vitamin B_6 therapy, "but not a return to normal even when clinical improvement was significant."

The recommended dietary allowance (RDA) of vitamin B_6 is 2.2 mg/day for men, 2.0 mg for women, and 2.6 mg for pregnant women. "I recommend that 50 mg of vitamin B_6 be given daily, beginning with diagnosis of pregnancy," states the author. "In the presence of edema of pregnancy, 200 mg is preferrable to prevent CTS in the third trimester of pregnancy." For the general population, Ellis concluded, "the US RDA of 2 mg for vitamin B_6 is marginal for CTS."

CPMC seeking participants for three multicenter trials

Columbia-Presbyterian Medical Center (CPMC) in New York is participating in three national, multicenter trials to study new treatment methods for Parkinson disease, stroke, and sudden cardiac death. The Neurological Institute at CPMC will test the effectiveness of two drugs—deprenyl and tocopherol—in delaying the progressive, debilitating effects of Parkinson disease.

The institute is also involved in the five-year, Asymptomatic Carotid Atherosclerosis Study, which will include 1,500 patients nationwide. The study will determine whether a surgical approach to stroke prevention, using the carotid endarterectomy, is more effective than treatment with aspirin. Eligible patients will exhibit significant narrowing of the carotid artery due to atherosclerosis, with no symptoms of stroke.

The Cardiac Arrhythmia Suppression Trial will encompass 138 hospitals and 5,000 patients in the United States, Canada, and Sweden. Researchers will study the effectiveness of three antiarrhythmic drugs—encainide, flecainide, and morcizine—in preventing sudden cardiac death.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Leon Benton, MD, St Louis, Missouri. Died April 17, 1987; age 78. Dr Benton was a 1936 graduate of Deutsche Univerzita Medizinishe Fakulta, Praha, Czechoslovakia. He was a member of the Medical Society of the County of Ulster and the Medical Society of the State of New York.

George Blinick, MD, Boca Raton, Florida. Died June 6, 1987; age 79. Dr Blinick was a 1932 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Academy of Medicine, the New York Obstetrical Society, the New York County Medical Society, and the Medical Society of the State of New York.

Bernard Bloom, MD, Kiamesha Lake. Died September 10, 1986; age 66. He was a 1948 graduate of Chicago Medical School, Chicago. Dr Bloom was a member of the Sullivan County Medical Society and the Medical Society of the State of New York.

Frances L. Hacker Bogatko, MD, Rome. Died June 27, 1987; age 95. Dr Bogatko was a 1917 graduate of the Medical College of Pennsylvania, Philadelphia. She was a member of the New York County Medical Society and the Medical Society of the State of New York.

Jean Pierre Bonin, MD, Sidney. Died June 6, 1987; age 58. Dr Bonin was a 1962 graduate of Faculte de Medicine de l'Universite de Lausanne, Lausanne, Switzerland. He was a member of the Delaware County Medical Society and the Medical Society of the State of New York.

Eugene Braude, MD, New York. Died May 31, 1987; age 87. He was a 1925 graduate of Medizinische Fakultaet der Ludwig Maximilians Universitaet, Muenchen, Bayern, Germany. Dr Braude was a member of the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Lubomyr Bylow, MD, Port Crane. Died June 1987; age 75. Dr Bylow was a 1945 graduate of Charles Univerzita Fakulta of Ped-Gen Medicine, Praha, Czechoslovakia. Dr Bylow was a member of the American Psychiatric Association, the Broome County Medical Society, and the Medical Society of the State of New York.

Lawrence Thomas Cacioppo, MD, St Augustine, Florida. Died June 25, 1987; age 87. Dr Cacioppo was a 1924 graduate of State University of New York Health Science Center at Brooklyn. He was a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Otolaryngology. His memberships included the American Academy of Otolaryngology, the American Academy of Facial Plastic and Reconstructive Surgery, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Alexander Cameron, MD, Sarasota, Florida. Died May 8, 1987; age 92. Dr Cameron was a 1919 graduate of New York University College of Medicine, New York. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Gerard Ross Carroll, MD, Highland Falls. Died June 6, 1987; age 66. He was a 1952 graduate of Faculty of Medicine University of Glasgow, Glasgow, Scotland. Dr Carroll was a Diplomate of the American Board of Family Practice and a member of the Medical Society of the County of Orange and the Medical Society of the State of New York.

June G. Corey, MD, Suffern. Died June 21, 1987; age 38. She was a 1974 graduate of New York Medical College, New York. Dr Corey was a Diplomate of the American Board of Family Practice and a member of the American Academy of Family Practice, the Medical Society of the County of Rockland, and the Medical Society of the State of New York.

Marie Leonore Cote, MD, Newburgh. Died June 6, 1987; age 67. She was a 1945 graduate of Albany Medical College of Union University, Albany. Dr Cote was a Diplomate of the American Board of Pediatrics and a member of the American Academy of Pediatrics, the Medical Society of the County of Orange, and the Medical Society of the State of New York.

Clarence De La Chapelle, MD, New York. Died June 13, 1987; age 90. Dr De La Chapelle was a 1922 graduate of New York University School of Medicine, New York. He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the Academy of Medicine, the New York Pathological Society, the International Academy of Pathology, the American Association of Pathologists and Bacteriologists, the New York County Medical Society, and the Medical Society of the State of New York.

Felice J. De Stefano, MD, Brooklyn. Died July 5, 1987; age 73. Dr De Stefano was a 1938 graduate of Creighton University of Medicine, Omaha. Dr De Stefano was a Qualified Fellow of the International College of Surgeons and a member of the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Harry David Fein, MD, New York. Died July 21, 1987; age 75. He was a 1936 graduate of New York University School of Medicine, New York. Dr Fein was a Fellow of the American College of Physicians and the American College of Gastroenterology and a Diplomate of the American Board of Internal Medicine. His memberships included the Academy of Medicine, the New York Gastroenterological Society, the American Gastro-Enterological Association, the New York County Medical Society, and the Medical Society of the State of New York.

Louis Joel Feit, MD, New York. Died May 2, 1987; age 79. He was a 1934 graduate of New York Medical College. Dr Feit was a Fellow of the American College of Surgeons, a Qualified Fellow of the International College of Surgeons, and a Diplomate of the American Board of Otolaryngology. His memberships included the American Academy of Otolaryngology, the American Academy of Facial Plastic and Reconstructive Surgery, the New York County Medical Society, and the Medical Society of the State of New York.

Mayer Fisch, MD, New York. Died May 7, 1987; age 61. Dr Fisch was a 1948 graduate of New York Medical College, New York. He was a Diplomate of the American Board of Psychiatry and Neurology and a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Joseph Osias Fisher, MD, Portland, Oregon. Died May 30, 1987; age 89. Dr Fisher was a 1927 graduate of the University of Maryland School of Medicine, Baltimore. He was a member of the American Academy of Family Practice, the American Geriatrics Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

Jesse Morris Frankel, MD, Brooklyn. Died June 23, 1987; age 87. He was a 1923 graduate of New York University School of Medicine, New York. Dr Frankel was a Fellow of the American College of Surgeons, a Diplomate of the American Board of Obstetrics and Gynecology, and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Meyer Friedenson, MD, Yonkers. Died January 19, 1987; age 82. Dr Friedenson was a 1927 graduate of Yale University School of Medicine, New Haven. He was a Fellow of the American College of Physicians, a Diplomate of the American Board of Internal Medicine, and a member of the New York County Medical Society and the Medical Society of the State of New York.

Antonio R. Ginorio, MD, Fort Lauderdale, Florida. Died July 15, 1987; age 69. He was a 1944 graduate of Loyola University Stritch School of Medicine, Maywood, Illinois. Dr Ginorio was a Diplomate of the American Board of Orthopedic Surgery and a member of the American Academy of Orthopaedic Surgeons, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Joseph G. Glasser, MD, New York. Died June 12, 1987; age 77. Dr Glasser was a 1937 graduate of Medizinische Fakultaet der Universitaet Basel, Basel, Switzerland. He was a Fellow of the American Academy of Family Practice and a member of the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Meyer E. Golob, MD, New York. Died July 10, 1987; age 75. Dr Golob was a 1938 graduate of New York Medical College, New York. He was a Fellow of the American Psychiatric Association and a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Henry Abraham Gozan, MD, Coconut Creek, Florida. Died July 6, 1987; age 75. He was a 1935 graduate of State University of New York Health Science Center at Brooklyn. Dr Gozan was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the American Fertility Society, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Julius Hauer, MD, Brooklyn. Died July 5, 1986; age 86. Dr Hauer was a 1927 graduate of Deutsche Univerzita Medizinische Fakulta, Praha, Czechoslovakia. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Frank Manley Higgins, MD, Roswell, Georgia. Died July 11, 1987; age 84. Dr Higgins was a 1928 graduate of Queens University Faculty of Medicine, Kingston, Ontario, Canada. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Margot Hoffman, MD, San Jose, California. Died June 15, 1987; age 88. Dr Hoffman was a 1927 graduate of Schlesische-Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Breslau, Germany. Dr Hoffman was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Albert W. Horowitz, MD, North Miami Beach, Florida. Died June 5, 1987; age 76. He was a 1937 graduate of Universitaet Wien, Medizinische Fakultaet, Wien, Austria. Dr Horowitz was a Fellow of the American College of Surgeons and the American College of Obstetricians and Gynecologists, and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Harriet Hosmer, MD, Buffalo. Died July 13, 1987; age 91. She was a 1921 graduate of the Cornell University Medical College, New York. Dr Hosmer was a member of the Academy of Medicine, the American Academy of Family Practice, the American Medical Women's Association, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Matthew H. Jaffe, MD, Vestal. Died July 10, 1987; age 75. Dr Jaffe was a 1937 graduate of New York University School of Medicine, New York. He was a Diplomate of the American Board of Urology and a member of the American Urological Association, the Broome County Medical Society, and the Medical Society of the State of New York.

Samuel F. Jenkins, MD, New York. Died April 20, 1987; age 91. Dr Jenkins was a 1928 graduate of New York University School of Medicine, New York. He was a Diplomate of the American Board of Pediatrics and a member of the American Public Health Association, the New York County Medical Society, and the Medical Society of the State of New York.

Sanford Kaminester, MD, San Diego, California. Died June 3, 1987; age 83. He was a 1927 graduate of State University of New York Health Science Center at Brooklyn. Dr Kaminester was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, a Diplomate of the American Board of Obstetrics and Gynecoloy, and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Aaron Kelman, MD, Richmond Hill. Died July 5, 1987; age 69. He was a 1945 graduate of Middlesex University School of Medicine, Waltham, Massachusetts. Dr Kelman was a member of the American Rheumatism Association, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Lionel Klein, MD, Great Neck. Died May 21, 1987; age 74. He was a 1938 graduate of State University of New York Health Science Center at Brooklyn. Dr Klein was a Qualified Fellow of the International College of Surgeons. His memberships included the New York State Society of Occupational Medicine, the American Occupational Medical Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Adalbert Kroo, MD, New York. Died May 5, 1987; age 86. Dr Kroo was a 1929 graduate of Facolta di Medicina e Chirurgia dell'Universita di Napoli, Napoli, Italy. Dr Kroo was a member of the American Academy of Family Practice, the New York County Medical Society, and the Medical Society of the State of New York.

Harry George LaForge, MD, Buffalo. Died May 31, 1987; age 85. He was a 1934 graduate of State University of New York at Buffalo School of Medicine, Buffalo. Dr LaForge was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. His memberships included the Medical Society of the County of Erie and the Medical Society of the State of New York.

Tadeusz J. Lewandowski, MD, Snyder. Died June 2, 1987; age 74. Dr Lewandowski was a 1939 graduate of Akademia Medyczna Wydzial Lekarski, Poznan, Poland. Dr Lewandowski was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

William W. Marmar, MD, New York. Died April 15, 1987; age 87. Dr Marmar was a 1925 graduate of Tufts University School of Medicine, Boston. He was a member of the American Academy of Family Practice, the New York County Medical Society, and the Medical Society of the State of New York.

William F. Meinhardt, MD, Saratoga Springs. Died June 5, 1987; age 58. Dr Meinhardt was a 1953 graduate of University of Pennsylvania School of Medicine, Philadelphia. He was a Diplomate of the American Board of Internal Medicine and a member of the New York State Society of Internal Medicine, the Medical Society of the County of Saratoga, and the Medical Society of the State of New York.

Francis P. Melomo, MD, Brooklyn. Died May 31, 1987; age 81. He was a 1930 graduate of State University of New York Health Science Center at Brooklyn. Dr Melomo was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Saul L. Meylackson, MD, New York. Died May 18, 1987; age 96. Dr Meylackson was a 1914 graduate of Fordham University School of Medicine, New York. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Alfred Adam Mitchell, MD, Elmira. Died June 20, 1987; age 73. Dr Mitchell was a 1938 graduate of State University of New York at Buffalo School of Medicine, Buffalo. He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. He was a member of the Medical Society of the County of Chemung and the Medical Society of the State of New York.

James R. Montgomery, MD, Naples, Florida. Died June 15, 1987; age 82. Dr Montgomery was a 1931 graduate of Case Western Reserve University School of Medicine, Cleveland. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

George E. Murphy, MD, New York. Died July 16, 1987; age 69. Dr Murphy was a 1943 graduate of the University of Pennsylvania School of Medicine, Philadelphia. He was a member of the Academy of Medicine, the New York Pathological Society, the American Association of Pathologists and Bacteriologists, the American Rheumatism Association, the Society for Experimental Biology and Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Imre Ney, MD, Brooklyn. Died May 14, 1987; age 73. Dr Ney was a 1938 graduate of Facolta di Medicina e Chirurgia dell'Universita di Bologna, Bologna, Italy. Dr Ney was a Fellow of the American College of Anesthesiologists and a member of the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Vincent A. Parnell, MD, Bayside. Died July 13, 1987; age 72. He was a 1943 graduate of Creighton University School of Medicine, Omaha. Dr Parnell was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. His memberships included the Medical Society of the County of Queens and the Medical Society of the State of New York.

Abraham Harold Perman, MD, Bronx. Died June 15, 1987; age 83. Dr Perman was a 1935 graduate of Rush Medical College, Chicago. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

William Alfred Petry, MD, Rochester. Died June 17, 1987; age 81. He was a 1932 graduate of McGill University Faculty of Medicine, Montreal, Quebec, Canada. Dr Petry was a Diplomate of the American Board of Internal Medicine and a member of the Academy of Medicine, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Jerome Pincus, MD, Cedarhurst. Died July 23, 1987; age 66. He was a 1944 graduate of State University of New York Health Science Center at Brooklyn. Dr Pincus was a member of the New York Cardiological Society, the New York State Society of Internal Medicine, the American Geriatrics Society, the American Public Health Association, the American Society of Internal Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

Daniel F. Primeau, MD, Kenmore. Died May 27, 1987; age 60. He was a 1950 graduate of State University of New York at Buffalo School of Medicine, Buffalo. Dr Primeau was a Diplomate of the American Board of Obstetrics and Gynecology and a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Russell Elsworth Reitz, MD, New Hartford. Died May 22, 1987; age 72. He was a 1940 graduate of State University of New York at Buffalo School of Medicine, Buffalo. Dr Reitz was a Diplomate of the American Board of Ophthalmology. His memberships included the American Academy of Ophthalmology and Otolaryngology, the Pan American Association of Ophthalmology, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Andries I. Roodenburg, MD, Rochester. Died June 23, 1987; age 77. He was a 1938 graduate of Faculteit der Geneeskunde Universiteit van Amsterdam, Amsterdam, Netherlands. Dr Roodenburg was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the Academy of Medicine, the American Rheumatism Association, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Stephan F. Saxl, MD, New York. Died July 17, 1987; age 89. Dr Saxl was a 1923 graduate of Universitaet Wien Medizinische Fakultaet, Wien, Austria. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Joseph F. Shanaphy, MD, Staten Island. Died July 10, 1987; age 66. He was a 1945 graduate of State University of New York Health Science Center at Brooklyn. Dr Shanaphy was a Fellow of the American College of Surgeons and the American College of Obstetricians and Gynecologists, a Qualified Fellow of the International College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. His memberships included the American Fertility Society, the Richmond County Medical Society, and the Medical Society of the State of New York.

Harold L. Siegelbaum, MD, Suffern. Died July 3, 1987; age 65. He was a 1946 graduate of New York Medical College, New York. Dr Siegelbaum was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Medical Society of the County of Rockland and the Medical Society of the State of New York.

Victor Stern, MD, Westport, Connecticut. Died June 20, 1987; age 79. Dr Stern was a 1933 graduate of Universitaet Wien Medizinische Fakultaet Wien, Austria. He was a member of the American Geriatrics Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

S. Henry Thaler, MD, Bal Harbor, Florida. Died May 5, 1987; age 83. Dr Thaler was a 1931 recipient of registrable qualification granted by the Society of Apothecaries of London, England. Dr Thaler was a Qualified Fellow of the International College of Surgeons and a member of the Pan American Association of Ophthalmology, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Joseph C. Todoro, MD, Hamburg. Died May 22, 1987; age 65. He was a 1947 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. Dr Todoro was a Diplomate of the American Board of Ophthalmology. His memberships included the American Academy of Ophthalmology and Otolaryngology, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Burton W. Wilcke, MD, Rensselaer. Died May 28, 1987; age 78. Dr Wilcke was a 1935 graduate of University of Iowa College of Medicine, Iowa City. He was a member of the American Academy of Family Practice, the Medical Society of the County of Rensselaer, and the Medical Society of the State of New York.

Willard H. Willis, MD, New Hartford. Died May 29, 1987; age 77. He was a 1936 graduate of Ohio State University College of Medicine, Columbus. Dr Willis was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the New York State Society of Internal Medicine, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Charles C. Wolcott, MD, Bronxville. Died June 8, 1987; age 95. Dr Wolcott was a 1917 graduate of the University of Michigan Homeopathic Medical School, Ann Arbor. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Otolaryngology. His memberships included the American Academy of Otolaryngology; the American Broncho-Esophagological Association; the American Laryngological, Rhinological and Otological Society; the Medical Society of the County of Westchester; and the Medical Society of the State of New York.

NOTICE TO ALL PHYSICIANS

MSSNY SYMPOSIUM

"FOCUS ON WOMEN PHYSICIANS"

Saturday November 7, 1987
8:30 AM – 3:30 PM
(*Luncheon Included*)

Where: THE WARWICK HOTEL
54th Street & Avenue of the Americas
New York City: (212) 247-2700

Program Topics:

• BREAST CANCER	VICKI L. SELTZER, MD, *Author of Every Woman's Guide to Breast Cancer*
• CHALLENGES FACING WOMEN PHYSICIANS *. . . A Welcome to a Crowded Field*	JUDITH LORBER PhD, *Author of Women Physicians: Careers, Status & Power*
• AIDS	JOYCE WALLACE, MD, discusses her Greenwich Village practice, consisting largely of AIDS patients (more and more of whom are women)
• WORKING IN ORGANIZED MEDICINE	PALMA FORMICA, MD, President-Elect, Medical Society of New Jersey
• WOMEN PHYSICIANS AND STRESS: *Patterns of Drug and Alcohol Abuse from Medical School to Retirement*	MATILDA RICE, MD, Assistant Clinical Professor of Psychiatry, SUNY HSC, Stony Brook
• LIFESTYLE PANEL	Women physicians look at lifestyle issues from different perspectives, including dual physician marriages and marriages with non-physicians

CME Credit: Application has been made for 6 Category I CME credits.

Registration Fees:

Members of the MSSNY	$50
Nonmembers	$65
Student or resident members of the MSSNY	$30
Nonmember students or residents	$40

MSSNY will refund the difference between the member and nonmember registration fee to any registrant whose completed membership application and dues payment are received by the symposium date.

TO REGISTER, Please complete the form on the reverse side of this page.

REGISTRATION FORM

Please complete and return to:

FOCUS ON WOMEN PHYSICIANS

Division of Membership Support Services
Medical Society of the State of New York
420 Lakeville Rd, Lake Success, NY 11042
(516) 488-6100, Ext. 252

NAME (please print) ____

ADDRESS ____

CITY ______________________ **STATE** ________________ **ZIP** __________

PHONE NUMBER (   ) ____________________

Enclosed is a check made payable to MSSNY in the amount checked below:

(   ) $30—Medical student member of MSSNY

(   ) $30—Resident member of MSSNY and ______________ County Medical Society

(   ) $40—Nonmember student or resident

(   ) $50—Other MSSNY member (through ______________ County Medical Society)

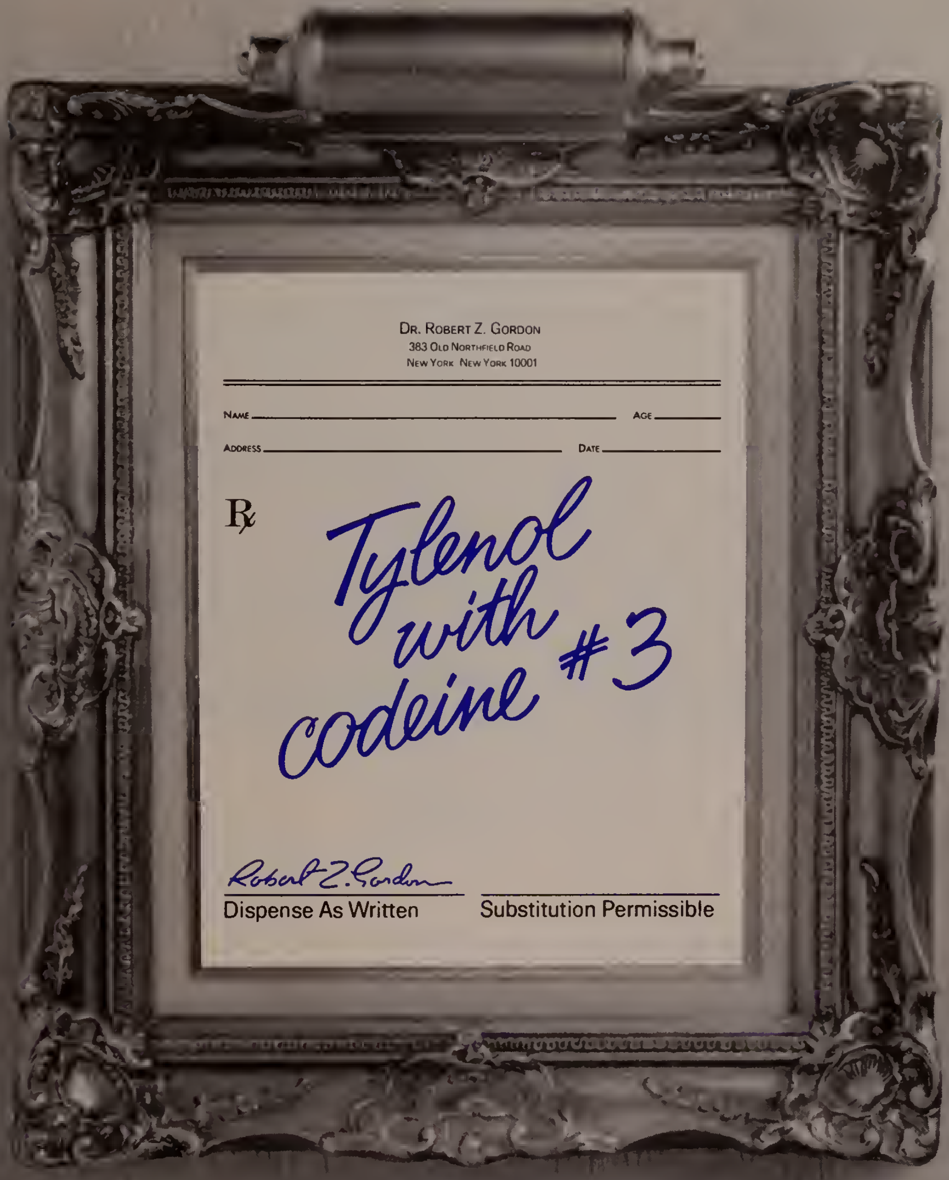
(   ) $65—Other nonmember

(   ) Please send me membership information and an application form for membership through ______________ County Medical Society

MSSNY WILL REFUND THE DIFFERENCE BETWEEN THE MEMBER AND NONMEMBER REGISTRATION FEE FOR ANY REGISTRANT WHOSE COMPLETED MEMBERSHIP APPLICATION FORM AND DUES PAYMENT ARE RECEIVED BY THE SYMPOSIUM DATE

(   ) I cannot attend at this time. Please keep me informed of future programs.

REFUNDS WILL BE GIVEN IF REQUESTED UP TO ONE WEEK PRIOR TO SYMPOSIUM



DR. ROBERT Z. GORDON
383 OLD NORTHFIELD ROAD
NEW YORK NEW YORK 10001

NAME ____________ AGE ______

ADDRESS ____________ DATE ______

℞

Tylenol
with
codeine #3

Robert Z. Gordon

Dispense As Written

Substitution Permissible

CREATE A SIGNED ORIGINAL

to be sure your patients get...

Tylenol®

ACETAMINOPHEN
with codeine
phosphate
TABLETS Ⓒ III ELIXIR Ⓒ V

Tablets: Contain Codeine Phosphate*: No. 3— 30 mg;
No. 4—60 mg—plus Acetaminophen 300 mg.
Elixir: Each 5 mL contains 12 mg Codeine Phosphate* plus
120 mg Acetaminophen (Alcohol 7%).
*Warning: May be habit forming.

© McNEILAB, INC. 1987

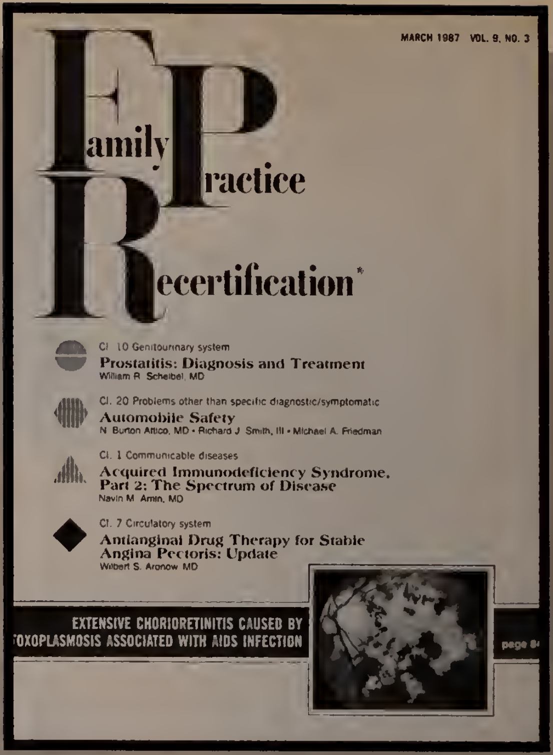
TYCT 6445-A

The complete journal for family practice physicians

- Reaches 79,000 family physicians monthly
- Presents the most commonly seen patient problems in family practice
- Written by physicians for physicians
- The most current clinical updates in:

Cardiology	Ob/Gyn
Diabetes	Psychiatry
Pediatrics	Gastroenterology

- Provides 20 hours of CME Category 1 Credit



MARCH 1987 VOL. 9, NO. 3

Family Practice Recertification*

Cl 10 Genitourinary system
Prostatitis: Diagnosis and Treatment
William R Scheibel, MD

Cl. 20 Problems other than specific diagnostic/symptomatic
Automobile Safety
N Burton Attico, MD • Richard J Smith, III • Michael A. Friedman

Cl. 1 Communicable diseases
Acquired Immunodeficiency Syndrome, Part 2: The Spectrum of Disease
Navin M Amin, MD

Cl. 7 Circulatory system
Antianginal Drug Therapy for Stable Angina Pectoris: Update
Wilbert S. Aronow MD

EXTENSIVE CHORIORETINITIS CAUSED BY OXOPLASMOSIS ASSOCIATED WITH AIDS INFECTION

page 8

PRACTICAL ▪ CLINICAL ▪ EDUCATIONAL ▪ CURRENT

(*continued from p 4A*)

FLORIDA

Nov 12–15. **Flexible Fiberoptic Sigmoidoscopy in the Diagnosis of Colorectal Diseases.** 15 Cat 1 Credits. Buena Vista Palace, Lake Buena Vista. Contact: Seminars & Symposia, Inc, 132 East 70th Street, New York, NY 10021. Tel: (212) 517-7520.

Nov 13–14. **The Distal Radioulnar Joint—Problems and Procedures.** 12¼ Cat 1 Credits. Hotel Royal Plaza, Lake Buena Vista. Contact: American Society for Surgery of the Hand, 3025 Parker Rd, Suite 65, Aurora, CO 80014. Tel: (303) 755-4588.

ILLINOIS

Nov 5–7. **11th National Conference on Correctional Health Care.** Palmer House and Towers, Chicago. Contact: Jodie Manes, National Commission on Correctional Health Care, 2000 North Racine, Suite 3500, Chicago, IL 60614. Tel: (312) 528-0818.

MARYLAND

Nov 9–11. **Recent Advances in the Biomedical Significance of TRH.** Hyatt Regency Hotel, Bethesda. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd Street, New York, NY 10021.

MASSACHUSETTS

Nov 2–5. **Broadening the Skills of the General Care Internist, VI.** Ritz-Carlton Hotel, Boston. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

Nov 4–6. **Gynecologic Microsurgery and Laser.** 21 Cat 1 Credits. University of Massachusetts Medical Center, Worcester. Contact: Registrar, The American College of Obstetricians and Gynecologists, 600 Maryland Ave SW, Suite 300 East, Washington, DC 20024-2588. Tel: 1-800-633-8811.

AROUND THE WORLD

MEXICO

Nov 30–Dec 2. **Infertility and Reproductive Endocrinology.** 16 Cat 1 Credits. Acapulco Princess, Acapulco. *Dec 3–5.* **Advanced Colposcopy.** Acapulco Princess, Acapulco. Contact: American College of Gynecologists, Registrar, 600 Maryland Ave SW, Suite 300 East, Washington, DC 20024-2588. Tel: 1-800-533-8811.

SINGAPORE

Nov 9–14. **First International Congress on Oral Cancer and Jaw Tumors.** Singapore. Contact: Dr N. Ravindranthan, Organizing Secretary, First International Congress on Oral Cancer and Jaw Tumors, Department of Oral Surgery, Faculty of Dentistry, National University of Singapore, Kent Ridge, Singapore 0511. □

Lactaid. Real Milk for Lactose Intolerant Patients.

LACTOSE THE PROBLEM. If your patients drink milk and have indigestion, gas or diarrhea even up to twelve hours afterwards, it may be due to the lactose in milk and dairy foods. One out of five people in the United States may have this problem.

LACTAID THE SOLUTION. Lactaid "Specially Digestible" lactose reduced milk is great tasting lowfat milk with all the protein, calcium and nutritional benefits. With Lactaid, your patients can continue or resume real milk consumption without later discomfort since the lactose is converted into simple sugars which are fully digestible.

DAIRY FOODS ENJOYED ANYTIME—ANYWHERE. Prescribe one to two Lactaid Tablets with any other milk or dairy food, and let your patients enjoy their favorite foods without discomfort. Lactaid Liquid Enzyme can be added to a quart of any milk to hydrolyze up to 99 percent of the lactose if desired..

Lactaid®

For more information on Lactaid products — or to receive your FREE Lactaid Patient Starter Kit including brochures and product samples — call our Lactaid Hotline at:

1-800-257-8650

Monday–Friday, 9 a.m.–4 p.m.

EMERGENCY! MEDICAL CLAIMS OVERLOAD!

The EMC*EXPRESS™ *System*

TO THE RESCUE!

If claims processing paperwork can get your office all backed up, you need to call in GE Information Services' EMC*EXPRESS rescue squad.

The **EMC*EXPRESS System** is a computerized medical claims system which enables you to deliver medical claims to participating carriers within hours of treating a patient. One phone call is all it takes to have your computer speed all the information needed to process your claims to **multiple** carriers, including Medicare, commercial and private.

Working with the software already in your office, the **EMC*EXPRESS** System helps you:

- Reduce your administrative costs for processing claims
- Reduce the number of claims rejected because of incorrect or incomplete information
- Speed up your claims payment—**and that can put new life into your cash flow.**

The **EMC*EXPRESS** System and Your Office

The EMC*EXPRESS System accommodates a broad range of practice sizes and a wide variety of office computer systems, from easy-to-use claims entry systems to the most sophisticated practice management systems.

So whatever the size of your office, whatever the magnitude of your paperwork problem, we recommend the **EMC*EXPRESS** System as your treatment of choice.

For more than 20 years, GE Information Services has been helping companies around the world do business better. And now GE has teamed up with authorized system vendors to bring rapid, accurate, and cost-effective electronic medical claims communications to the healthcare field.

To have the EMC* EXPRESS rescue squad come to your aid, call an authorized EMC*EXPRESS system vendor or contact us directly at 1-800-433-3683, extension 7321. We'll send help your way before you can say, "Emergency!"

INFORMATION SERVICES

USA

The
EMC*EXPRESS™
System

PARTICIPATING CARRIERS

Empire Blue Cross and Blue Shield
Medicare B and Private Business Claims
Metropolitan Life Insurance Co.
(Includes Empire NY State Employees)

NEIC ACTIVE PARTICIPANTS

Aetna Life & Casualty
Allstate Life Insurance Co.
Benefit Trust Life
CNA Insurance Companies
Confederation Administration Life Insurance Co.
Confederation Life Insurance Co.
Connecticut Life Insurance Co.
Connecticut General Life Insurance Co. (CIGNA)
Equitable Life Assurance Society of the U.S.
General American Life Insurance Co.
The Great-West Life Assurance Co. of America
Gulf Group Services
The Hartford Insurance Group
John Hancock Mutual Life Insurance Co.
Liberty Life Assurance Co.
Lincoln National Life Insurance Co.
Massachusetts Mutual Life Insurance Co.
Metropolitan Life Insurance Co.
Mutual of Omaha Insurance Co.
New England Mutual Life Insurance Co.
New York Life Insurance Co.
Pacific Mutual Life Insurance Co.
Philadelphia American Life Insurance Co.
Philadelphia Life Insurance Co.
Phoenix Mutual Life Insurance Co.
Pilot Life Insurance Co.
Principal Mutual Financial Group
Provident Life and Accident Insurance Co.
Prudential Insurance Co.
State Mutual Insurance Co.
The Travelers Insurance Co.
(includes Railroad Retirement)
Unionmutual Stock Life Insurance Co.

AUTHORIZED SYSTEM VENDORS

Annson Systems, Inc.	312 564-8310
Articulate Publications, Inc.	800 872-2282
Artificial Intelligence, Inc.	206 271-8633
CALYX	800 558-2208
Colwell Systems, Inc.	800 637-1140
Coopervision	800 772-CVIS
EDP Systems, Inc.	214 881-8454
Health America Systems	312 362-3730
LDS, Inc.	913 648-7111
MOS, Inc.	800 323-6671
Physicians' Micro Systems, Inc.	206 441-8490
Physicians' Office Computer	213 603-0555
Prism Data Systems	800 428-2310
ProSource Systems	800 645-5609
Santiago Data Systems, Inc.	800 652-3500
UNIVAIR, Inc.	314 426-1099
Westland Software House, Inc.	818 992-0081

NEW VENDORS AND CARRIERS ADDED REGULARLY. FOR MOST RECENT LISTINGS, CALL 1-800-433-3683.

INFORMATION SERVICES
USA

401 N. WASHINGTON ST., ROCKVILLE, MD 20850
NYSJM1087

SIGNATURE LOANS—$5,000 to $60,000

DOCTORS ONLY. No collateral, simple interest, 6 years to repay. No points, no fees, no prepay penalties. Use for any reason. Residents, D.O.'s, start-up welcome. For appointment and info call:

(NY) NORTHSTAR FUNDING (212) 323-8076
or MEDIVERSAL 1-800-331-4952 Dept. #135

EQUIPMENT LEASING—$10K-$10 MILLION
NORTHSTAR FUNDING (212) 323-8076 Apply by Phone

REAL ESTATE FOR SALE OR RENT—CONT'D

LOOKING FOR A GOOD PROFESSIONAL LOCATION? Set up your office in prestigious North Syosset. Bring the family with you to this large center hall colonial with new country kitchen and baths. Separate entry to office, new circular driveway. Many extras SD#2 walk to all. $379,000. Pat Randazzo, Coldwell Banker, (516) 364-6460.

FOR RENT: Medical Office Kew Gardens, suitable for group or solo practice. Completely furnished and equipped, spacious, Picker x-ray, physical therapy treatment rooms. Separate wing of private house, tree lined street, excellent neighborhood. Retired Orthopedist, phone (718) 849-8844.

DOCTORS OFFICE FOR RENT. Fully furnished and equipped. East 3rd Street, Cortelyou Road Brooklyn, 2 blocks away from Ocean Parkway. No doctor in immediate area. Great future, also apartment above the office available. Call (718) 436-6751.

MISCELLANEOUS

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuarial certification and employee statements of participation—partnership agreements and professionals incorporation—No insurance required—references upon request—Wachstock and Dienstag Attorneys at Law, 122 Cutter Mill Road, Great Neck, NY 11021 (516) 773-3322.

1988 CME CRUISE/CONFERENCES on Medicolegal Issues & Risk Management—Caribbean, Mexico, Alaska, China/Orient, Europe, New England/Canada, Trans Panama Canal, South Pacific. Approved for 24-28 CME Cat. 1 Credits (AMA/PRA) and AAFP prescribed credits. Distinguished lecturers. Excellent Group Rates on Finest Ships. Registration limited. Prescheduled in compliance with IRS requirements. Information: International Conferences, 189 Lodge Ave., Huntington Station, NY 11746. (516) 549-0869.

MISCELLANEOUS—CONT'D

HAVE YOU RECEIVED ANY COMMUNICATION FROM THE OFFICE OF PROFESSIONAL MEDICAL CONDUCT? If affirmative, contact Susan Kaplan, Attorney-at-Law, (212) 877-5998. Practice limited to the crucial legal problems directly affecting your license to practice medicine—with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professions), and as an Assistant District Attorney in Nassau County. Susan Kaplan, Esq., 165 West End Avenue, Suite 27P, New York, N.Y. 10023.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta, GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

ADVICE, COUNSEL & REPRESENTATION of physicians and other health care professionals in licensure and professional conduct matters, audit preparations, reimbursement issues and practice-related litigation. David E. Ruck, Esq., former Chief, Criminal Division, Office of the New York State Special Prosecutor for Medicaid Fraud Control and Alain M. Bourgeois, Esq., former Acting Justice, Supreme Court of the State of New York. Bourgeois & Ruck, 101 Park Avenue, New York, New York 10178. Telephone 212-661-8070.

PORTABLE RADIOLOGY SERVICE: abdominal + pelvic ultrasound, cardiac echos including 2D + m-mode portable x-rays and portable osteoporosis bone densitometry screening. NYC and suburbs. Schedule weekly/bi-weekly in your office or visits to your patient's home. New portable hi-tech equipment. Hi-quality like standard office images, far superior to hospital portable images. No lead shielding needed for x-rays. University Radiology Service, (212) 265-7392 or (718) 339-5363.

PROFESSIONAL MISCONDUCT ATTORNEYS. William L. Wood, Jr., formerly Executive Director of the New York State Office of Professional Discipline and Anthony Z. Scher, formerly Director of Prosecutions. Our recent tenure as chief enforcement officers for the regulation of New York State's one-half million licensed professionals has given us experience which allows us to represent physicians in professional misconduct proceedings, malpractice, license restoration, controlled drug proceedings, insurance company reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practitioners. Wood & Scher, Attorneys at Law, One Chase Road, Scarsdale, New York 10583. Telephone (914) 723-3500.

Added Control For
America's Coughs

The expectorant action of guaifenesin plus the
Advantage of Codeine!

Robitussin and codeine. Each 5 ml (*1 teaspoonful*) contains: Guaifenesin, USP, 100 mg; Codeine Phosphate, USP, 10 mg (*warning: may be habit forming*); alcohol, 3.5 percent; in a palatable, aromatic syrup

A·H·ROBINS

Pharmaceutical Division, Richmond, Virginia 23261-6609 ©*A.H Robins Company 1985*

Robitussin
A-C®Ⓒ

TIME to subscribe

Consider a few features of
The Western Journal of Medicine:

- **Epitomes of Progress** — Each month this section highlights the major advances in a different specialty with 15 or 20 brief epitomes by experts in the field.
- **Clinical Investigation** — Five major clinical research societies coordinate important new research findings, emphasizing clinical value.
- **Socioeconomics** — WJM is often the first to predict, examine and evaluate socioeconomic changes, trends.
- **Yearly Special Issue** — Each December's issue is devoted to a topic vital to physicians: clinical nutrition (1979), cost of care (1980), geriatrics (1981), occupational/environmental (1982), cross-cultural medicine (1983), personal health maintenance (1984), high-tech medicine (1985), medical informatics (1986).

NYSJM 10/87

Enclosed is $______ to cover ______ subscriptions.
Subscription renewal ☐ Regular subscription ☐
Foreign subscription ☐

TO: ____________________

Street ____________________

City/State/Zip ____________________

Specialty ____________________

Gift Card to read "From ____________________"

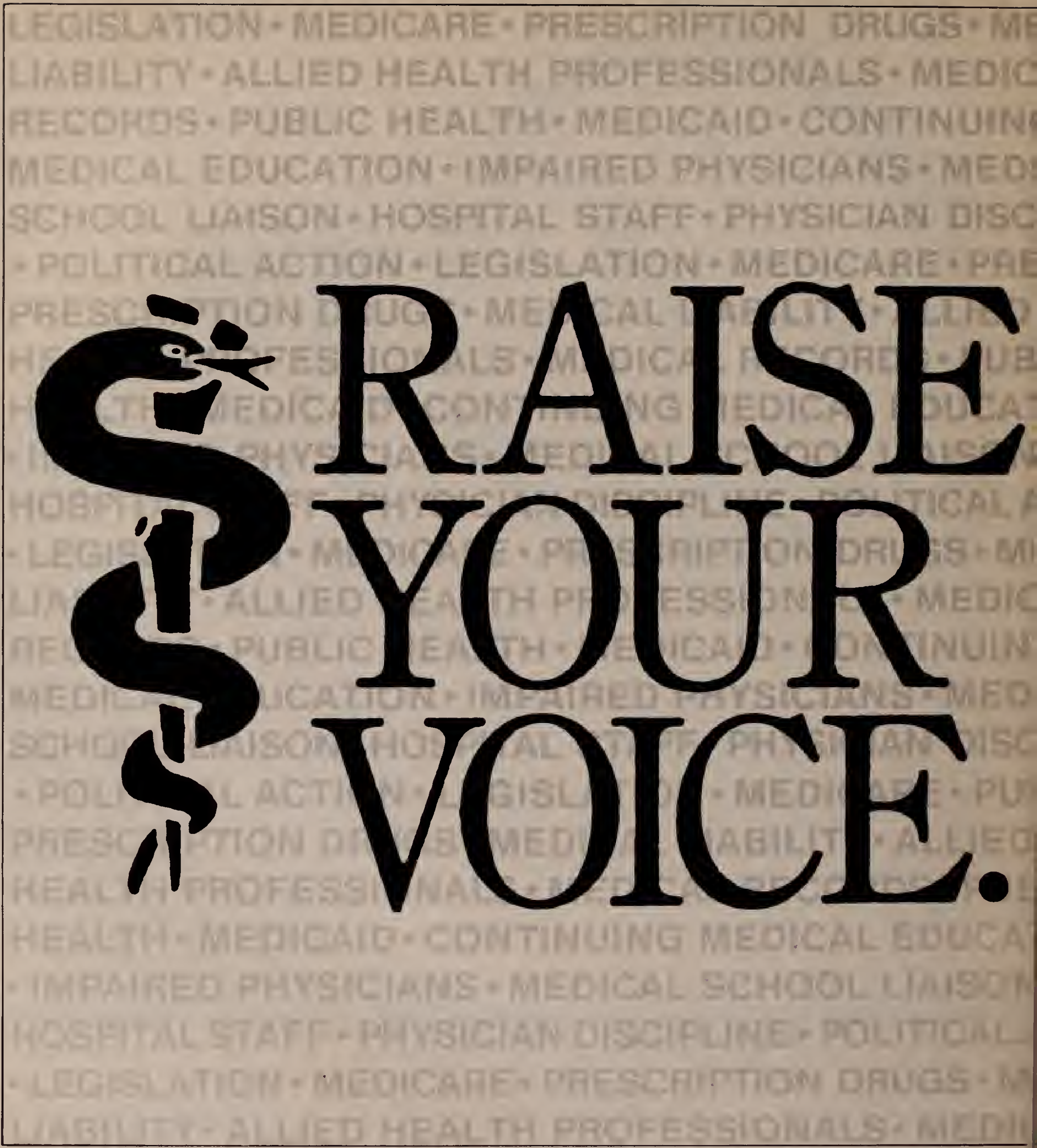
The Western Journal of Medicine

Please mail this coupon with
enclosed payment to:

Circulation Department
P.O. Box 7602, San Francisco,
California 94120-7602

Subscriptions, 12 issues Per Year
U.S. and Canada $30 1 year,
$50 2 years

All other countries $50 1 year,
$90 2 years



N Y M P A C

New York Medical Political Action Committee
An Arm of the Medical Society of the State of New York
420 Lakeville Road, Lake Success, NY 11042 • 516-488-6100

Political Action for Physician

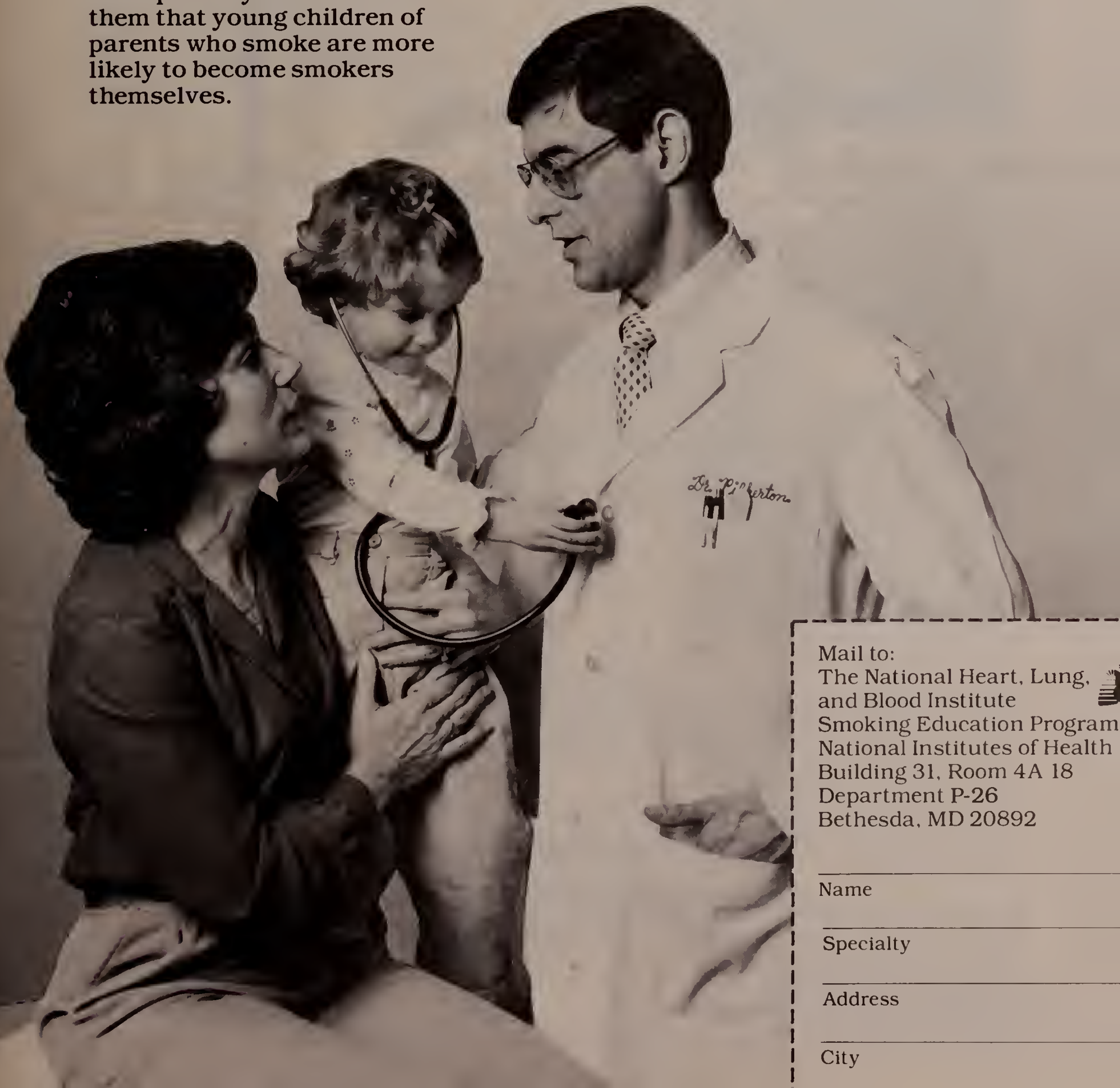
A Clinical Opportunity for Smoking Intervention

You can play a special role in reaching smokers. Encouraging parents not to smoke can improve the health of the entire family.

Take a few minutes to explain that children of parents who smoke are often more prone to respiratory infections. Tell them that young children of parents who smoke are more likely to become smokers themselves.

The minutes you spend can make a difference now, and in the years ahead.

For a free copy of *Clinical Opportunities for Smoking Intervention: A Guide for the Busy Physician*, complete the form below.



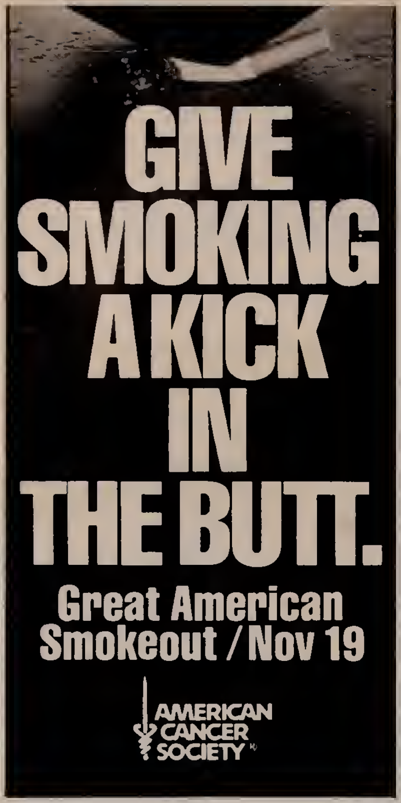
Mail to:
The National Heart, Lung,
and Blood Institute
Smoking Education Program
National Institutes of Health
Building 31, Room 4A 18
Department P-26
Bethesda, MD 20892

Name

Specialty

Address

City


GIVE SMOKING A KICK IN THE BUTT.
Great American Smokeout / Nov 19
AMERICAN CANCER SOCIETY

MISCELLANEOUS—CONT'D

SIGNATURE LOANS—$5,000 to $60,000 doctors only. No collateral, simple interest, 4, 5, or 6 year terms. No points, no fees, no prepay penalties. No use restrictions. Residents, D.O.'s, new practitioners welcome. For application and info call: (NY) Northstar Funding (212) 323-8076 or Mediversal 1-800-331-4952 Dept. #135.

PROFESSIONAL MISCONDUCT ADVOCACY with dignity and concern. George Weinbaum, Esq. former deputy director of the New York State Attorney General's office for Medicaid Fraud Control and also former Director of Medicare and private insurance investigations for Empire Blue Cross/Blue Shield is pleased to announce the opening of his office for the practice of health law. Attention is devoted to misconduct avoidance, disciplinary proceedings, reimbursement maximization, controlled drug issues and medical staff representation among other physician needs. George Weinbaum, Esq., 3 Barker Avenue, White Plains, New York 10601. Telephone (914) 686-9310 office hours, (212) 621-7776-24 hours.

LEASE YOUR NEXT AUTOMOBILE—from the people who know their profession and yours. Just as your patients seek the advice of a professional, so should you! All leases are custom tailored to your needs. A wide variety of services are available. Our rates are highly competitive. We can make the acquisition of your next vehicle inexpensive, swift and easy. All makes and models leased. Madison Leasing Corp., 1983 Marcus Ave., Lake Success, NY 11042, (516) 775-5200.

EB TRANSCRIPTION SPECIALISTS offering expert work with professional service to fit your needs. All medical specialties. Letters and correspondence, patients charts and manuscript typing. Fast turn around time, free pick up and delivery in the New York area and competitive rates. For additional information call Emily at (718) 849-1987.

Doctor . .

Do you, or a physician you know, have a problem with alcohol, drugs, or emotional illness?

If so, please contact the Medical Society of the State of New York's Committee for Physicians' Health, whose function is to assist, *confidentially*.

Telephone: 516-488-7777.

Index to Advertisers

AMA Advisers, Inc.	5A
St. Clare's Hospital	1A
Classified Advertising	6A, 8A, 19A, 24A
Family Practice Recertification Magazine	16A
General Electric Information Services	18A, 19A
Knoll Pharmaceuticals	3A, 4A
Lactaid	17A
Eli Lilly and Company	10A
McNeil Laboratories	15A
Milstein Properties Corp.	8A
A. H. Robins Company	7A, 20A
Roche Laboratories	3rd & 4th Cover
Sarantos and Company, Inc.	8A
Upjohn Company	2nd Cover

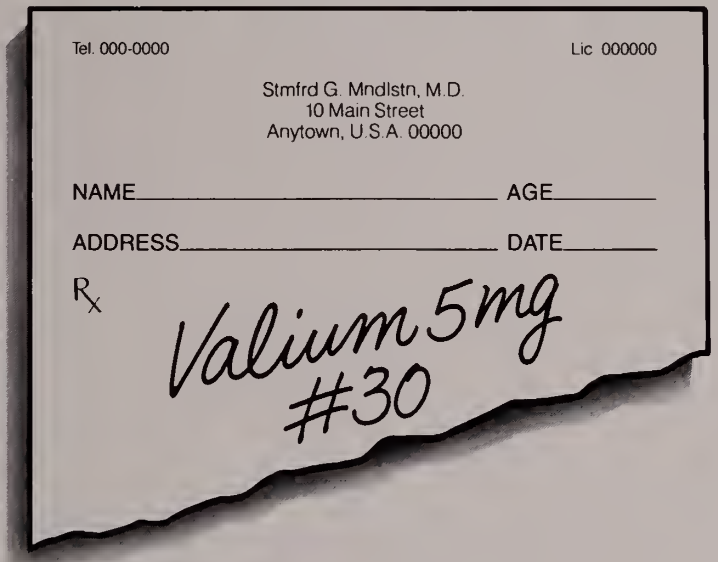
Do Not Substitute

VALIUM®

brand of
diazepam/Roche Ⓘⓥ

2 mg 5 mg 10 mg

The One You Know Best


Tel. 000-0000 Lic 000000

Stmfrd G. Mndlstn, M.D.
10 Main Street
Anytown, U.S.A. 00000

NAME__________ AGE______

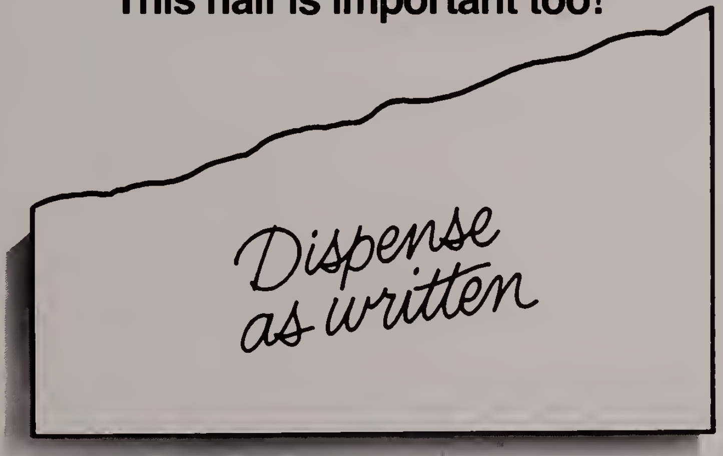
ADDRESS__________ DATE______

℞

Valium 5mg
#30

Are you writing only half a prescription for it?

This half is important too!


Dispense
as written

Be sure to write a complete prescription.

Specify "Dispense as written."

ROCHE

Roche Products Inc.
Manati, Puerto Rico 00701

Copyright © 1987 by Roche Products Inc.
All rights reserved.

State flag of New York

Flag It.

To complete your prescription,
be sure to specify
"Dispense As Written."
This "flags" both pharmacist and patient
that you want the *brand* to be dispensed.
And it protects your decision.

VALIUM®
brand of
diazepam/Roche Ⓒ IV

scored tablets

2 mg 5 mg 10 mg

The one you know best.

The cut out "V" design is a registered trademark
of Roche Products Inc.

ROCHE®

STACKS

NEW YORK STATE JOURNAL OF MEDICINE

NOVEMBER 1987 • Volume 87, Number 11

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA
M. D. S.
NOV 13 1987

Contents

COMMENTARIES

A view of the proposed New York State regulations governing the professional activities of residents 587
ASSOCIATED MEDICAL SCHOOLS OF NEW YORK

Artificial breathing support systems: Preferred nomenclature for medicine and the courts 589
ROBERT D. BRANDSTETTER, MD, FRANK TAMARIN, MD

Withdrawing fluids and nutrition: An alternate view 591
FRED ROSNER, MD

RESEARCH PAPER

Incidence of placenta previa and abruptio placentae in New York State 594
ELINA HEMMINKI, MD; DONNA M. GLEBATIS, MS; GENE D. THERRIAULT, MSPH; DWIGHT T. JANERICH, DDS

THE CPC

Myelofibrosis, renal vein thrombosis, and pulmonary emboli 599

READERSHIP SURVEY

Readership survey of the *New York State Journal of Medicine* 606
PASCAL JAMES IMPERATO, MD

SPECIAL ARTICLE

A response to the recommendations of the New York State Commission on Graduate Medical Education concerning teaching in ambulatory care settings 613
ELEANOR Z. WALLACE, MD; LOUIS M. ALEDORT, MD; RICHARD LEVERE, MD; ALVIN I. MUSHLIN, MD

CASE REPORTS

Stillbirth following maternal Lyme disease 615
ALAN B. MacDONALD, MD; JORGE L. BENACH, PhD; WILLY BURGDORFER, PhD

Visual loss and deafness associated with meningeal carcinoma 616
MICHAEL GAROFALO, JR, MD; CAROLYN MARTIN, MD; MICHAEL BYKOFSKY, MD; JOSEPH CASARONA, MD

Microscopic bilateral adenocarcinoma arising in ovarian endometrial cysts 617
MARY F. A. C. DiMAIO, MD; ELLEN KAHN, MD; A. FENTON, MD

LETTERS TO THE EDITOR

Variations in energy expenditure during physical exercise 621
GREGG S. MEYER, MD, VERNON REYNOLDS, PhD

House staff supervision and working hours 621
EMANUEL E. GARCIA, MD
STANLEY L. GORDON, MD

Minilaparotomy: An approach to postpartum tubal sterilization 622
JOHN M. ESPOSITO, MD; ANIELL T. MASTELLONE, MD; GEORGE RUBINO, MD

POSTER CONTEST 623

LEADS FROM EPIDEMIOLOGY NOTES 625

BOOK REVIEWS 629
BOOKS RECEIVED 633
NEWS BRIEFS 634
OBITUARIES 636
MEDICAL MEETINGS AND LECTURES 12A

Annual CME Assembly, April 22–24, 1988, New York Hilton, New York City
(See January 1988 issue for Preliminary Program)

Motrin® 800 TABLETS mg
ibuprofen

Extra strength
Convenience
Economy

Upjohn
100
A Century
of Caring
1886·1986

©1986 The Upjohn Company

J-6138 January 1986

News from DISTA about a new dosage form of cephalexin

ANNOUNCING NEW

Keflet®

TABLETS

cephalexin

All the advantages of cephalexin in a convenient tablet form

- Backed by over 15 years of clinical experience
- Smaller tablet is specially shaped and coated for easier swallowing
- May enhance patient compliance, particularly among the elderly
- Tablet dosage form may be appreciated by patients of all ages

NEW Keflet Tablets are available as:

250-mg Tablets

500-mg Tablets

Keflet is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-sensitive patients.

© 1987, DISTA PRODUCTS COMPANY KX-9003-B-849326

Brief Summary. Consult the package literature for prescribing information. Indications and Usage: Keflet® Tablets (cephalexin, Dista) are indicated for the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

Respiratory tract infections caused by *Streptococcus pneumoniae* and group A β-hemolytic streptococci (Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. Keflet is generally effective in the eradication of streptococci from the nasopharynx; however, substantial data establishing the efficacy of Keflet in the subsequent prevention of rheumatic fever are not available at present.)

Otitis media due to *S pneumoniae, Haemophilus influenzae*, staphylococci, streptococci, and *Neisseria catarrhalis*

Skin and skin structure infections caused by staphylococci and/or streptococci

Bone infections caused by staphylococci and/or *Proteus mirabilis*

Genitourinary tract infections, including acute prostatitis, caused by *Escherichia coli, P mirabilis*, and *Klebsiella* sp.

Note—Culture and susceptibility tests should be initiated prior to and during therapy. Renal function studies should be performed when indicated.

Contraindication: Keflet is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: BEFORE CEPHALEXIN THERAPY IS INSTITUTED, CAREFUL INQUIRY SHOULD BE MADE CONCERNING PREVIOUS HYPERSENSITIVITY REACTIONS TO CEPHALOSPORINS AND PENICILLIN. CEPHALOSPORIN C DERIVATIVES SHOULD BE GIVEN CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS.

SERIOUS ACUTE HYPERSENSITIVITY REACTIONS MAY REQUIRE EPINEPHRINE AND OTHER EMERGENCY MEASURES.

There is some clinical and laboratory evidence of partial cross-allergenicity of the penicillins and the cephalosporins. Patients have been reported to have had severe reactions (including anaphylaxis) to both drugs.

Any patient who has demonstrated some form of allergy, particularly to drugs, should receive antibiotics cautiously. No exception should be made with regard to Keflet.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening.

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C difficile*. Other causes of colitis should be ruled out.

Usage in Pregnancy—Safety of this product for use during pregnancy has not been established.

Precautions: *General*—Patients should be followed carefully so that any side effects or unusual manifestations of drug idiosyncrasy may be detected. If an allergic reaction to Keflet occurs, the drug should be discontinued and the patient treated with the usual agents (eg, epinephrine or other pressor amines, antihistamines, or corticosteroids).

Prolonged use of Keflet may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug.

Keflet should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

Indicated surgical procedures should be performed in conjunction with antibiotic therapy.

As a result of administration of Keflet, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy—Pregnancy Category B—The daily oral administration of cephalexin to rats in doses of 250 or 500 mg/kg prior to and during pregnancy, or to rats and mice during the period of organogenesis only, had no adverse effect on fertility, fetal viability, fetal weight, or litter size. Note that the safety of cephalexin during pregnancy in humans has not been established.

Cephalexin showed no enhanced toxicity in weanling and newborn rats as compared with adult animals. Nevertheless, because the studies in humans cannot rule out the possibility of harm, Keflet should be used during pregnancy only if clearly needed.

Nursing Mothers—The excretion of cephalexin in the milk increased up to 4 hours after a 500-mg dose; the drug reached a maximum level of 4 μg/mL, then decreased gradually, and had disappeared 8 hours after administration. Caution should be exercised when Keflet is administered to a nursing woman.

Adverse Reactions: *Gastrointestinal*—Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely. The most frequent side effect has been diarrhea. It was very rarely severe enough to warrant cessation of therapy. Dyspepsia and abdominal pain have also occurred. As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely.

Hypersensitivity—Allergic reactions in the form of rash, urticaria, angioedema, and, rarely, erythema multiforme, Stevens-Johnson Syndrome, or toxic epidermal necrolysis have been observed. These reactions usually subsided upon discontinuation of the drug. Anaphylaxis has also been reported.

Other reactions have included genital and anal pruritus, genital moniliasis, vaginitis and vaginal discharge, dizziness, fatigue, and headache. Reversible interstitial nephritis has been reported rarely. Eosinophilia, neutropenia, thrombocytopenia, and slight elevations in SGOT and SGPT have been reported.

PV 2211 DPP

[111786]

Additional information available to the profession on request from

DISTA

Dista Products Company
Division of Eli Lilly and Company
Indianapolis, Indiana 46285
Mfd by Eli Lilly Industries, Inc
Carolina, Puerto Rico 00630

NEW YORK STATE
JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
JOHN T. PRIOR, MD
ELIZA H. CALDWELL, MD
GITA S. SINGH*
JOSEPH F. MURATORE, MD
STANFORD WESSLER, MD

*Medical student

Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Consulting Editor and Book Review Editor	RICHARD B. BIRRER, MD
Consulting Editor	NAOMI R. BLUFSTONE, MD, MPH
Consulting Editor	CARL POCHEDLY, MD
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Managing Editor	CAROL L. MOORE
Advertising Director	ROBERT MILLER
Consulting Medical Writer	VICKI GLASER
Editorial Assistant	MILDRED J. AREFMANN
Secretary	ELIZABETH J. SOMERS
Librarian	ELLA ABNEY
Assistant Librarian	ELEANOR BURNS

ASSOCIATE EDITORIAL BOARD 1987

MICHAEL E. BERLOW, MD
RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MYLES S. GOMBERT, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
JAMES M. MORRISSEY, ESQ
STEPHEN NORDLICHT, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. United Media Associates, Inc, 16 Bruce Park Ave, Greenwich, CT 06830. Telephone: (203) 661-9702.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

SAMUEL M. GELFAND, MD,	*President*
JOHN A. FINKBEINER, MD,	*Past-President*
CHARLES D. SHERMAN, JR, MD,	*President-Elect*
DAVID M. BENFORD, MD,	*Vice-President*
JOHN H. CARTER, MD,	*Secretary*
GEORGE LIM, MD,	*Assistant Secretary*
MORTON KURTZ, MD,	*Treasurer*
ROBERT A. MAYERS, MD,	*Assistant Treasurer*
CHARLES N. ASWAD, MD,	*Speaker*
SEYMOUR R. STALL, MD,	*Vice-Speaker*

Councilors

Term Expires 1988

JAMES H. COSGRIFF, JR, MD,	Erie
RICHARD A. HUGHES, MD,	Warren
ANTONIO F. LASORTE, MD,	Broome
SIDNEY MISHKIN, MD,	Nassau

Term Expires 1989

ROBERT E. FEAR, MD,	Suffolk
STANLEY L. GROSSMAN, MD,	Orange
THOMAS D. PEMRICK, MD,	Rensselaer
RALPH E. SCHLOSSMAN, MD,	Queens

Term Expires 1990

STUART I. ORSHER, MD, (*elected to serve until 1988*)	New York
DUANE M. CADY, MD,	Onondaga
WILLIAM A. DOLAN, MD,	Monroe
ROBERT E. GORDON, MD,	Kings

Resident Councilor (representing the resident physician membership)

KATHLEEN E. SQUIRES, MD, New York

Student Councilor (representing the medical student membership)

MICHAEL PACIOREK, Onondaga

Trustees

RICHARD D. EBERLE, MD, (*Chairman*)	Onondaga
EDGAR P. BERRY, MD,	New York
JAMES M. FLANAGAN, MD,	Wayne
ALLISON B. LANDOLT, MD,	Westchester
DANIEL F. O'KEEFFE, MD,	Warren
BERNARD J. PISANI, MD,	New York
VICTOR J. TOFANY, MD,	Monroe

Executive Vice-President	DONALD F. FOY
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	GEORGE J. LAWRENCE, JR, MD
Director, Division of Policy Coordination	IRMA A. ERICKSON
Director, Division of Scientific Publications	PASCAL JAMES IMPERATO, MD
Director, Division of Communications	EDWARD A. HYNES
Director, Division of Computer Services	IVAN H. NAJMAN
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Governmental Affairs	GERARD F. CONWAY, ESQ
General Counsel	DONALD R. MOY, JD
Director, Division of Management and Meeting Services	ROBERT MILLER
Director, Division of Membership Support Services	EUNICE M. SKELLY
Director, Division of Scientific and Educational Activities	PETER B. FARNSWORTH, MD
Director, Division of Socio-Medical Economics	MARIO V. MENGHINI
Medical Director, Committee for Physicians' Health	BARTON A. HARRIS, MD
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York.

Copyright © 1987, Medical Society of the State of New York. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100

POSTAGE Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.

Freedom from pain

Just one part of pain relief therapy.

Vicodin® provides greater patient acceptance

COMPARATIVE PHARMACOLOGY OF THREE ANALGESICS

	CONSTIPATION	RESPIRATORY DEPRESSION	SEDATION	EMESIS	PHYSICAL DEPENDENCE
HYDROCODONE		X			X
CODEINE	X	X	X	X	X
OXYCODONE	XX	XX	XX	XX	XX

Blank space indicates that no such activity has been reported.

Table adapted from Facts and Comparisons (Nov.) 1984 and Catalano RB. The medical approach to management of pain caused by cancer. "Semin Oncol" 1975; 2; 379-92 and Reuler JB, et. al. The chronic pain syndrome: misconceptions and management. "Ann Intern Med" 1980; 93; 588-96.

◆ Vicodin offers: less nausea, less sedation, less constipation.

...and longer lasting pain relief– up to 6 hours.

◆ Vicodin contains hydrocodone not codeine. In one study, 10 mg. of hydrocodone alone was shown to be as effective as 60 mg. of codeine.[1]

◆ In a double-blind study, Vicodin (2 tablets), provided longer lasting pain relief than 60 mg. of codeine.[2]

Plus...

◆ Vicodin offers the convenience of CIII prescribing.

◆ Dosage flexibility–1 tablet every 6 hours or 2 tablets every 6 hours (up to 8 tablets in 24 hours).

1. Hopkinson JH III: *Curr Ther Res* 24: 503-516, 1978
2. Beaver, WT *Arch Intern Med*, 141:293-300, 1981.

vicodin®

hydrocodone bitartrate 5 mg. (Warning: May be habit forming) with acetaminophen 500 mg.

The original hydrocodone analgesic.

Please see adjacent page for brief summary of prescribing information.

vicodin ©

hydrocodone bitartrate 5mg (Warning: May be habit forming)
and acetaminophen 500mg

INDICATIONS AND USAGE: For the relief of moderate to moderately severe pain.
CONTRAINDICATIONS: Hypersensitivity to acetaminophen or hydrocodone.
WARNINGS
Drug Abuse and Dependence: VICODIN is subject to the Federal Controlled Substances Act (Schedule III). Psychic dependence, physical dependence and tolerance may develop upon repeated administration of narcotics; therefore, VICODIN should be prescribed and administered with the same caution appropriate to the use of other oral-narcotic-containing medications.
Respiratory Depression: At high doses or in sensitive patients, hydrocodone may produce dose-related respiratory depression by acting directly on brain stem respiratory centers. Hydrocodone also affects centers that control respiratory rhythm, and may produce irregular and periodic breathing.
Head Injury and Increased Intracranial Pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a preexisting increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.
Acute Abdominal Conditions: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions.
PRECAUTIONS
Special Risk Patients: VICODIN should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture.
Information for Patients: VICODIN, like all narcotics, may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery; patients should be cautioned accordingly.
Cough Reflex: Hydrocodone suppresses the cough reflex; caution should be exercised when VICODIN is used postoperatively and in patients with pulmonary disease.
Drug Interactions: The CNS-depressant effects of VICODIN may be additive with that of other CNS depressants. When combined therapy is contemplated, the dose of one or both agents should be reduced. The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone. The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus.
Usage in Pregnancy: Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose. There are no adequate and well-controlled studies in pregnant women. VICODIN should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.
Nonteratogenic Effects: Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent. The intensity of the syndrome does not always correlate with the duration of maternal opioid use or dose.
Labor and Delivery: Administration of VICODIN to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used.
Nursing Mothers: It is not known whether this drug is excreted in human milk; therefore, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.
Pediatric Use: Safety and effectiveness in children have not been established.
ADVERSE REACTIONS
Central Nervous System: Sedation, drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, dizziness, psychic dependence, mood changes.
Gastrointestinal System: Nausea and vomiting may occur; they are more frequent in ambulatory than in recumbent patients. Prolonged administration of VICODIN may produce constipation.
Genitourinary System: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported.
Respiratory Depression: (See WARNINGS.)
DOSAGE AND ADMINISTRATION: Dosage should be adjusted according to the severity of the pain and the response of the patient. However, tolerance to hydrocodone can develop with continued use, and the incidence of untoward effects is dose related. The usual dose is one tablet every six hours as needed for pain (if necessary, this dose may be repeated at four-hour intervals). In cases of more severe pain, two tablets every six hours (up to eight tablets in 24 hours) may be required.
Revised January 1987 MR 1987 5786

Knoll Pharmaceuticals
A Unit of BASF K&F Corporation
Whippany, New Jersey 07981

BASF Group

knoll®

ORTHOPEDISTS

Tired of the hassles of practice?
Tired of the malpractice burden?

I need two additional full-time orthopedists to join our 15 man group. One for our facility in Syracuse, and one for Rochester.

We do evaluations only, no treatment.

Banker's hours, pleasant work, good remuneration. No nights or weekends.

If you are ready to give up surgery for a more placid life, send your C.V. to me:

Harvey Lewis, M.D.
3155 G. Eggert Road
Tonawanda, NY 14150

ecmc erie county
medical center

MEDICAL DIRECTOR

The Erie County Medical Center, a major teaching hospital in Western New York, offers an excellent opportunity in a 490-bed, acute care, and 120-bed Skilled Nursing Facility in a public hospital in Buffalo, New York.

The Medical Director will report to the Chief Executive Officer and Board of Managers and will plan activities designed to enhance the medical staff and recruit members in the community to ensure continuing excellence in medical care.

Candidates must possess board certification in a medical specialty and have four (4) years of post-residency experience in a medical specialty, two (2) years of which must have been in an administrative/supervisory capacity.

If interested, send resume outlining your employment history and salary requirements to George McCoy, Chief Executive Officer, 462 Grider Street, Buffalo, NY 14215.

ECMC is an AA/EO Employer.

88
NEW YORK

American College of Physicians
presents
69th Annual Session
March 3–6, 1988
Jacob K. Javits
Convention Center

Medicine's foremost educational opportunity in the nation's most exciting city

- ☐ More than 200 presentations for a curriculum suited to your needs
- ☐ Critical findings in Molecular Biology and Clinical Medicine in six State of the Art lectures
- ☐ Pre-Session courses on critical care, geriatrics and infectious diseases
- ☐ Clinical controversies featured in 12 panel discussions
- ☐ Analyses that confront the social, ethical and environmental issues surrounding today's dilemmas
- ☐ Rewarding activities planned especially for family and guests

Request Your 1988 Scientific Program Guide

The Scientific Program Guide—with schedules, course and faculty lists, registration and housing information, and the family and guest program—enables you to plan the most valuable, personalized Session possible. Because many popular programs fill up quickly, you'll want to schedule your curriculum well in advance. Reserve your copy now by filling out and returning the request form.

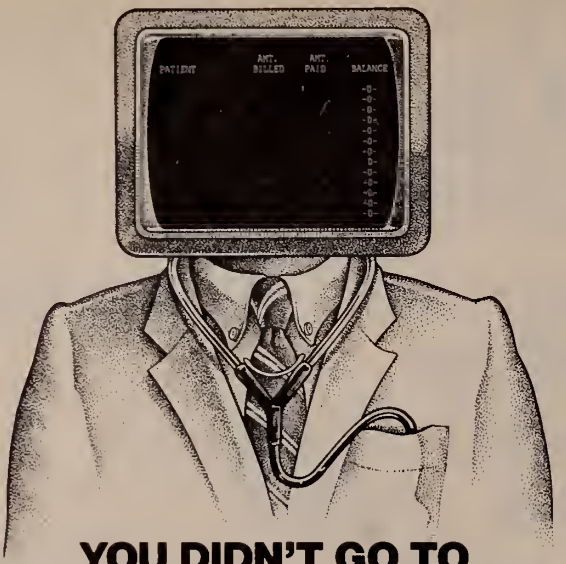
YES, mail me the '88 Scientific Program Guide NY11

PLEASE PRINT
NAME ____________
ADDRESS ____________
____
CITY/STATE/ZIP ____________

Telephone toll-free: (800) 523-1546 ext. 3675
In Pennsylvania: (215) 243-1200 ext. 3675

☐ ACP Member ACP# _ _ _ _ _ _
☐ Nonmember

American College of Physicians
4200 Pine Street
Philadelphia, PA 19104



YOU DIDN'T GO TO MEDICAL SCHOOL TO EXAMINE ACCOUNTS RECEIVABLE.

Is insurance and patient billing claiming too much of your time? For a surprisingly nominal fee we'll relieve you of most of your paperwork. No hardware or software to purchase. No staff to put on your payroll and train. For more information, perform a simple office procedure. Call us today.

bill of health services, inc.

SPECIALISTS IN COMPUTERIZED
MEDICAL INSURANCE BILLING

914-623-0022

In New York City, for the Newest and Finest Medical Space, Remember:

THE 4 ESSENTIAL REQUIREMENTS

1. LOCATION!
Liberty Residences at Battery Park City

2. LOCATION!
Windsor Pavilion
(3rd Avenue & 32nd Street)

3. LOCATION!
205 East 64th Street (3rd Avenue)

AND

4. LOCATION!
Normandie Pavilion
(3rd Avenue & 95th Street)

For Further Information Contact Lester Schwalb
(212) 708-0846
Milstein Properties Corp.

LIBRIUM®
chlordiazepoxide HCl/Roche Ⓥ
5-mg, 10-mg, 25-mg capsules

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Management of anxiety disorders; short-term relief of anxiety symptoms, acute alcohol withdrawal symptoms, preoperative apprehension and anxiety. Usually not required for anxiety or tension associated with stress of everyday life. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended.

Contraindications: Known hypersensitivity to the drug.

Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage. Withdrawal symptoms (including convulsions) reported after abrupt cessation of extended use of excessive doses are similar to those seen with barbiturates. Milder symptoms reported infrequently when continuous therapy is abruptly ended. Avoid abrupt discontinuation; gradually taper dosage.

> ***Usage in Pregnancy:*** **Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically. Due to isolated reports of exacerbation, use with caution in patients with porphyria.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral—Adults:* Mild and moderate anxiety disorders and symptoms, 5 or 10 mg *t.i.d.* or *q.i.d.*; severe states, 20 or 25 mg *t.i.d.* or *q.i.d.* *Geriatric patients:* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)

Supplied: Librium® (chlordiazepoxide HCl/Roche) *Capsules,* 5 mg, 10 mg and 25 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in boxes of 4 reverse-numbered cards of 25, and in boxes containing 10 strips of 10. Libritabs® (chlordiazepoxide/Roche) *Tablets,* 5 mg and 10 mg—bottles of 100 and 500; 25 mg—bottles of 100. With respect to clinical activity, capsules and tablets are indistinguishable.

ROCHE
Roche Products Inc.
Manati, Puerto Rico 00701

Do not substitute

Librium®

brand of

chlordiazepoxide HCl/Roche Ⓒⅳ

Nobody does it better.

ROCHE®

Copyright © 1986 by Roche Products Inc. All rights reserved.

Please see adjacent page for summary of product information.

Corporate Physician

American Airlines is seeking a highly qualified individual to assume the responsibilities of Corporate Physician for its New York City Medical Department.

The qualified candidate will be a licensed Physician in the state of New York with a background in Aerospace, Occupational, Industrial, Preventive, Emergency Medicine or other clinical experience. Specialty certification and FAA Examiner status highly desirable. U.S. medical training preferred.

American Airlines offers an excellent compensation and benefits package. For immediate consideration, please forward your resume, including salary history, in confidence to:

Manager Personnel Services
P.O. Box 619616
Mail Drop 3K28, Dept. NYCDR
D/FW Airport, Texas 75261-9616

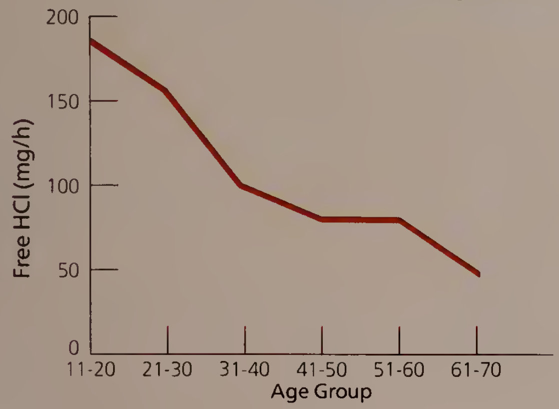
AmericanAirlines

Specialized ulcer therapy

When advancing age signals reduced acid secretion

If your duodenal ulcer patient is over 55, decreased mucosal resistance is more likely to cause an ulcer than hypersecretion of acid-pepsin.[1] A tendency toward lower acid secretion with advancing age has been shown.[2,3]

Declining gastric secretion and age[3]



CARAFATE® (sucralfate/Marion) makes sense as initial ulcer therapy for the elderly. Carafate provides ulcer healing rates comparable to H_2 antagonists without the risk of systemic side effects or drug interactions—an important benefit for older patients.

The unique, nonsystemic action of Carafate enhances the body's own ulcer healing ability, strengthening the mucosal structure as it protects damaged tissue from further injury.

When advancing age signals reduced acid secretion, choose the specialized ulcer therapy of safe, nonsystemic Carafate.

Nothing works like

CARAFATE®
sucralfate/Marion

Please see adjoining page for references and brief summary of prescribing information.

1595H7

CARAFATE®
(sucralfate)

BRIEF SUMMARY

CONTRAINDICATIONS
There are no known contraindications to the use of sucralfate.

PRECAUTIONS
Duodenal ulcer is a chronic, recurrent disease. While short-term treatment with sucralfate can result in complete healing of the ulcer, a successful course of treatment with sucralfate should not be expected to alter the post-healing frequency or severity of duodenal ulceration.

Drug Interactions: Animal studies have shown that the simultaneous administration of CARAFATE with tetracycline, phenytoin, or cimetidine will result in a statistically significant reduction in the bioavailability of these agents. This interaction appears to be nonsystemic in origin, presumably resulting from these agents being bound by CARAFATE in the gastrointestinal tract. The bioavailability of these agents may be restored simply by separating the administration of these agents from that of CARAFATE by two hours. The clinical significance of these animal studies is yet to be defined.

Carcinogenesis, Mutagenesis, Impairment of Fertility: No evidence of drug-related tumorigenicity was found in chronic oral toxicity studies of 24 months' duration conducted in mice and rats at doses up to 1 gm/kg (12 times the human dose). A reproduction study in rats at doses up to 38 times the human dose did not reveal any indication of fertility impairment. Mutagenicity studies have not been conducted.

Pregnancy: Pregnancy Category B. Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS
Adverse reactions to sucralfate in clinical trials were minor and only rarely led to discontinuation of the drug. In studies involving over 2,500 patients, adverse effects were reported in 121 (4.7%). Constipation was the most frequent complaint (2.2%). Other adverse effects, reported in no more than one of every 350 patients, were diarrhea, nausea, gastric discomfort, indigestion, dry mouth, rash, pruritus, back pain, dizziness, sleepiness, and vertigo.

DOSAGE AND ADMINISTRATION
The recommended adult oral dosage for duodenal ulcer is 1 gm four times a day on an empty stomach.

Antacids may be prescribed as needed for relief of pain but should not be taken within one-half hour before or after sucralfate.

While healing with sucralfate may occur during the first week or two, treatment should be continued for 4 to 8 weeks unless healing has been demonstrated by x-ray or endoscopic examination.

HOW SUPPLIED
CARAFATE (sucralfate) 1-gm pink tablets are supplied in bottles of 100 and in Unit Dose Identification Paks of 100. The tablets are embossed with MARION/1712. Issued 3/84

References:
1 Grossman MI: *Scand J Gastroenterol* 58 (suppl 15):7-16, 1980.
2 Marks IN, in Hellemans J, Vantrappen G (eds): *Gastrointestinal Tract Disorders in the Elderly.* Edinburgh, Churchill Livingstone, 70-81, 1984.
3 Krentz K, Jablonowski H, in Hellemans J, Vantrappen G (eds): *Gastrointestinal Tract Disorders in the Elderly* Edinburgh, Churchill Livingstone, 62-69, 1984

Another patient benefit product from
PHARMACEUTICAL DIVISION
MARION LABORATORIES INC.
KANSAS CITY MO 64137

1595H7

Classified Advertising

PHYSICIANS WANTED

EMERGENCY MEDICINE POSITIONS—Full/part time emergency medicine physicians sought by multi state professional association for openings in metropolitan NY, PA, MD, DC, FL, New England and throughout U.S. Contact or send CV to Liberty Healthcare Corporation, 399 Market Street, Suite 400, Philadelphia, PA 19106, (215) 592-7400 or outside Pa (800) 331-7122.

ARIZONA-BASED PHYSICIAN RECRUITMENT firm has quality opportunities coast to coast. Available positions in most primary care and surgical specialties to include OB/GYN, Orthopedics, ER, and ENT. "Quality Physicians for Quality Clients since 1972." Call 602-990-8080; or send CV to: Mitchell & Associates, Inc., PO Box 1804, Scottsdale, AZ 85252.

INTERNIST WANTED—Busy three man General Internal Medicine practice in community setting seeks board eligible/board certified internist for primary care responsibilities. Excellent benefits and opportunties offered for association. Contact (201) 837-3200 or send C.V. to Dr. Peter W. Coppola, 751 Teaneck Road, Teaneck, NJ 07666.

GENERAL SURGEON, University trained needed immediately, Upstate New York. Guaranteed income. With benefits. Reply Dept. 451 c/o NYSJM.

HEALTH COMMISSIONER: Physician needed to administer and manage Rural Orange County, NY (50 miles north of NYC) Public Health Department. Send resume and salary history to: Commissioner of Personnel, Orange County Government Center, 255-275 Main Street, Goshen, NY 10924. An Equal Opportunity Employer M/F.

EMERGENCY ROOM PHYSICIANS: BC/BE attending physicians being recruited for part and full time positions. Major teaching hospital's Emergency Department, with 75,000 annual visits. Emergency Medicine, Internal Medicine, Surgery and Family practice physicians will be considered. We offer excellent salaries and benefits. Send CV to: Director of Human Resources, Employment Division, Montefiore Medical Center, 111 East 210 Street, Bronx, NY 10467. Equal Opportunity Employer M/F.

ORTHOPAEDIST NEEDED: for busy 4 man orthopaedic group practice, Jersey shore, credentialled/experienced in spinal surgery or sportsmedicine. Early partnership potential. Superb opportunity for physician who wants to reap the professional, emotional and financial rewards which hard work and dedication bring. Exceptional compensation package with many options for successful candidate who can report by January or February 1988. In New York, call toll free 1-800-822-6374.

PHYSICIANS WANTED—CONT'D

SIX MONTH PULMONARY FELLOWSHIP at the Mount Sinai Bronx Veterans Administration facility emphasizing clinical pulmonary medicine including critical care, pulmonary physiology and bronchoscopy. Requires six years internal medicine residency and U.S. license. Position available from 1/1/88 to 6/30/88. Good salary plus fringe benefits for the six months. Send c.v. to Alvin S. Teirstein, M.D., Chief, Pulmonary Division, Mount Sinai Medical Center, Box 1232, One Gustave L. Levy Place, New York, NY 10029.

POSITIONS WANTED

SURGEON FOR HIRE. Experienced general surgeon available 3 days a week, for first assisting, Westchester area. O.R. work only. Must include monetary offer in your reply. Dept. 450 c/o NYSJM.

PRACTICES AVAILABLE

FOR SALE—VERY BUSY FAMILY PRACTICE, Nassau, Queens border, excellent location. Physician's Assistant available familiar with patients. Please call (516) 437-1237 or (516) 627-2149 after 7 P.M.

EQUIPMENT

50% OFF PREVIOUSLY OWNED MEDICAL—Laboratory, x-ray, ultrasound equipment. Buy, Sell, Broker, Repair. Appraisals by certified surgical consultants. Medical Equipment Resale & Repair, 24026 Haggerty Road, Farmington Hills, MI 48018. 1-800-247-5826. (313) 477-6880.

REAL ESTATE FOR SALE OR RENT

FOR SALE—Combination professional office and home, Oceanside, NY; opposite South Nassau Community Hospital on 1/3rd acre lot. Never pay rent again. Telephone (516) 536-6279.

CUSTOM BUILD superbly designed professional home located in the East Northport area on highly visible 1/2 acre, just minutes from local hospitals, parkways and schools. Reception room plus 900 sq. ft. useable space on lower level. Upper level features wonderful contemporary living area with highhat lighting, skylights and cathedral ceilings. Asking $425,000. The Daniel Gale Agency, Camille Sonesen (516) 754-3400 or Joan Gannon (516) 692-6770.

FOR SALE—OFFICE BUILDING, L.I. Suffolk County. Family practitioner retiring after 33 years. Building has 3 office suites, C/A, W/W carpeting, paneling, separate entrances, private parking and 3 zone heat. Excellent location, near hospitals. Suitable all specialties. For information please call (516) 667-7872.

Added Control For America's Coughs

The expectorant action of guaifenesin plus the Advantage of Codeine!

Robitussin and codeine. Each 5 ml (*1 teaspoonful*) contains: Guaifenesin, USP, 100 mg; Codeine Phosphate, USP, 10 mg (*warning: may be habit forming*); alcohol, 3.5 percent; in a palatable, aromatic syrup

Robitussin A-C®Ⓥ

A·H·ROBINS

Pharmaceutical Division, Richmond, Virginia 23261-6609

©A.H. Robins Company 1985

AIM HIGH

YOU CAN KEEP THEM
IN BALANCE—
YOUR FAMILY LIFE
AND YOUR
MEDICAL PRACTICE.

We'd like to help you spend more time with your family yet receive professional satisfaction from your medical practice. As a member of the Air Force health care team, you'll be able to participate in our group practice concept which will free you of most administrative duties.
Air Force benefits are also very attractive. You and your family will enjoy 30 days of vacation with pay each year plus many more Air Force advantages. Call

Capt Robert Schawelson
516-484-6940

AIR FORCE

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

DECEMBER 1987

AROUND THE STATE

ALBANY

Dec 10–11. **Managing the AIDS Crisis: A Multidisciplinary Approach.** Albany Hilton Hotel. Contact: Albany Medical College, Office of Continuing Medical Education, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

BRONX

Dec 9. **Courts on Trial: Fractured Forensic Sciences.** Lehman College. Contact: Mark L. Taff, MD, President, New York Society of Forensic Sciences, 130 Gold Pl, Malverne, NY 11565. Tel: (516) 887-4691.

MANHATTAN

Dec 3–4. **Current Topics in Gastroenterology and Hepatology.** 15 Cat 1 Credits. College of Physicians and Surgeons of Columbia University. Contact: Center for Continuing Education, 630 W 168th St, New York, NY 10032. Tel: (212) 305-3682.

Dec 3–4. **Surgical Health Policy and Finance.** 14 Cat 1 Credits. The Waldorf Astoria. Contact: Ann J. Boehme, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

Dec 3–4. **Third Biennial Cornell Course in Infectious Diseases.** New York City. Contact: Dr Henry Murray, Division of Infectious Diseases, Cornell University Medical College, 1300 York Ave, New York, NY 10021. Tel: (212) 472-6800.

Dec 3. **Innate and Environmental Factors in the Development of the Visual Cortex.** 1 Cat 1 Credit. New York Academy of Medicine. *Dec 4.* **Visual Processing in the Striate Cortex.** 1 Cat 1 Credit. The Hotel Roosevelt. Contact: Mary Cunningham, New York Academy of Medicine, 2 E 103rd St, New York, NY 10029. Tel: (212) 876-8200, ext 220.

Dec 5–6. **Third Annual Infection/Immunology in Obstetrics and Gynecology.** 12 Cat 1 Credits. New York City. Contact CRISIS, PO Box 4853, New York, NY 10185. Tel: (212) 532-1205.

Dec 4–5. **Contemporary Concepts in Facial Surgery.** 11 Cat 1 Credits. The Waldorf Astoria Hotel. Contact: Francine Leinhardt, Course Coordinator, Manhattan, Eye, Ear and Throat Hospital, 210 E 64th St, New York, NY

(*continued on p 22A*)

A better alternative for hypertensives who are going bananas...

Spare your patients the extra cost— in calories, sodium and dollars.

Spare your patients the rigors of dietary K+ supplementation.

DYAZIDE®

25 mg Hydrochlorothiazide/50 mg Triamterene/SKF

Effective antihypertensive* therapy...without the bananas

DAW

'DYAZIDE' AS WRITTEN.

* Not for initial therapy. See brief summary.

Before prescribing, see complete prescribing information in SK&F CO. literature or PDR. The following is a brief summary.

* **WARNING**

This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

Contraindications: Concomitant use with other potassium-sparing agents such as spironolactone or amiloride. Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: The bioavailability of the hydrochlorothiazide component of 'Dyazide' is about 50% of the bioavailability of the single entity. Theoretically, a patient transferred from the single entities of triamterene and hydrochlorothiazide may show an increase in blood pressure or fluid retention. Similarly, it is also possible that the lesser hydrochlorothiazide bioavailability could lead to increased serum potassium levels. However, extensive clinical experience with 'Dyazide' suggests that these conditions have not been commonly observed in clinical practice. Angiotensin-converting enzyme (ACE) inhibitors can elevate serum potassium, use with caution with 'Dyazide'. Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids, and during concurrent use with amphotericin B or corticosteroids or corticotropin[ACTH]). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Cumulative effects of the drug may develop in patients with impaired renal function. Thiazides should be used with caution in patients with impaired hepatic function. They can precipitate coma in patients with severe liver disease. Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic and hemolytic anemia have been reported with thiazides. Thiazides may cause manifestation of latent diabetes mellitus. The effects of oral anticoagulants may be decreased when used concurrently with hydrochlorothiazide; dosage adjustments may be necessary. Clinically insignificant reductions in arterial responsiveness to norepinephrine have been reported. Thiazides have also been shown to increase the paralyzing effect of nondepolarizing muscle relaxants such as tubocurarine. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. Triamterene has been found in renal stones in association with the other usual calculus components. Therefore, 'Dyazide' should be used with caution in patients with histories of stone formation. A few occurrences of acute renal failure have been reported in patients on 'Dyazide' when treated with indomethacin. Therefore, caution is advised in administering nonsteroidal anti-inflammatory agents with 'Dyazide'. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia is uncommon with 'Dyazide', but should it develop, corrective measures should be taken such as potassium supplementation or increased dietary intake of potassium-rich foods. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Concurrent use with chlorpropamide may increase the risk of severe hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function. Thiazides may add to or potentiate the action of other antihypertensive drugs. Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth, anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances; postural hypotension (may be aggravated by alcohol, barbiturates, or narcotics). Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and respiratory distress including pneumonitis and pulmonary edema, transient blurred vision, sialadenitis, and vertigo have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components. Rare incidents of acute interstitial nephritis have been reported. Impotence has been reported in a few patients on 'Dyazide', although a causal relationship has not been established.

Supplied: 'Dyazide' is supplied as a red and white capsule, in bottles of 1000 capsules; Single Unit Packages (unit-dose) of 100 (intended for institutional use only); in Patient-Pak™ unit-of-use bottles of 100.

BRS-DZ:L45

a product of
SK&F CO.
Cidra, P.R. 00639

SK&F Co., 1987

1988 ANNUAL CME ASSEMBLY

Medical Society of the State of New York
The New York Hilton, New York City

Newer Approaches to Habit Abuse and Dependency

FRIDAY, APRIL 22

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Plenary Session: Human Abuses Through the Ages: "From the Womb to the Tomb," *Sutton South/Regent Parlor*
2:00 p.m. - 5:00 p.m.	Women's Medical Society/Occupational Health, *Gramercy B*
	Cardiology, *Sutton South*
	Physical Medicine & Rehabilitation/Family Practice/Orthepedics, *Gramercy A*
	Emergency Medicine/Critical Care, *Murray Hill B*
	Pathology/Pediatrics, *Bryant Suite*
	General Surgery/Radiology, *Regent Parlor*
	Neurosurgery, *Murray Hill A*
	Medical Liability Mutual Insurance Co., *Rendezvous Trianon*

SATURDAY, APRIL 23

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Internal Medicine (All Day), *Regent Parlor*
	Ophthalmology (All Day), *Sutton South*
	Hand Surgery/Neurology (All Day), *Sutton Parlor North*
	Psychiatry (All Day), *Gramercy B*
	Urology/Radiology, *Nassau A*
	Dermatology, *Murray Hill A*
2:00 p.m. - 3:00 p.m.	Medical Technology
2:00 p.m. - 5:00 p.m.	Internal Medicine, *Regent Parlor*
	Ophthalmology, *Sutton South*
	Hand Surgery/Neurology, *Sutton Parlor North*
	Psychiatry, *Gramercy B*
	Chest Diseases/Otolaryngology, *Murray Hill B*
	Alcoholism, *Madison Suite*
3:00 p.m. - 5:00 p.m.	Computers in Medicine, *Nassau A*

SUNDAY, APRIL 24

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Allergy & Immunology, *Gramercy B*
	Obstetrics and Gynecology, *Murray Hill A*
	Gastroenterology/Colon and Rectal Surgery, *Nassau A*
	Orthopedics/Family Practice/Physical Medicine & Rehabilitation/ Anesthesiology, *Regent Parlor*
	Sleep Disorders—Apnoea, *Nassau B*

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis. The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians.

* * *

Technical Exhibits

NEW YORK STATE
JOURNAL OF MEDICINE

November 1987 • Volume 87, Number 11

COMMENTARIES

A view of the proposed New York State regulations governing the professional activities of residents

The Associated Medical Schools of New York, representing 13 medical schools, is gravely concerned over the potential harmful impact of a series of proposed New York State regulations governing the work of residents in New York hospitals. Several of these recommended regulations, if enacted, would undercut the ability of academic medical centers to properly train future physicians, would impair the quality of care provided in these hospitals, and could increase the costs of medical education in New York.

At issue are two sets of proposed revisions to Section 405 of the New York State Health Code. In a separate statement, AMS has commented on those recommendations related to the clinical training of medical students.[1] The present statement is focused on the proposed revisions in the Code dealing with the work of residents and attending physicians. Four of these recommendations were announced on June 2, 1987, by the Ad Hoc Committee on Emergency Services appointed by Dr David Axelrod, State Health Commissioner.[2] The committee's recommendations include the following:

1. There shall be at least one emergency service attending physician on duty 24 hours a day, seven days a week, in all hospitals which have more than 15,000 unscheduled visits annually. An attending physician shall be present and available in person to provide patient care in the emergency service.
2. Emergency service attending physicians shall be licensed and board certified in emergency medicine, surgery, internal medicine, or family practice.
3. House officers and attending physicians with direct patient care responsibilities shall not work for more than 12 consecutive hours per shift in the emergency service, separated by no less than eight hours off. House officers and attendings with direct patient care responsibilities in other areas should, in general, not work more than 16 hours per shift. Individuals who have worked the maximum in one facility would not be allowed to work a consecutive shift in another hospital.
4. Contemporaneous, in-person supervision by attending physicians of all residents and interns was endorsed in concept by the committee with the caveat that this recommendation needed further study.

A series of additional recommended changes in the code proposed by the State Health Department flowed directly from these recommendations of the ad hoc committee. The code revisions include the following:

> (ii) The medical staff shall, based on successful completion of this review, grant privileges that are specific to each individual in such programs prior to delivery of patient care services.
>
> (iii) Privileges, regardless of whether the individual is full-time, part-time or in rotating status, shall be individually reviewed and reapproved, when appropriate, at least annually.
>
> (iv) Specific treatment/procedures that each individual is authorized to perform shall be stated in writing.

AMS, the members of which have affiliations with the majority of teaching hospitals in the state, supports any effort to improve the quality of care delivered in New York hospitals. However, in this instance the state Department of Health has greatly exceeded its mandate, involving itself in the details of medical education, and in such a way as to be harmful to the very goals it espouses. The department has done so with minimal consultation and discussion with the medical schools and teaching hospitals of New York which have the primary responsibility for and are accountable for assuring the content and quality of medical education.

The first two recommendations of the ad hoc committee, which deal with emergency room services, are appropriate and AMS supports them. Indeed, two thirds of the hospitals in New York City designated by the NYC Emergency Medical Services to receive emergency patients already have the prescribed attending coverage.

Our major concerns are focused on recommendations 3 and 4.

The intent of recommendation 3 is to guard against fatigue that would impair the performance of a resident or attending. First, it must be emphasized that the tragic circumstances in a New York City hospital which triggered these recommendations had nothing whatsoever to do with impairment of performance or judgment as a result of fatigue. Second, if this recommendation is approved and implemented, its potential effect would be to increase rather than decrease fatigue and burnout.

In the current system, residents are "on call" every third or fourth night, meaning they are in the hospital for up to 36 consecutive hours, but not necessarily awake for that period of time. In the intensive care unit, where residents may be up for long periods, the resident on night duty usually leaves the following morning after sign-out rounds. There are good reasons for this traditional system. The first 24 to 36 hours of hospitalization are a critical period for many patients. Having a single physician or small team of physicians continuously care for the patient during that time assures continuous quality care.

There is also no doubt that the practice of medicine requires clear decision making at 4 AM as well as 4 PM, and the development of that capacity in physicians is part of the residency experience. To have a "platoon system," as is implied by the recommendations of the committee, would suggest that it is acceptable for a physician to walk away from the bedside because his or her "shift" is over when 16 hours are up.

The work schedule recommended by the ad hoc committee—a maximum of 16 hours a day for five or six days a week—is, in fact, potentially more oppressive than the current system of being on call every third or fourth night with opportunities to sleep. If patient coverage is not to be reduced, the total number of hours worked per week could well increase. This would be inconsistent with national standards.

The Essentials of Accredited Residencies of the Accreditation Council of Graduate Medical Education, which accredits residency programs, specifically states that resident on-call rotations should occur with a frequency of every third or fourth night. An alternative might be establishing a maximum number of hours per week which a resident could work, permitting flexibility while assuring a reasonable limit on the workload.

There are also ways of making the time of a resident more efficient and effective when that individual is on call. Residents should be relieved of their nonphysician duties, particularly at night, when they often are required to perform tasks such as drawing blood, starting intravenous lines, delivering laboratory samples, and transporting patients. Ancillary personnel should be made available to perform these duties to decompress residents' workloads.

It is also important to emphasize that, if this recommendation is implemented and residents are required not to work more than six consecutive 16-hour shifts, more residents may be required. Decisions on the number of residents in a teaching program, however, are not made solely by the hospital or medical schools based on service needs. Independent residency review committees, taking into account a program's educational resources, have a decisive influence on the number of residents in a program. However, even if additional residency positions were approved, this would be in direct conflict with the conclusions of the New York State Commission on Graduate Medical Education, which recommended a statewide reduction in residency positions of up to 30%.

The question of number of residents also bears on the fourth recommendation, regarding 24-hour supervision by attendings. Given the commission recommendations for reductions in residency positions and the probability that residency review committees will not permit increases in positions, there seems no likelihood that New York will see any increase in the number of physicians trained. Thus a readily available pool of adequately trained attending physicians willing to spend additional hours in the hospital is not in sight. One of the most disturbing consequences of these regulations would be their effect on public hospitals and medical care for the indigent. These hospitals already face manpower problems because of financial concerns, and would be at a serious disadvantage in competition for additional attendings. These hospitals might then be forced to use inadequately trained house physicians, thus leading toward a lesser standard of care for low income and medically indigent people.

Furthermore, "in-person" and "contemporaneous" supervision of all residents' activities would destroy one of the fundamental goals of the present system: the development of a capacity for independent judgment and action, which is crucial to the practice of medicine.

The ad hoc committee appropriately recommended further study of this grand jury recommendation. This study must be accomplished with broad representation from the medical schools and hospital communities.

The recommendations of the ad hoc committee are troublesome, inconsistent with state health policy, and are a direct consequence of permitting political pressures growing out of the New York City tragedy to override thoughtful consideration of policies that will have major implications for patient care and medical education. AMS strongly urges the ad hoc committee to carefully review its recommendations in light of these concerns.

The proposed changes in regulations governing the credentialing of residents also are puzzling and troublesome. They are totally insensitive to the way in which residents develop competence. If interpreted as written, they could produce a blizzard of paper work as hospitals provide a centralized mechanism for continuously re-credentialing residents on an individual basis.

The most effective way of assuring quality in graduate medical education is to put the responsibility where it belongs—on program directors, who must make sure that residents are not permitted to carry out any procedure beyond their competence, and who must ensure that they are properly supervised by licensed and appropriately credentialed physicians. The proposed changes might threaten accreditation of graduate medical education programs in this state. They may adversely affect the ability of teaching hospitals to attract top quality candidates to our residency programs, with ultimate effects on the overall quality of physicians practicing in this state and on the care of our patients.

To reiterate our overall concern, the recommendations for changes in the health code and those of the ad hoc committee intrude the state Health Department into the heart of medical education in a way that is both potentially harmful and totally without cause. Associated Medical Schools of New York recognizes and supports strongly the interest of the state in assuring quality of care. We are willing to work with the ad hoc committee, the State Hospital Review and Planning Council, and representatives of other medical constituencies to move expeditiously toward steps that will safeguard quality patient care while preserving and strengthening existing medical education programs.

ASSOCIATED MEDICAL SCHOOLS OF NEW YORK
Bellevue Hospital Center
First Ave and 27th St
New York, NY 10016

1. *Position Statement on Proposed Regulations Pertaining to the Activities of Medical Students*, New York, Associated Medical Schools of New York, August 3, 1987.
2. Letter from Bertrand Bell, MD, Chairman, Ad Hoc Committee on Emergency Services for Dr David Axelrod, Commissioner of Health, State of New York, June 2, 1987.

Artificial breathing support systems: Preferred nomenclature for medicine and the courts

The recently decided case of *Delio*[1] addresses the issue of the prolongation of life by artificial means. Daniel Delio was a patient at Westchester County Medical Center, who was transferred there in a persistent vegetative state after allegedly suffering cerebral anoxia during surgery at another hospital. His ability to breathe was never compromised, although he did need to receive feedings through a surgically placed gastrostomy tube. A few months later his wife sought court permission to remove the feeding tube as she thought her husband would have requested. In December 1986, New York State Supreme Court Justice Anthony J. Cerrato rejected the request.[2] That ruling was appealed, and on June 1, 1987, the Appellate Division of the New York State Supreme Court ruled that removal of the feeding tube was permissible.[1]

In New York State and throughout the country, physicians, attorneys, legislators, and the public are deeply concerned about the development of appropriate guidelines for resuscitation and the maintenance of life by artificial means. The controversy rages as litigation has recently expanded to encompass the issues of transfusion of blood and blood products, infusion of fluids, and placement of feeding tubes for nutrition. Despite occasional examinations of the "right to die" concept and of the issue of disconnecting respirators, which have occurred in such court cases as *The matter of Eichner*[3] (the Brother Fox case), the laws in New York remain vague on several issues of extraordinary and not-extraordinary treatment. In evaluating whether the removal of Mr Delio's feeding tube would be allowed, the Appellate Division of the New York State Supreme Court looked at similar cases in other states.

In 1976, after critical review of many ethical and medical considerations, the Supreme Court of the State of New Jersey permitted discontinuance of the life support systems for Karen Ann Quinlan in a landmark decision. An integral component of Quinlan's life support process was the Bennett MA-1 respirator, and was so termed when the decision stated that "she requires a respirator" as opposed to "being on a ventilator." [4] Even eleven years later, as the New York court deliberated on Mr Delio's case, the term "respirator" appears to have become inculcated into the legal phraseology of life support system decisions. "The withdrawal or withholding of feeding by artificial means," the New York Appellate Court added, "should be evaluated in the same manner as any other medical procedure. In this respect, we view nutrition and hydration by artificial means as being the same as the use of a respirator." [5] Are the words *respirator* and *ventilator* synonymous, or is there an important difference between them? If there is a difference, what then should we call this mechanical box for breathing that rests at the patient's bedside? Should a court view this debate as properly belonging to the medical community alone, or are there possible legal ramifications with the use of each term?

Historically, methods to sustain life when the customary respiratory mechanisms fail extend back to biblical times, when the first descriptions of mouth-to-mouth breathing as the simplest form of positive airway pressure delivery were recorded.[6] By the time of the Renaissance, Vesalius had devised a method for tracheal intubation and lung inflation.[7] Entering the 20th century, interest moved away from the use of external bellows devices to devices that duplicated the negative pressure caused by the bellows-like function of the thoracic structure. By 1930 the tank respirator—the so-called iron lung—was in widespread use, and was used to aid many polio patients. The respirator worked by lowering the pressure inside the tank to below that of atmospheric pressure, causing air to flow into the lungs. When atmospheric pressure was allowed to return, the intrinsic lung recoil forced air back out. The cuirass, or shell respirator, was also utilized for some patients with neuromuscular disorders, although with less success. During the 1940s and 1950s, new positive pressure techniques were developed, and a variety of pressure-flow cycled, time-cycled, and volume-cycled machines emerged over the following decades as small refinements were added.[8] Today, high-frequency ventilators are available as well, which deliver low tidal volumes at rapid respiratory rates. With the development of this advanced technology in response to medical problems, a plethora of ethical, religious, and legal problems were soon to follow. These led to the necessity of distinguishing between the prolongation of life and the prolongation of existence.

We believe there is a difference between a respirator and a ventilator, although a fine line probably exists in selecting one term over the other. Certainly, a random look at the literature does not make it clear whether the term *ventilator* is preferred over *respirator.* A review entitled "Chemical regulation of respiration" [9] and an editorial entitled "Chemical regulation of ventilation: Who needs it?" [10] leave one more confused as to which term is more appropriate and when to use either one.

Perhaps a brief review of the definitions of respiration and ventilation will allow a better understanding of these terms and how they apply to this mechanical box at the patient's bedside. In the simplest terms, the major function of respiration is to supply the cells of the body with adequate oxygen and to remove carbon dioxide. In mammals, respiration consists of a series of four interlinked processes.[11] The first is ventilation, which involves the delivery of fresh atmospheric air (inspiration) to the alveoli where it is exposed to the blood. Gas is also removed (expiration) after the blood has given up oxygen and received carbon dioxide. The second process is diffusion, which involves gas exchange between the blood in the lung capillaries and the lung air. The third is the circulation of blood through the systemic and pulmonary circulations. The fourth and final process is internal respiration between the tissues and the blood in the capillaries of the general systemic circulation.

Ventilation, therefore, in its simplest form, is part of respiration, and not vice versa. The major indication for mechanical ventilation is acute respiratory failure, of which there are two basic forms: ventilatory pump failure, causing an inadequate minute ventilation with consequent hypercapnea; and inefficient pulmonary gas exchange, causing predominantly hypoxemia. Is it now safe, on this basis, to make conclusions as to whether the mechanical box should be called a ventilator or a respirator? If it is called a ventilator, is merely ventilation or the entire process of respiration achieved by the box? A ventilator unquestionably delivers oxygen from the machine to the lung (inspiration) and then passively removes carbon dioxide (expiration). Although a ventilator brings oxygen into the alveoli, diffusion of gases is a separate process, independent of ventilation. Of note is the fact that inefficient gas exchange due to a diffusion disorder can be ameliorated in part by a ventilator with the administration of a higher oxygen concentration. Indeed, today there are ventilators in which the principle of movement of air centers around high velocity of the gas with passive diffusion as the means for gas exchange, rather than the usual mechanism of convection. In patients without cardiopulmonary disease, a ventilator has minimal effect on cardiac function and thus alters circulation marginally. In this same group of patients, a ventilator affects internal respiration only inasmuch as an adequate concentration of oxygen is provided so that oxygen consumption is satisfied at the tissue level.

It appears, therefore, that for patients who require a mechanical box to breathe, and who do not have coexisting cardiopulmonary disease, the appropriate term for the box providing breathing support is a ventilator. Circulatory alterations and tissue respiration remain primarily independent variables in these patients. Patients who have brain stem dysfunction with the absence of spontaneous respirations are probably also supported by a ventilator.

In patients with cardiopulmonary disease, the interaction of pulmonary mechanics on cardiac function may be substantial, and vice versa. A breathing support system probably affects all of the interlinked processes of respiration. Under these circumstances, a breathing box may alter cardiac output and may adversely affect oxygen delivery. This may result in a change in cellular respiration. Accordingly, in order to maintain basal oxygen consumption with an alteration in cardiac output, it is necessary to change the oxygen content of arterial or mixed venous blood. Acknowledging the impact of the breathing box on the third process of circulation and the fourth process of respiration, it would seem reasonable to call the box a respirator.

We should be careful, both in the hospital and in court, to use the most appropriate terminology. This is not merely a question of semantics, as the connotations of words for legislators and the judiciary may be substantial. While it is unlikely that any decisions will be significantly altered, the court system ought to have available the expertise of specialists to define the proper designation of the machine each time it is used. If the box is a ventilator, the implication is that it does not provide total support of respiration for the body. If the device is a respirator, it is responsible for exchange of oxygen at even the cellular level. Thus there is more physiological impact when a respirator is withdrawn than when a ventilator is withdrawn. There is a greater emotional meaning as well. In the case of a young person who is without cardiopulmonary problems, as was Ms Quinlan when her oxygen-supplying life support system was stopped, the correct term for the device should be ventilator. If Karen Ann Quinlan were on a respirator, its removal ought to have led to her immediate demise, for the ability to respire must be completely absent in the brain-dead patient. To the surprise of all, she continued to breathe independently and existed for several additional years. Logic dictates that for her the machine was a ventilator as she could function independently of it, even though throughout the court deliberations the machine was repeatedly identified as a respirator.

As we attempt to define the name of this device for each patient connected to it, we may become more alert to the impressive interactions that may follow. Accordingly, there may be better comprehension of the changes in cardiac, hepatic, and renal functions that ensue. We watch the machine at work, yet the domino effect which this machine has on systemic function is, at best, poorly understood. Possibly, as our knowledge of the physiology of respiration is enhanced, our views will change. On the other hand, legislators and jurists may not debate this particular question too long, for so many other critical aspects of the life support controversy have taken precedence. The New York State legislature has just enacted an amendment in relation to orders not to attempt cardiopulmonary resuscitation. This makes New York the first state in the country to pass a law detailing do-not-resuscitate orders for terminally ill patients.[12] In one portion of the bill, cardiopulmonary resuscitation was defined as "measures . . . to restore cardiac function or to support ventilation in the event of a cardiac or respiratory arrest." [13] This would seem to be an accurate method of working with the nomenclature.

ROBERT D. BRANDSTETTER, MD
Assistant Director
Department of Medicine

FRANK TAMARIN, MD
Chief Resident
Department of Medicine
New Rochelle Hospital Medical Center
New Rochelle, NY 10802

Acknowledgments. The authors thank Mr James M. Morrissey, Esq, for his comments during the preparation of the manuscript, and Mr Jeffrey M. Tamarin, Esq, for his acquisition of some of the reference material.

1. *Matter of Delio* (App Div 2d Dept). *NY Law J*. June 4, 1987, p 17.
2. *Delio on Behalf of Delio v Westchester County*, 510 NYS 2d 415 (Supp 1986), pp 415–420.
3. *In the Matter of Eichner*, 52 NY 2d 363, 438 NYS 2d 266, 420 NE 2d 64 (1981).
4. *In the matter of Karen Quinlan, an alleged incompetent*. New Jersey Reports; 70:10–55 (Supreme Court of New Jersey, 1976).
5. Nagourney E: Court OKs removal of feeding tubes. *The Standard Star*, June 2, 1987, sec A, p 1.
6. The Holy Scriptures. Second Kings iv, 32–37.
7. Vesalius A: Pulmonis motuum de humani corporis fabrica libri septem. *Basel Oporinus*, 1543, p 658.
8. Schacter EN: Respiratory care. Assessing the benefits [editorial]. *Arch Intern Med* 1983; 143:428.
9. Hedemark LL, Kronenberg RS: Chemical regulation of respiration. Normal variations and abnormal responses. *Chest* 1982; 82:488–494.
10. Severinghaus JW: Chemical regulation of ventilation: Who needs it? [editorial]. *N Engl J Med* 1976; 295:895–896.
11. Rohrer F: Physiologie der Ademberwegung, in *Hanbuch der normalen und pathologischer Physiologie*. Berlin, Springer-Verlag, 1925, vol 2, pp 70–127.
12. Pinkney DS: New York passes DNR procedure law. *American Medical News*, July 24, 1987, p 1.
13. State of New York (Senate 413-A, Assembly 678-A) Article 29-B. Act to amend the Public Health Law, as prefiled, January 7, 1987 (in Section 2961, Definitions, #4).

Withdrawing fluids and nutrition: An alternate view

In a landmark statement regarding its policy on terminating treatment, the American Medical Association's (AMA) Council on Ethical and Judicial Affairs announced in 1986 that it is ethically acceptable for physicians to withdraw artificial feeding and hydration from terminally ill or permanently comatose patients, provided certain conditions are satisfied, including the accuracy of the diagnosis, the irreversibility of the coma, and the knowledge of the patient's wishes.[1] The AMA is thus clearly on record as concluding that artificial feeding and hydration should be considered no different from other forms of medical treatment and may ethically be discontinued if physicians deem the treatment to be of no further benefit and/or a burden to the dying patient.

I believe that the provision of nutrition and hydration by feeding tubes or intravenous lines is not medical treatment, but supportive care no different from washing, turning, or grooming a dying patient. There is a morally significant difference between medical treatment and supportive care. Supportive care is always required, regardless of the benefit/burden relationship, whereas medical care must be given when the possibility exists, however remote, of restoring health by improving or reversing the medical illness.

In 1976, the New Jersey Supreme Court authorized the removal of Karen Ann Quinlan's respirator. When Quinlan's father was asked whether he wanted her nasogastric tube feedings stopped, he expressed amazement, saying, "Oh, no, that is her nourishment," clearly making a sharp distinction between the provision of nutrition and the respirator.[2] Yet an extensive literature has emerged in the past several years supporting the notion that providing nutrition and hydration by intravenous lines or nasogastric tubes constitutes medical treatment no different from antibiotics, transfusions, or other forms of medical intervention, including respirators or other mechanical means of life support, and that such treatment should only be used if it benefits the patient, not if it is useless and/or futile and is a great burden on the patient. Why the sudden about-face in our ethical, medical, and legal thinking? Where does one draw the line? Can oral feeding and hydration also be withheld from patients who are able to eat and drink? What about ice sucking or lip moistening? Why is the practice of withdrawing or withholding fluids and nutrition gaining support from bioethicists, physicians, nurses, and other health care providers? Why is this practice no longer considered to be morally objectionable? Why is feeding a patient different from providing skin care, bowel and bladder care, grooming, alcohol rubs, and other general supportive measures?

Although a rare author pleads for caution in approaching this issue, few articles speak out against this unexpected development which runs counter to the tradition of medical care. In an attempt to prevent the all too rapid acceptance of this new, emergent, medical practice of withholding or withdrawing fluids from some classes of patients, Siegler and Weisbard[3] argue that "this development bears the seeds of great potential abuse" and "may threaten patients, physicians, the patient-physician relationship, and other societal values." Continuing to provide fluids, even to dying patients, "provides an important clinical, psychological, and social barrier that should be retained." Derr[4] cites six mutually reinforcing biological, social, historical, and ethical considerations which combine to provide a clear and plausible distinction between the withholding of food and fluids and the withholding or withdrawal of medical or surgical therapy. First, the denial of food and fluids is biologically final, in that it will certainly and directly lead to the patient's death, since survival without food and fluids is impossible. Second, food and fluids are universal human needs, whereas modern medical and surgical therapy is not. Third, the physician-patient relationship may be seriously harmed, since the patient's presumption is that physicians always aim to preserve life and never to induce death. Fourth, to permit physicians to deny food and fluid to patients who are capable of receiving and utilizing them directly attacks the very foundation of medicine as an ethical profession. Fifth, the denial of food and fluids administered by "artificial" means is no different from such denial when food can be administered in a "normal" manner. The food provided is not transformed into an exotic medical substance by the simple act of pouring it into a gastrostomy tube. Sixth, the food and fluid given to a handicapped person or to a dying patient does not become medical therapy simply because another person is needed to provide it. Derr's position and arguments have considerable merit and are worthy of further discussion.

Judeo-Christian teaching proclaims the sanctity of human life. The physician is given divine license to heal but not to hasten death. When a physician has nothing further to offer a patient medically or surgically, the physician's license to heal ends and he becomes no different from a lay person. Every human being is morally expected to help another human in distress. A dying patient is no exception. The physician, family, friends, nurses, social workers, and other individuals close to the dying patient are all obligated to provide supportive care, including psychosocial and emotional care, until the very end. Fluids and nutrition are part and parcel of that supportive care, no different from walking, turning, talking, singing, reading, or just listening to the dying patient. There are times when specific medical and/or surgical therapies are no longer indicated, appropriate, or desirable for a terminal, irreversibly ill, dying patient. There is no time, however, when general supportive measures can be abandoned, thereby hastening the patient's demise.

In the opinion of this writer and of others,[5-7] the Judicial Council of the American Medical Association is in error in equating the provision of "extraordinary" life support technology with "ordinary" feeding and hydration. Thus, "pulling the plug" is equated by the AMA with "pulling the tube," and permissiveness in the new and revolutionary AMA guidelines represents the equivalent of negative euthanasia.[5] The withholding or withdrawing of nutrition and fluid should not be practiced, since death by starvation, dehydration, volume depletion, or a combination of these is not death from the underlying disease process but rather from euthanasia, which is still illegal in this country.[5] Another writer argues that opportunities for the implementation of significant future advances in the rehabilitation of neurologically impaired persons will be lost if it becomes standard practice to withdraw food and water from such patients.[7] Such practice actually causes the patient's death by culpable omission.

The United States is rapidly becoming a country where human rights seem to take precedence over all other considerations. People have a right to privacy, a right to health care, a right to refuse treatment, a right to die, a right not to be resuscitated, and many other rights. Yet Judeo-Christian principles consider people to have obligations rather than rights: an obligation to live, an obligation to seek healing, an obligation to be cured of illness, an obligation to help one's fellow man. Suicide is a crime in Anglo-Saxon law as well as in religious teaching. Scarce resources make the absolute patient right to have access to all sophisticated biomedical technology such as artificial hearts and experimental monoclonal antibodies impossible to achieve for all people.

The state has both the right and the obligation to interfere with an individual's rights under certain circumstances. The state can mandate immunization for all its citizens, or compulsory treatment of patients with tuberculosis, or the compulsory wearing of seat belts. Even when no other person is involved, the state has at least the moral obligation of protecting individual citizens from their self-destruction.

Many legal jurisdictions and state medical and legal societies have enacted or are considering the adoption of guidelines on forgoing (ie, withdrawing or withholding) life-sustaining treatment. The slippery slope has now reached the point where we are reclassifying the provision of basic supportive care such as fluids and nutrition as medical treatment to justify withholding or withdrawing it in certain cases. Where will the trend end? Will we soon consider active hastening of a person's death by a lethal injection to be acceptable legally and/or medically? Where will such decision-making lead us in relation to the senile, the elderly, the incompetent, the terminally ill, the malformed newborn? Are we willingly condemning such patients to death to allow them to "die in dignity?" Is it not much more dignified to live? The dying and the dead must be treated with dignity, but it is even more dignified to live. Are there, lurking in the decisions to deny or withdraw fluids and nutrition from severely handicapped newborns, thoughts about this potential source of organ donors?

Most courts of final jurisdiction that have ruled on the issue of withdrawing or withholding fluids and nutrition from permanently comatose patients take the position that the right to discontinue treatment is a personal decision, and that the right to die is an integral part of our right to control our own destinies, so long as the rights of others are not affected. The AMA's Judicial Council, the President's Commission, and many writers cited in this commentary agree with this approach.

My position is that even if the courts legally sanction the withdrawal or withholding of fluids and nutrition in some instances, legal permissibility is not synonymous with moral license. What is legal is not always moral. The social, ethical, religious, biologic, and public policy issues involved in this sensitive and controversial area of human life and death have not been fully explored and examined. Caution is urged before the acceptance of fluid and nutrition as medical treatment rather than supportive care becomes uncontrollable, like a snowball rolling down a hill.

FRED ROSNER, MD
Director
Department of Medicine
Queens Hospital Center
Affiliation of the Long Island
Jewish Medical Center
Jamaica, NY 11432

Professor
Department of Medicine
State University of New York
Health Sciences Center at
Stony Brook
Stony Brook, NY 11794

1. Dickey NW: Withholding or withdrawing treatment [letter]. *JAMA* 1986; 256:471.
2. Ramsey P: The Quinlan decision: Five commentaries. Prolonged dying: Not medically indicated. *Hastings Cent Rep* 1976; 6:14-17.
3. Siegler M, Weisbard AJ: Against the emerging stream. Should fluids and nutritional support be discontinued? *Arch Intern Med* 1985; 145:129-131.
4. Derr PG: Why food and fluids can never be denied. *Hastings Cent Rep* 1986; 16:28-30.
5. White RJ: Economic pressure reshaping medical ethics. *Am Med News* 1986; 29(35):24.
6. Davila F: Withholding or withdrawing treatment [letter]. *JAMA* 1986; 256:470-471.
7. Barry R: Withholding or withdrawing treatment [letter]. *JAMA* 1986; 256:469-470.

Incidence of placenta previa and abruptio placentae in New York State

ELINA HEMMINKI, MD; DONNA M. GLEBATIS, MS; GENE D. THERRIAULT, MSPH; DWIGHT T. JANERICH, DDS

ABSTRACT. The purpose of this research was to study the incidence of placenta previa and abruptio placentae in New York State, exclusive of New York City. Data from certificates of live births and hospital discharge records were used. Estimated from the certificate of live birth data, the incidence of abruptio placentae did not markedly change during the period 1976 through 1982; the data for placenta previa suggested a slight decrease in incidence. From 1980 through 1983, there were 9.9 discharges from hospitals with a diagnosis of placenta previa per 1,000 live births and 10.3/1,000 with a diagnosis of abruptio placentae. These rates differed by Health Service Area. The data from certificates of live birth showed a difference in the rates of placenta previa and abruptio placentae by age, gravidity, method of delivery, and history of previous induced abortions.

(NY State J Med 1987; 87:594–598)

The two major placental attachment problems are placenta previa and abruptio placentae. Placenta previa refers to the placenta's being located over or near the internal cervical os; abruptio placentae is the premature separation of the normally implanted placenta. Previous studies of placenta previa have shown incidence rates that vary from 3 to 6.5 per 1,000 deliveries/births; for abruptio placentae, the incidence rates were from 4.9 to 20.5 per 1,000 deliveries/births.[1-11] This paper presents recent estimates of the occurrence of placenta previa and abruptio placentae in New York State using two different data sources, hospital discharge records and birth certificates. (Birth certificates from the City of New York were not used because they are collected and stored separately from those of the rest of the state.) This study was considered important because most of the previous studies are dated, and the newer estimates based on single hospitals may be biased due to selective admissions. Placek and Taffel[3,4] and Mosher and Keppel[5] give recent national estimates, but they provide no detailed break-down.

PATIENTS AND METHODS

Hospital Discharge Records. Since 1980, information on all patients discharged from general hospitals in New York State has been collected through the statewide planning and research cooperative system (SPARCS), maintained by the New York State Department of Health. The unit of observation in SPARCS is a hospital discharge, thus patients may appear several times in the data base if they are hospitalized several times during a year. For each discharge, the main reason for the hospital stay is coded as the principal diagnosis using the clinical modification of the International Classification of Diseases (ICD).[12] Secondary diagnoses (up to four) that contribute to the hospital stay are also noted. The coding of diagnosis is performed in the hospitals. In the case of several complications, classification into principal and secondary diagnosis apparently depends largely on how the people completing the hospital records view the importance of each. For this study, all hospital discharge records with diagnoses of placenta previa (ICD codes 641.0, 641.1) and abruptio placentae (641.2), either as a primary or a secondary diagnosis, were obtained for New York State for the years 1980–1983, and rates per live births were calculated.

Certificates of Live Birth. The certificate of live birth in New York State includes information on complications during labor and delivery. Since 1976, placenta previa and abruptio placentae have been specified as the first two of the 15 complications listed on the certificate. All records indicating these placental problems were obtained for all recorded births in New York State, excluding New York City, during the period 1976–1982; and rates per 1,000 live births were calculated.

RESULTS

Figure 1 gives the rates of placenta previa and abruptio placentae in New York, excluding New York City, for each year from 1976 to 1982, as derived from certificates of live birth, and from 1980 to 1983 as derived from SPARCS data. The incidence of placenta previa decreased 24% from 1978 to 1982, but the rate of abruptio placentae fluctuated and has shown a less clear trend. Figure 1 also shows that SPARCS reported more placental problems than did the birth certificates. The difference between these two data sources was larger for placenta previa (2.6 times more among the hospital discharges) than for abruptio placentae (1.6 times more).

Figure 2 shows the geographical distribution of placenta previa and abruptio placentae by Health Service Areas (HSA) for hospital discharges from 1980 to 1983. The rate of placenta previa ranged from a low of approximately 7/1,000 in HSA2 to a high of 13/1,000 for HSA5. Abruptio placentae ranged from a low of 7/1,000 in HSA7 to a high of 17/1,000 in HSAs 3 and 4.

Data from the upstate records of live births were pooled for the years 1976–1982, and the following variables were examined for

---

From the Department of Public Health, University of Tampere (Dr Hemminki), Tampere, Finland, and the New York State Department of Health, Division of Epidemiology, Albany, NY (Dr Janerich, Ms Glebatis, and Mr Therriault).

Address correspondence to Ms Glebatis, Research Scientist, Division of Epidemiology, State of New York Department of Health, Corning Tower, Room 511, Governor Nelson A. Rockefeller Empire State Plaza, Albany, NY 12237.

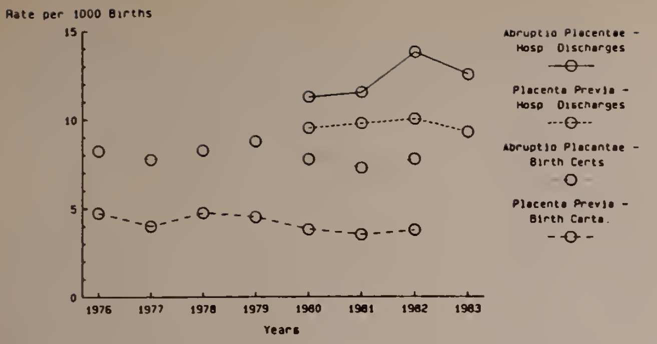


FIGURE 1. Rate of placenta previa and abruptio placentae per 1,000 live births for hospital discharges from 1980 to 1983 and for birth certificates from 1976 to 1982 for New York State, exclusive of New York City.

both placenta previa and abruptio placentae: maternal age, gravidity, race, previous cesarean section, and previous induced abortion. Figure 3 presents the rate of placenta previa per 1,000 live births by maternal age for the three gravidity groups 0, 1–2, and 3+ previous pregnancies. For all three subgroups, the greatest increase in the rate occurred after age 30. For gravidity 0 and 1–2 there was a steady increase in the rate up to age 30, however, for gravidity 3+ an increase is less obvious. Figure 3 also shows the rate of placenta previa per 1,000 live births by gravidity for the two age groups <35 years and 35+ years. These two age categories were chosen because the four age groups <19, 20–24, 25–29, and 30–34 all showed a similar pattern to each other, as did the two older age groups, 35–39 and 40–44. For patients less than 35 years of age, the rate increased steadily with gravidity. However, for patients 35 years of age and older the pattern was U-shaped, with the rate decreasing from gravidity 0 to gravidity 1, then increasing for gravidity 2 and 3+.

Figure 4 presents the rate of abruptio placentae per 1,000 live births by maternal age for the three gravidity subgroups 0, 1–2, and 3+ previous pregnancies. All three subgroups of gravidity showed a U-shaped pattern; the rates being highest for patients less than 20 years old and more than 35 years old. Figure 4 also shows the rate of abruptio placentae per 1,000 live births by gravidity for the two age groups <25 and 25+. Again, these two age categories were chosen because the age groups within each category showed similar patterns, allowing them to be pooled together. For patients under 25 years of age, the rate increased with increasing gravidity. For patients 25 years of age and older, the rate decreased from gravidity 0 to 1, but again increased with gravidities 2 and 3+.

Table I presents the rate of placenta previa and abruptio placentae by race. As can be seen, there appears to be little difference in the rate among subgroups of race for either condition. Table II shows the rate of placenta previa and abruptio placentae per 1,000 live

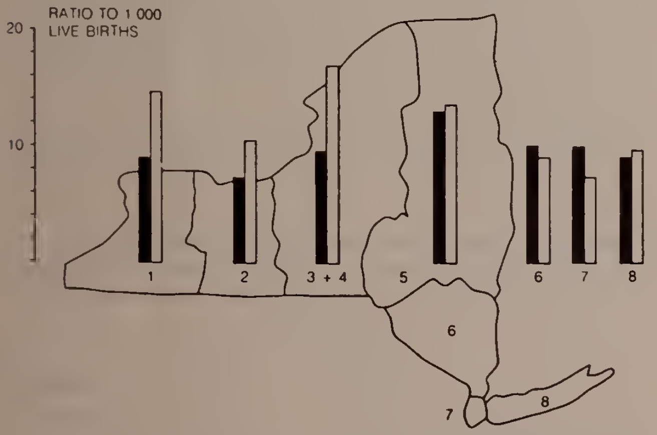


FIGURE 2. Rate of placenta previa (solid bars) and abruptio placentae (open bars) per 1,000 live births by Health Service Areas, based on hospital discharge records from 1980 to 1983.

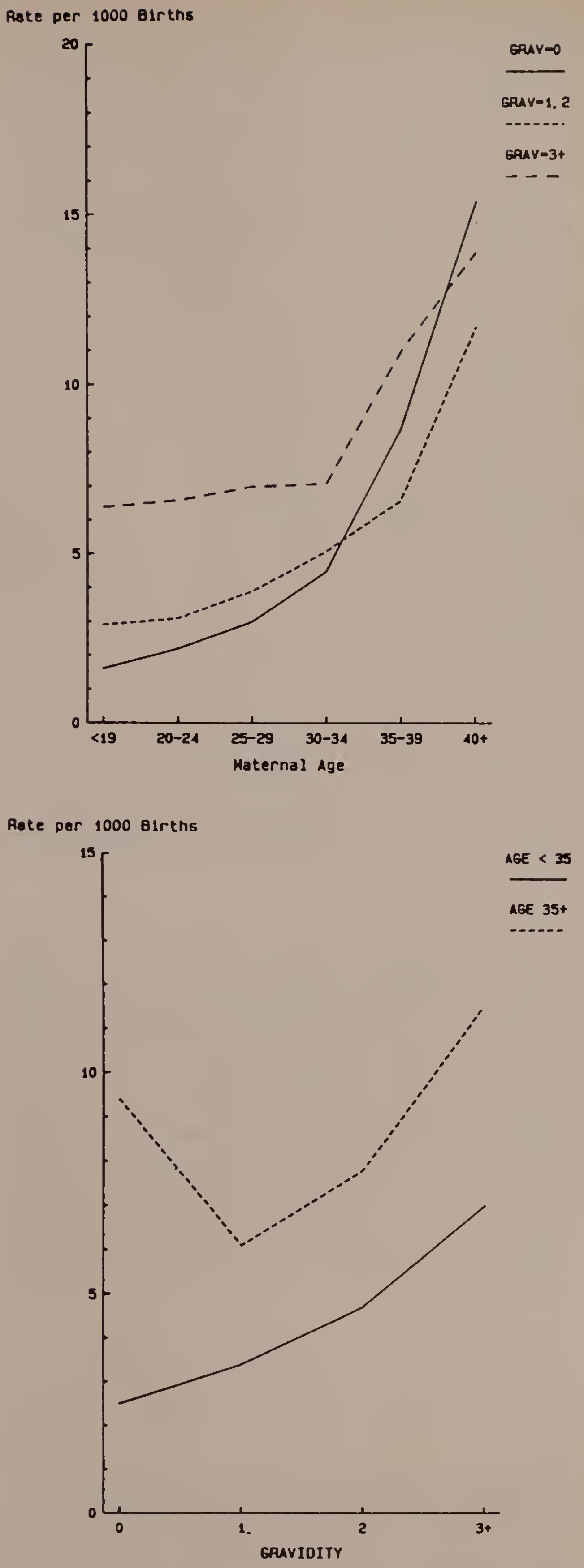


FIGURE 3. Rates of placenta previa per 1,000 live births by maternal age **(top)** and gravidity **(bottom)**. Rates are based on recorded births in New York State, excluding New York City, for 1976–1982.

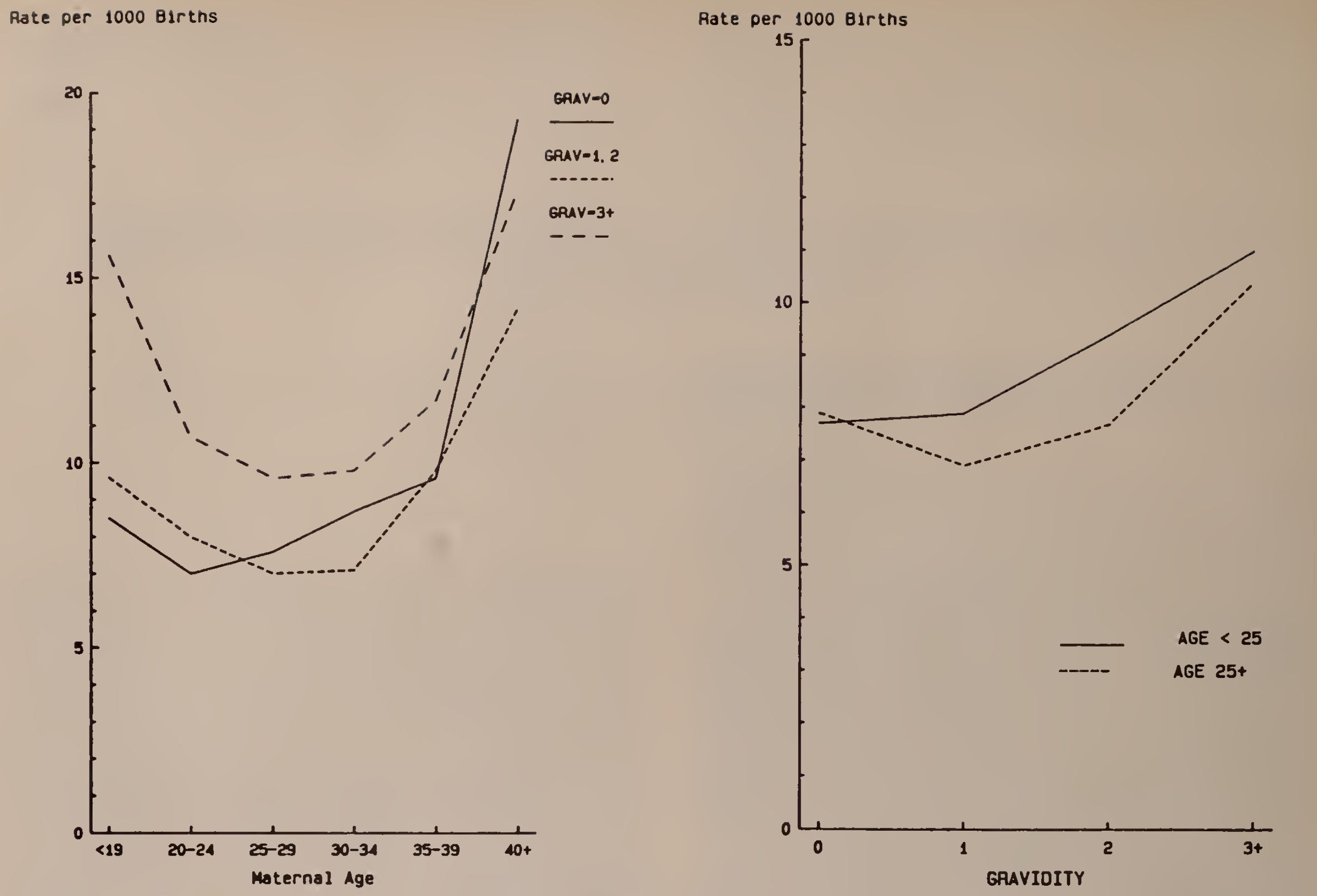


FIGURE 4. Rates of abruptio placentae per 1,000 live births by maternal age (left) and gravidity (right). Rates are based on recorded births in New York State, excluding New York City, for 1976–1982.

births by method of delivery. As would be expected, the rates were highest for women delivering by a primary cesarean section. While the rates for both placenta previa and abruptio placentae were higher for repeat cesarean sections compared to spontaneous delivery, the difference was greater for placenta previa than for abruptio placentae. Because most women who had had a previous cesarean section commonly had a repeat cesarean section, there is no selection bias regarding placental problems. The rate of placenta previa and abruptio placentae per 1,000 births by previous induced abortions is shown in Table III. The rate of placenta previa increases directly with the number of previous abortions. This is most likely a reflection of an increase in placenta previa with overall gravidity rather than a function of previous induced abortions. The rate of abruptio placentae, however, does not increase with the number of previous abortions, except among patients who had had three or more previous terminations.

Discussion

The incidence of placenta previa as estimated from SPARCS data (9.9 cases per 1,000 live births) was much higher than the incidences given in earlier studies. For abruptio placentae, earlier literature has given varying incidence rates, and the estimate of this study from the SPARCS data (10.3 cases per 1,000 live births), is within the range previously cited. The SPARCS data may have overestimated the incidence of placenta previa occurring at the time of delivery, because it includes women who were suspected of having placenta previa during pregnancy, and whose placenta previas moved upwards before delivery. In addition, a woman may have had several visits to the hospital. Overestimation of abruptio placentae due to visits before delivery is less likely since it is less frequently diagnosed during pregnancy.

The finding that the incidence rates estimated from the certificates of live birth were lower than those obtained

TABLE I. Rate of Placenta Previa and Abruptio Placentae per 1,000 Live Births for 1976–1982 Recorded Births for New York State, Excluding New York City, by Race

	White	Black	Other	Total
Placenta previa	4.1	4.2	4.6	4.1
Abruptio placentae	8.1	8.4	7.3	8.1

TABLE II. Rate of Placenta Previa and Abruptio Placentae per 1,000 Live Births for 1976–1982 Recorded Births for New York State, Excluding New York City, by Method of Delivery

	Primary Cesarean Section	Repeat Cesarean Section	Spontaneous Delivery
Placenta previa	21.0	16.0	0.9
Abruptio placentae	23.7	14.7	5.1

TABLE III. Rate of Placenta Previa and Abruptio Placentae per 1,000 Live Births for 1976–1982 Recorded Births for New York State, Excluding New York City, by Previous Induced Abortions and Total Previous Pregnancies

	Previous Induced Abortions				Total Previous Pregnancies			
	0	1	2	3+	0	1	2	3+
Placenta previa	4.0	4.6	7.6	9.3	2.6	3.5	4.9	7.9
Abruptio placentae	8.1	7.3	8.4	10.1	7.8	7.3	8.2	10.5

from the SPARCS data was to be expected. In certificates of live birth, underreporting of medical problems may be notable,[13,14] and pregnancies that end in fetal death are not included. In Sweden in 1977–1978, of the 2,061 deliveries with a diagnosis of placenta previa or abruptio placentae, 10% ended with perinatal death; the proportion of fetal deaths was not specified.[15] However, it is assumed that the underreporting had been similar over the years and consistent among subgroups, allowing for an examination of time trends and variations among subgroups.

In addition to the problems of these routinely collected data, studying the incidence of placenta previa and abruptio placentae is impeded by inherent problems of diagnosis. The distinction between a low-lying placenta and placenta previa is not clear. When placenta previa is asymptomatic, its finding depends on when and how frequently ultrasound is used, and how often elective cesarean sections are done. Frequent early screenings with ultrasound tend to result in overdiagnosis of placenta previa, because those cases found before delivery tend to "move" during pregnancy and reach a normal position before delivery.[16,17] In elective cesarean sections, placenta previa may be overlooked because the cervix is not dilated and less of a chance for bleeding exists. The differential diagnosis of other causes of antepartum hemorrhage, especially abruptio placentae, is not easily made by clinical signs. Also, abruptio placentae in its mild and partial form is hard to diagnose and to distinguish from other causes of antepartum bleeding.

The causes of placenta previa and abruptio placentae are poorly understood. The risk of placenta previa has been found to increase with increasing parity, age, smoking, previous abortion, and previous cesarean section, and is greater with male and twin infants.[1,6,8,11,18] In this study, an association was also noted between both placenta previa and abruptio placentae and maternal age, gravidity, and previous cesarean section. The risk ratio of placenta previa of women over 34 years to that of women under 25 years has been reported to be 2.8[8] and 2.3,[1] respectively, which is comparable to the risks found in this study (2.6). As in the study by Brenner et al,[8] we also noted that the age effect was most marked among women having their first child.

The incidence of placenta previa and abruptio placentae varied markedly in the different health service areas, which provides a stimulus for further studies to quantify the known risk factors of these placental problems and to identify new risk factors. For example, abruptio placentae, which is associated with hypertension, dietary deficiency, smoking, and uterine anomalies,[18] occurred less frequently in New York City than in the health service area 3+4. It is unlikely that women in the city of New York have fewer of these health problems than women in the health service area 3+4, and new etiologic factors should be sought. However, these area differences may be due to differences in diagnosis and recording.

Data from the New York State Vital Statistics demonstrate that the population of women having children in New York State has changed in the last ten years. The finding of only small changes in the incidence of these placental problems over the years is unexpected. It may have resulted from concurrent changes in risk factors in opposite directions, or the changes may have been overcome by competing trends in the accuracy of diagnosis and recording. For example, parity has decreased, but previous cesarean sections have become more common, and the average maternal age has increased.

On the basis of the data reported here, it can be estimated that in the early 1980s, about 2% of births in New York State were complicated by placenta previa and/or abruptio placentae. These problems are often serious, resulting in high infant and maternal mortality and morbidity. Further research to study the cause of these disorders, in particular, the impact of cesarean sections, is needed. Cesarean sections have become increasingly common (21% of all live births in upstate New York in 1983), and our results support previous reports showing increased risk of placenta previa after a previous cesarean section.[8,11,16,19] We found no studies that investigated the effect of cesarean section on abruptio placentae, but our results did show an increased rate of abruptio placentae among women having a repeat cesarean section compared to women having a spontaneous delivery.

Acknowledgments. The authors thank Andrew Stacy, Barry Graubard, and Akseli Hemminki for their help in obtaining and analyzing data, and Anna-Liisa Kulo for typing the report. Part of this work was done while Dr Hemminki was Visiting Scientist in the Biometry Branch, National Institute of Child Health and Human Development, National Institutes of Health.

REFERENCES

1. Record RG: Observations related to the aetiology of placenta praevia with special reference to influence of age and parity. *Brit J Prev Social Med* 1956; 10:19–24.
2. Knab DR: Abruptio placentae. An assessment of the time and method of delivery. *Obstet Gynecol* 1978; 52:625–629.
3. Placek PJ, Taffel SM: The frequency of complications in cesarean and noncesarean deliveries, 1970 and 1978. *Public Health Rep* 1983; 98:396–400.
4. Taffel SM, Placek PJ: Complications in cesarean and non-cesarean deliveries: United States, 1980. *Am J Public Health* 1983; 73:856–860, and unpublished data from the 1980 National Hospital Discharge Survey.
5. Mosher WD, Keppel KG: Social and clinical correlates of postpartum sterilization in the United States, 1972 and 1980. *Public Health Rep* 1984; 99:128–138.
6. Bender S: Placenta previa and previous lower segment cesarean section. *Surg Gynecol Obstet* 1954; 98:625–628.
7. Naeye RL: Abruptio placentae and placenta previa: Frequency, perinatal mortality, and cigarette smoking. *Obstet Gynecol* 1980; 55:701–704.
8. Brenner WE, Edelman DA, Hendricks CH: Characteristics of patients with placenta previa and results of "expectant management." *Am J Obstet Gynecol* 1978; 132:180–191.
9. Harlap S, Davies M: Late sequelae of induced abortion: Complications and outcome of pregnancy and labor. *Am J Epidemiol* 1975; 102:217–224.
10. Gorodeski IG, Bahari CM, Schachter A, et al: Recurrent placenta previa. *Eur J Obstet Gynaecol Reprod Biol* 1981; 12:7–11.
11. Singh PM, Rodrigues C, Gupta AN: Placenta previa and previous cesarean section. *Acta Obstet Gynecol Scand* 1981; 60:367–368.
12. *International Classification of Diseases, 9th Revision, Clinical Modification*. Ann Arbor, Mich, Commission of Professional and Hospital Activities, 1978.
13. Carucci PM: Reliability of statistical and medical information reported on birth and death certificates. New York State Department of Health monograph No. 15. Albany, NY, May 1979.

14. Frost F, Starzyk P, George S, et al: Birth complication reporting: The effect of birth certificate design. *Am J Public Health* 1984; 74:505-506.

15. Bakketeig LS, Hoffman HJ, Titmuss Oakley AB: Perinatal mortality, in Bracken MB (ed): *Perinatal Epidemiology*. New York, Oxford University Press, 1984.

16. Newton ER, Barss V, Cetrulo CL: The epidemiology and clinical history of asymptomatic midtrimester placenta previa. *Am J Obstet Gynecol* 1984; 148:743-748.

17. Rizos N, Doran TA, Miskin M, et al: Natural history of placenta previa ascertained by diagnostic ultrasound. *Am J Obstet Gynecol* 1979; 133:287-291.

18. Naeye RL: Placenta previa. Predisposing factors and effects on the fetus and surviving infants. *Obstet Gynecol* 1978; 52:521-525.

19. Hemminki E: Pregnancy and birth after cesarean section. A survey based on the Swedish birth register. *Birth*, in press.

FROM THE LIBRARY

PLACENTA PRAEVIA

Of the danger to the female, in unavoidable hemorrhage, from presentation of the placenta, Professor Simpson says, that the "peril of life is as great as though the patient were seized with yellow fever, or malignant cholera; that it is twice as dangerous and fatal as the operation of lithotomy—one in every three perishing under the former, and one in every six or eight perishing under the latter." Dr. Meigs compares the danger to that from the use of fire-arms. He believes the peril to the woman to be greater than it would be, were a pistol ball fired at her head at the distance of a few paces.

The testimony of the earlier obstetric authors, Rigly, Collins, Dewees, and others, is in accord with the more recent writers, in regard to the extreme peril in this complication in midwifery—this "error in nature."

Whether or not, with more knowledge, if such were possible, and with improved methods for the arrest of flooding, these extreme fears are to be shared, in large degree, by obstetricians of the present day, or by those who are to come after us, admits of some shadow of doubt. Most certainly has the climax of the opprobrium medicorum been reached, if science, or art, or experience, are powerless to bring abatement to the necessity for such fears. I am not yet without hope that such essential advances will be made in this branch of the science and art of midwifery, as will ultimately lead, at least, to the abatement of the peril, if not, finally, to immunity from danger and death; to this end, any personal observation or experience, or even suggestion, having relation to and bearing upon the subject, should be gladly hailed and welcomed by all who practice a humane profession.

To know the cause or causes of abnormal implantation, however desirable such knowledge would be, would certainly not help us in the least in efforts to ward off fatal results. Nevertheless, we would like much to be put into possession of the cause, or causes, with the hope that the knowledge thereof would enable us to prevent what we cannot remedy. The question of etiology, perhaps, comes more properly within the province of the embryologist.

Those curious to receive more light upon the subject matter, may be referred to the writings of Coste Sur Histoire générale et particulière du développement des corps organisés, or of Poucket Sur Théorie positive de la fécondation des mammifères et de l'espece humaine, etc. . . .

Our knowledge, in the main, has been gradually developed, but occasionally it has been marked by periods or epochs which have startled the careless and indifferent. Although the investigations of one person may afterward be proved futile and set at naught by the investigations of another; both are, perhaps, equally entitled to merit, for the former theory, which was false, may have incited to labors which proved the old theory false and another true, so that, on the whole, nothing is lost, but much gained.

The weary traveler, having taken the wrong road and returning reaches his starting point, is enabled to direct his fellow traveler aright, and dispel all doubts as to the proper course to pursue; the blunder, stupidity or failure of the one has been the means of success to the other. . . .

C. C. F. GAY, MD

(*Trans NY State Med Soc* 1869; 219-228)

Myelofibrosis, renal vein thrombosis, and pulmonary emboli

Case Presentation

Richard J. Horner, MD (Attending Physician, Hematology and Oncology): A 76-year-old woman was admitted to the Mary Imogene Bassett Hospital with shortness of breath, profound weakness, and radiographic evidence of a right middle lobe infiltrate.

The patient had been observed at this hospital for many years. In 1973, physical examination disclosed a questionably palpable spleen; the peripheral blood smear demonstrated multiple abnormalities, and the bone marrow showed mild fibrosis. The findings supported the diagnosis of myelofibrosis with myeloid metaplasia.

In 1975, the patient noted a change of bowel habits; several polypoid lesions were found in various segments of the colon, together with a moderately well differentiated mucin-secreting sigmoid carcinoma, Duke's Stage B II. A total colectomy with ileoproctostomy was performed. The neoplasm had penetrated the full thickness of the bowel wall; this invasiveness, together with the tumor cells' ability to secrete mucin, suggested a poor prognosis. However, the neoplasm was well differentiated and, during the rest of the patient's life, there was no indication of recurrence.

Between 1976 and 1981, the patient was vigorous and active, but her spleen was increasing in size. By early 1982, she was experiencing recurrent pain in the splenic area, several episodes of which were clearly caused by splenic infarction. Her hematocrit was in the 35% range, the white blood cell count was 55,000–60,000/μL, and the platelet count was 80,000–90,000/μL. The blood smear showed numerous nucleated red blood cells and immature myeloid precursors. There was also evidence of congestive heart failure. In January 1982, chiefly because of the splenic pain, the patient underwent splenectomy, with good recovery and significant improvement in symptoms.

Thereafter, the patient did well clinically until the spring of 1984, when shortness of breath and chest pain developed during a trans-Pacific flight. Following hospital admission in San Francisco, a detailed diagnostic evaluation for possible pulmonary embolism, including pulmonary angiography, failed to support that diagnosis.

During the next six months, the patient experienced intermittent chest pain, for which she was seen at this hospital in December 1984. At that time, her blood pressure was 230/92–120 mm Hg. Despite adjustment of antihypertensive medications, she continued to experience persistent chest pain, shortness of breath, and weakness. Within the month, she was again admitted and was found to have a nephrotic syndrome with excretion of 5.9 g of protein per 24 hours. The serum creatinine level was 2 mg/dL, the blood urea nitrogen (BUN) level was in the range of 25–30 mg/dL, and the creatinine clearance was calculated at 15 mL/min. Her chest film showed a right pleural effusion, which proved to be exudative.

A renal venogram disclosed right renal vein thrombosis. Dr Michael Schwartzman, nephrology consultant, thought that the patient had membranous glomerulonephropathy, and postulated that her nephrotic syndrome might be causing excessive loss of antithrombin III. This in turn might have led to intravascular thromboses and to the recurrent pulmonary emboli during the preceding eight months. Anticoagulant therapy was begun, and the patient was discharged after one month in the hospital.

For the next several months, the patient did well, but she was again readmitted in July 1985 because of shortness of breath and recurrent chest pain. A chest film demonstrated a left lower lobe infiltrate; pulmonary angiography confirmed the presence of pulmonary embolism.

In early August 1985, repeat venocavogram demonstrated clearing of the renal vein thrombosis; because a pulmonary embolism had developed while the patient was on anticoagulant therapy, a Greenfield filter was inserted into the inferior vena cava below the level of the renal veins. The operation was uneventful, but five days later the patient experienced a hypertensive crisis with blood pressures in the range of 220/110 mm Hg and lost consciousness. A computed tomographic (CT) scan revealed dilated cerebral ventricles; a ventricular shunt was placed, with prompt recovery of consciousness and no residual neurologic defect. Cerebral ventricular fluid was clear, although spinal fluid obtained via lumbar puncture on the following day was pink and xanthochromic. The etiology of the episode of loss of consciousness remained unclear; the suspicion was that the patient had experienced a subarachnoid hemorrhage, although the ventricular fluid obtained at the time of insertion of the shunt did not support that diagnosis.

Within the next three months, the patient required treatment for congestive heart failure and was readmitted to the hospital for treatment of right leg deep vein thrombosis. Again, recurrent pulmonary emboli were suspected, and mini-dose heparin therapy was continued during the following six months while she was in a nursing home.

Because of shortness of breath and profound weakness, the patient was again admitted to the hospital in April 1986. Her hematocrit was in the range of 30% to 35%; her

---

Based on a clinicopathologic conference (CPC) held at Mary Imogene Bassett Hospital, Cooperstown, NY, on August 12, 1986.

Editor: JOHN T. FLYNN, MD.

Address correspondence to Dr Flynn, Consulting Editor, *New York State Journal of Medicine*, PO Box 5404, 420 Lakeville Rd, Lake Success, NY 11042.

white blood cell count was 60,000/μL, with immature myeloid cells on blood smear. The BUN level was 67 mg/dL, and the serum creatinine level was 3.3 mg/dL. The chest film disclosed a dense right middle lobe infiltrate, which was thought to be either bacterial pneumonia or pulmonary infarction. The patient was treated with cefuroxime and heparin; but on the sixth day of hospitalization, clinical evidence of left leg deep vein thrombosis and gastrointestinal bleeding developed. The anticoagulant therapy was discontinued. Renal failure progressed rapidly, and the patient died 13 days after admission.

RADIOLOGIC PRESENTATION

Lawrence Barnowsky, MD (Attending Radiologist): Films from the gastrointestinal–small-bowel series in 1975 show a moderately enlarged spleen. A kidney, ureter, and bladder series following the splenectomy in 1982 shows the splenic flexure to be high in the abdomen; the bones are very dense, with smudgy trabeculae. With these changes and the earlier enlargement of the spleen, myelofibrosis with myeloid metaplasia should be suspected first. Lymphoma, metastatic neoplasm, and mastocytosis should be considered in the differential diagnosis.

In January 1985, a catheter was introduced into the right femoral vein and then into the right renal vein; contrast media was injected. This procedure demonstrated a filling defect in the right renal vein, which was caused by a thrombus that extended into the inferior vena cava (Fig 1). Again, the bones are seen to be markedly sclerotic on these films.

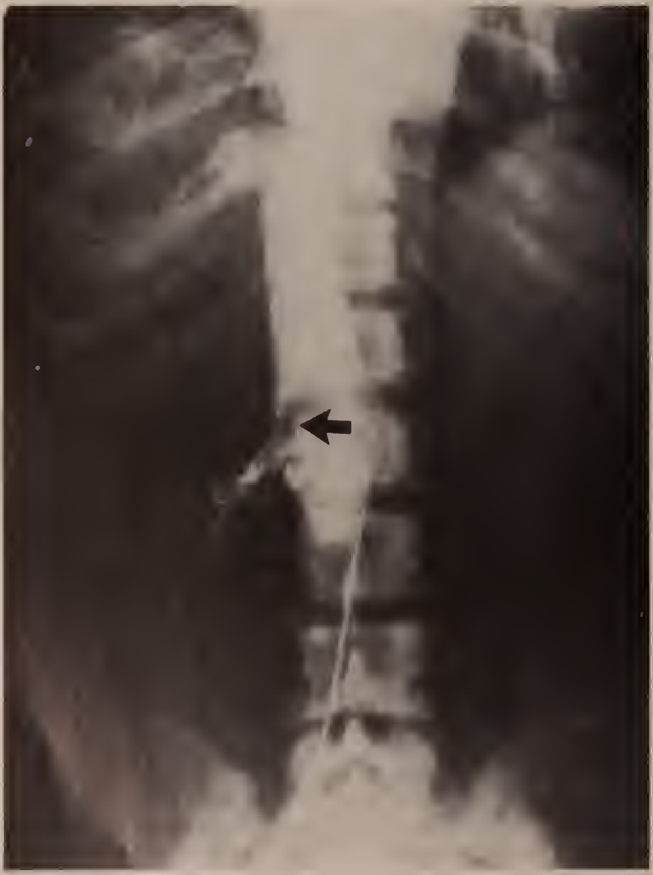


FIGURE 1. Right renal venogram showing thrombus in right renal vein (arrow), extending into the inferior vena cava.

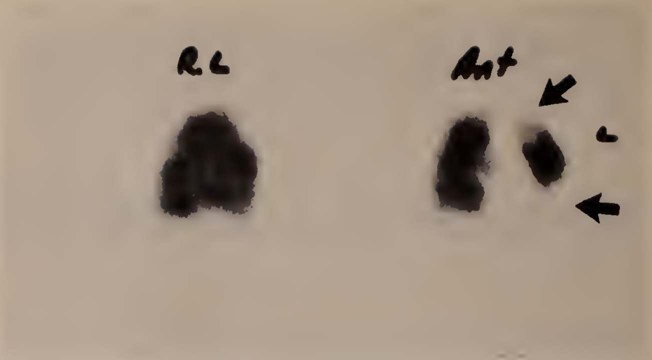


FIGURE 2. Perfusion lung scan (right lateral and anteroposterior views) indicating presence of emboli (arrows) in left upper lobe (superior segment and lingula).

In July 1985, a radionuclide perfusion lung scan disclosed a small subsegmental defect in the left upper lobe; the major finding, however, was a large defect in the lingula of the left upper lobe, compatible with pulmonary embolism (Fig 2). A left pulmonary angiogram confirmed that diagnosis (Fig 3). A follow-up renal venogram showed patency of the right renal vein.

A CT brain scan showed dilated ventricles and some periventricular leukomalacia, but no evidence of intracranial bleeding. Following the insertion of the ventricular

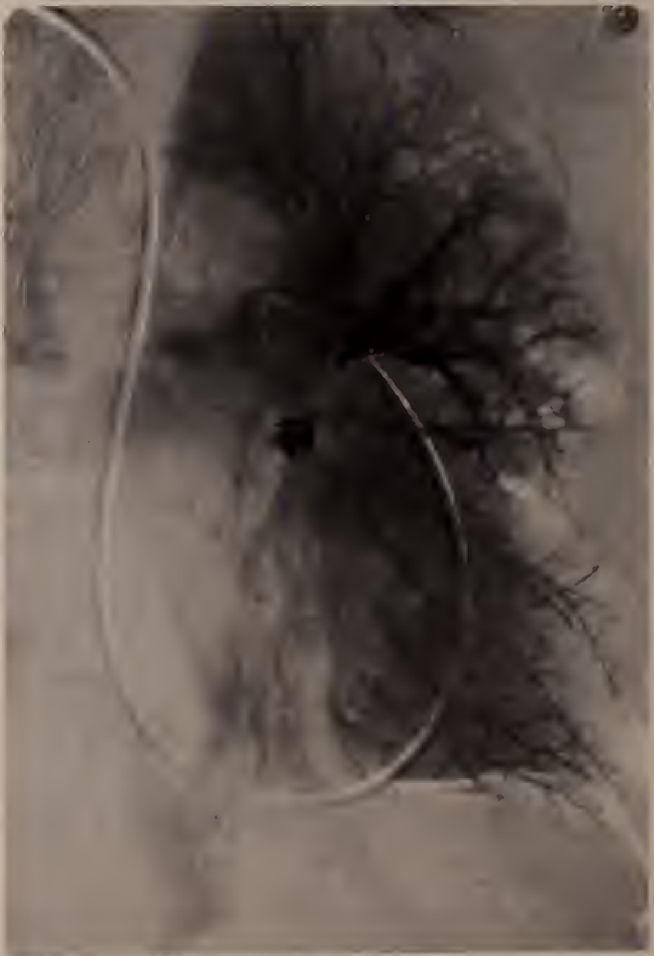


FIGURE 3. Left pulmonary angiogram showing cut-off of lingular branch of left pulmonary artery by embolus (arrows).

shunt, the CT scan showed a slight decrease in the size of the ventricles.

In September 1985, a venocavogram was repeated. Because of poor access from above the Greenfield filter, we elected to catheterize the vena cava from below. The catheter passed quite easily through the filter, and the injection of dye showed that the right renal vein was patent (Fig 4). But we also demonstrated a large thrombus adherent to the filter, probably generated in place rather than trapped by the device.

In April 1986, the chest film demonstrated a large heart, congestive pulmonary changes, an infiltrate in the right upper lobe, and a large infiltrate in the right middle lobe (Fig 5). The bones, including the ribs, were again seen to be quite dense.

Clinical Discussion

Dr Horner: This patient underwent total colectomy in 1975, not only because of her sigmoid cancer but because she had multiple polyps in other areas of the colon, notably in the cecum opposite the ileocecal valve. In addition, there were areas of hyperplastic colonic mucosa. By 1985, this patient had a strong family history of colon cancer, with her mother, two siblings, and her daughter afflicted. After review of the pedigree, the physicians caring for this patient were concerned that this family might represent a variant of the familial polyposis syndrome, and appropriate counseling of descendants was undertaken.

The patient's longstanding hematologic diagnosis was myelofibrosis with myeloid metaplasia. This disorder belongs to a family of diseases known as the myeloproliferative disorders, which is characterized by unrestrained clonal proliferation of one or more bone marrow cell lines, and by the frequent occurrence of splenomegaly and of bone marrow fibrosis.[1] The most common of the myeloproliferative disorders is chronic myelocytic leukemia (CML), which nearly always evolves into a condition resembling acute myelocytic leukemia (AML), which is resistant to therapy. The evolution to a syndrome resembling AML occurs in only 5–10% of the other myeloproliferative disorders.[2]

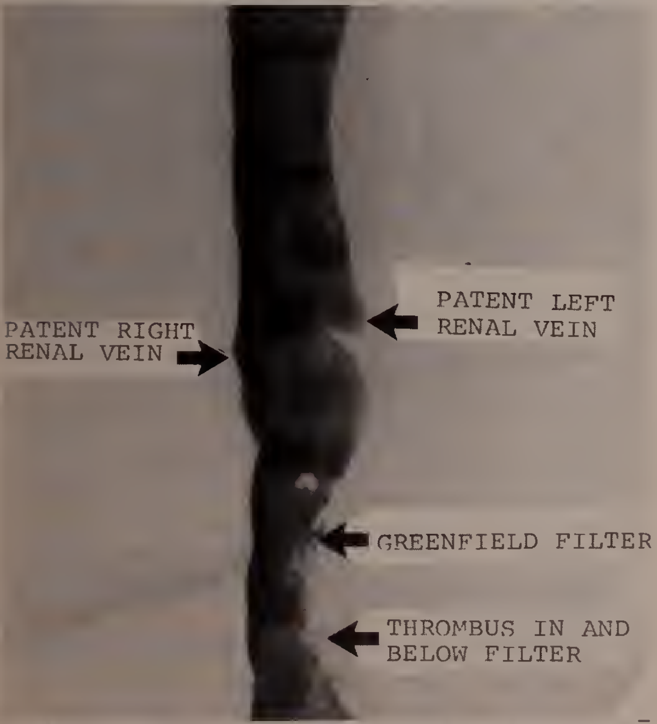


FIGURE 4. Inferior venacavogram showing patent renal veins and thrombus attached to Greenfield filter.

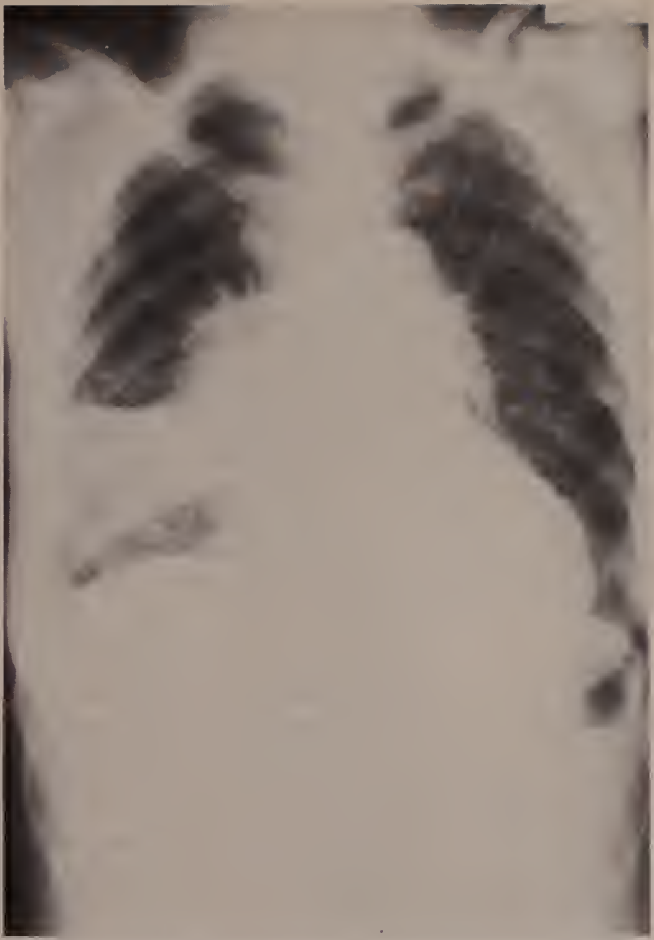


FIGURE 5. Posteroanterior chest film showing infiltrates in right upper and middle lobes, increased cardiac size, and bilateral pulmonary congestion.

Myelofibrosis with myeloid metaplasia is characterized by splenomegaly, the presence of immature myeloid elements, nucleated red cells, and teardrop poikilocytes in the peripheral smear, and by megakaryocytic hyperplasia and progressive fibrosis in the bone marrow. Splenomegaly gradually progresses, representing increasing blood production in the spleen. Ultimately, hematopoiesis may involve the liver, lymph nodes, and other parenchymal organs. Glucose-6-phosphate dehydrogenase (G6PD) isoenzyme studies have demonstrated the clonal origin of this disease.[3] There is evidence to suggest that the bone marrow fibrosis is a consequence of collagen-stimulating growth factor secreted by megakaryocytes.[4]

The differential diagnosis of myelofibrosis with myeloid metaplasia can be difficult. Marrow fibrosis and massive splenomegaly may characterize the late stages of CML. Tumor that metastasized to bone marrow, particularly breast and prostate carcinoma, may produce significant fibrosis and a leukoerythroblastic peripheral blood picture. However, in this situation there is almost always evidence of metastatic tumor elsewhere, and only rarely is there significant splenomegaly. Granulomatous disease such as tuberculosis can also lead to significant marrow fibrosis, but usually only with modest splenomegaly. Hairy cell leukemia can cause massive splenomegaly and extensive marrow fibrosis, but the characteristic circulating "hairy" lymphocytes usually make that differential diagnosis straightforward.

The treatment of myelofibrosis is symptomatic and unsatisfactory. When anemia is severe, transfusion of red cells is important. When other problems develop, such as hypersplenism (ie, the sequestration of circulating blood cells in the spleen) or pain due to the bulk of the spleen or to splenic infarction, splenectomy should be considered. The mean survival time after splenectomy was approximately two years in the Mayo Clinic experience, suggesting that massive splenomegaly and hypersplenism represent late events in the natural history of this disease.[5] Chemotherapy with oral alkylating agents or with hydroxyurea is generally reserved for patients who are poor surgical candidates; this treatment generally has shown only transient effectiveness.

It was never clear why this woman had such a severe predilection for venous thrombosis and pulmonary embolism. Her antithrombin III level was normal at a time when she had nephrotic-level proteinuria. She had no history of thrombotic episodes, and there was no evidence of recurrent colon carcinoma that might have been responsible for a hypercoagulable state.

Michael Schwartzman, MD (Attending Internist, Nephrologist): I wish to make only a few comments about the renal disease and its unusual manifestations.

Renal vein thrombosis has two clinical forms which are well recognized. The first is more commonly seen in pediatric patients and presents as an acute illness with flank pain and bloody urine, usually in a setting of dehydration, and sometimes associated with acute renal failure.

In adults, renal vein thrombosis can be due to some major circulatory disturbance in the kidney, particularly on the arterial side, with secondary low flow through the venous system. Usually, however, renal vein thrombosis in the adult is a complication of the nephrotic syndrome, most commonly occurring in the setting of membranous nephropathy.

Membranous nephropathy is the most common histologic pattern of injury in the idiopathic or primary nephrotic syndrome in adults. We thought that this patient had membranous nephropathy not only because of the clinical complications that were described, but also because of her history of colon cancer. Some patients with membranous nephropathy have no apparent illness other than the kidney disease itself; that is called the idiopathic nephrotic syndrome. However, it is known that in many patients, the nephrotic syndrome develops secondary to systemic illness such as diabetes, or to adverse drug reactions, or to an underlying infection. In some cases, the immunologic disturbance underlying the nephropathy is secondary to a solid tumor, such as colon cancer.

This patient had an elevated level of serum carcinoembryonic antigen, so although there was no clinical evidence of recurrent tumor, its presence was at least suspected. The treatment of membranous nephropathy secondary to malignancy depends on effective treatment of the underlying disease. Consequently, this patient was not subjected to a renal biopsy or to prednisone therapy, such as might be used in patients with the primary form of membranous nephropathy.

Pathogenetic factors in the nephrotic syndrome related to renal vein thrombosis include low serum antithrombin III levels related to urinary wasting, and high plasma fibrinogen levels associated with elevated acute-phase reactants. In addition, in this patient thrombocytosis developed after the splenectomy, which may have played a role in the hypercoagulable state. Renal venous blood has the lowest levels of antithrombin III, perhaps explaining the peculiar predilection for thrombosis in this site. Multiple sources of pulmonary emboli, including lower extremity deep venous thrombosis, may have been present in this case. An excess incidence of thromboembolic complications in patients with the nephrotic syndrome has frequently been commented on.[6] A prospective study of the incidence of renal vein thrombosis in association with idiopathic membranous nephropathy has identified this complication in about one third of patients.[7] In most individuals, renal vein thrombosis remains asymptomatic but would be detectable by careful renal venography. On occasion, its clinical presentations can be life-threatening; however, it is not common to treat patients with the nephrotic sydrome with anticoagulants in the absence of clinical evidence of thromboembolism. There are no clinical data to define how long anticoagulant therapy should be maintained in a patient with this condition. As long as the nephrotic syndrome persists, the possibility of recurrent thromboembolic events exists.

One useful observation in patients with renal vein thrombosis and the nephrotic syndrome is an increase in the level of proteinuria during active renal vein obstruction. In this patient, the degree of proteinuria increased from her baseline level of 2 g/24 hours to 6 g/24 hours when her pulmonary embolism occurred and we suspected renal vein thrombosis. Once anticoagulant therapy was initiated, the level of proteinuria declined to her prior baseline. We are inclined to suspect renal vein thrombosis when there is a sudden increase in the level of proteinuria or a drop in the serum albumin concentration. There may not be any concurrent change in renal function, although in this case we did see minor increases in serum creatinine levels following the pulmonary embolism.

Clinical Diagnoses

Myelofibrosis with myeloid metaplasia.

Severe osteosclerosis, secondary to myelofibrosis.

Past history of polyposis and carcinoma of colon; total colectomy.

History of splenic infarctions; splenectomy.

Thrombosis, left femoral and right renal veins, associated with recurrent pulmonary embolism and infarction.

Nephrotic syndrome with progressive renal failure, associated with renal vein thrombosis and membranous glomerulonephropathy.

Hypertensive cardiovascular disease and congestive heart failure.

Pathology Presentation

Harlan D. Alpern, MD (Pathologist-in-Chief): The cause of death was a large embolus lodged in the right pulmonary artery. There was an associated large, recent infarct involving the right upper and middle lobes. The embolus was probably several days old.

A thrombus was found in the left femoral vein, the most probable source of the pulmonary embolus. The Greenfield filter was in place in the vena cava inferior to the renal veins. The device consists of metallic prongs between which are large open spaces. In the filter was abundant thrombus. The most likely explanation is that a portion of the femoral vein thrombus broke off and made its way through the prongs of the Greenfield filter and lodged in the right lung. An alternative possibility would be that a portion of the thrombus attached to the filter could have broken off and embolized to the lung. The thrombus in the filter could represent a thrombus that formed at that site, or that developed secondary to an embolus from the thrombus in the left femoral vein, or an earlier thrombus.

The bones were rock-hard due to osteosclerosis complicating the myelofibrosis. The process of osteosclerosis represents increase in calcified bone and is seen in about half of patients who have myelofibrosis with myeloid metaplasia. The bone marrow itself was markedly cellular to an extent of about 90% (Fig 6). Megakaryocytes were increased in number, and the myeloid-to-erythroid ratio was markedly increased. While there was some myeloid maturation, the predominant myeloid cells were of immature types with increased numbers of myelocytes and promyelocytes and some blasts. Erythroid forms were decreased in numbers with maturation. Reticulin stains demonstrated marked marrow reticulin, as is typical of the late stage of myelofibrosis.

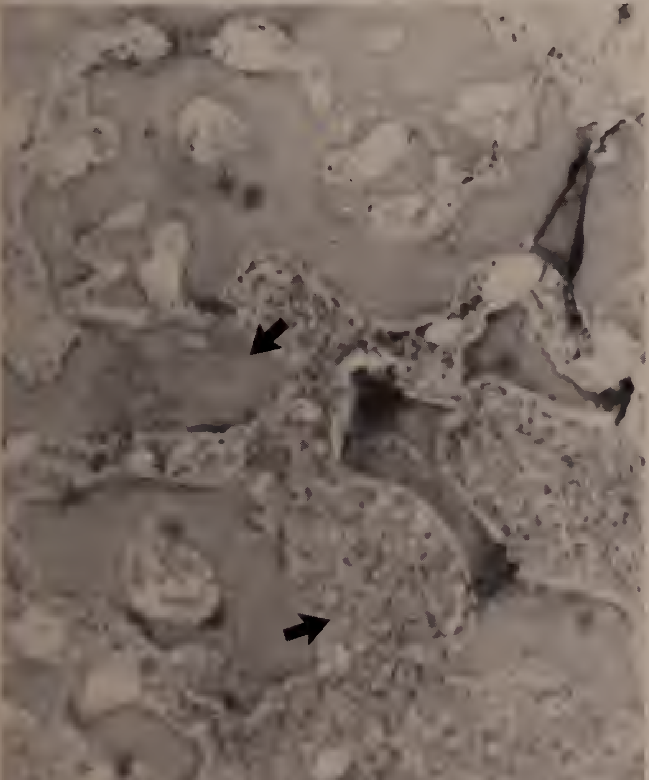


FIGURE 6. Myelofibrosis with myeloid metaplasia (lower arrow) and increased density of bone (upper arrow) (hematoxylin-eosin stain; original magnification × 40).

As Dr Horner said, myelofibrosis with myeloid metaplasia, also referred to as agnogenic myeloid metaplasia, is believed to be a hematopoietic stem-cell defect, a neoplastic condition of the stem cell in which all three hematopoietic cell lines are affected to various degrees. In this case, the predominantly affected cell lines were myeloid and megakaryocytic.

The liver weighed 2,500 g, nearly twice the normal size. Hepatomegaly is often found in this disease and is caused by the presence of extramedullary hematopoiesis. Megakaryocytes, numerous immature myeloid forms, and some erythroid forms were present in the liver. This extramedullary hematopoiesis is not felt to be a compensatory mechanism for inadequate bone marrow production of cellular blood elements, but rather to be part of the neoplastic proliferation.

In this patient, the lymph nodes were also involved in the extramedullary hematopoiesis; many megakaryocytes and myeloid forms were seen therein. Leder esterase stain showed the myeloid granules of the immature myeloid forms in these lymph nodes. Extramedullary hematopoiesis was also present in the right adrenal gland, as is occasionally the case in this disorder.

The ileoproctostomy anastamosis was examined, as was the remaining rectum; no residual carcinoma was found, nor was there evidence of metastatic carcinoma.

The brain showed slight dilation of the ventricles. There was no evidence of hemorrhage in the ventricles, meninges, or elsewhere in the central nervous system. There was a minute infarct in the right frontal cortex. There were no arachnoid adhesions. Within a cerebral vessel, but not within the brain parenchyma, there was stasis of immature myeloid cells. No anatomic correlation was found for the clinical neurologic changes described.

In the right renal vein, there was a fibrous, endothelial intimal band, which is the only residual evidence of an old, fully organized thrombus. There was no evidence of a more recent thrombus in either renal vein.

The kidneys were finely granular and small, weighing 80 g each. The cortices were slightly thinned. Light microscopy revealed that about 50–60% of the glomeruli were globally sclerotic. About 10% were segmentally sclerotic. The remaining glomeruli demonstrated a moderate and uniform diffuse thickening of the glomerular capillary walls (Fig 7). Mesangial matrix appeared increased, and there was some increase in mesangial cells. Light microscopic changes were felt to be consistent with membranous glomerulonephropathy. Confirmation requires electron microscopy, which would be expected to demonstrate electron-dense antibody-antigen deposits between the basement membrane and the epithelial cells (epimembranous). Formalin-fixed tissue was sent to Dr Vivette D'Agati, Renal Pathologist at Columbia University College of Physicians and Surgeons, our regular consultant in renal pathology. She found the routine histologic analysis also consistent with membranous glomerular nephropa-

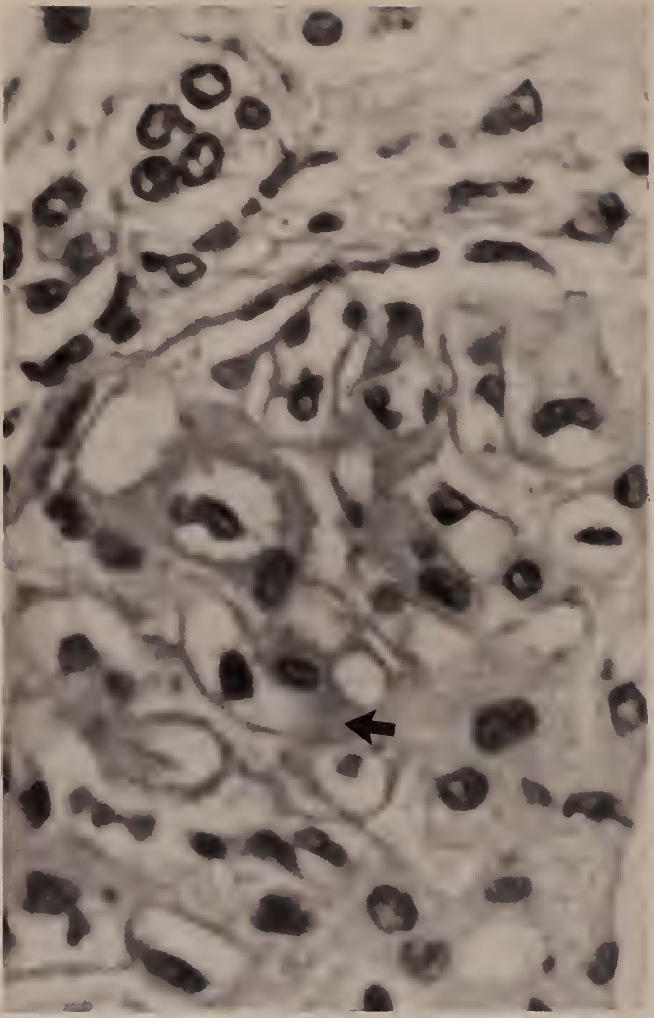


FIGURE 7. Moderate to marked diffuse thickening of glomerular basement membranes, with increased mesangial matrix (arrow) (hematoxylin-eosin stain; original magnification × 400).

thy. Because of mesangial proliferative features, she could not exclude other forms of glomerulonephritis, so that electron micrographic studies were done.

To our surprise, the electron microscopic examination revealed no evidence of epimembranous electron-dense deposits. The mesangial areas were expanded, with increased numbers of cells and increased matrix. The matrix contained numerous, randomly distributed fibrils measuring approximately 20 nm in diameter without periodicity (Fig 8). There was moderate to marked thickening of the glomerular basement membranes with similar fibrils present. The fibrils were felt to be thicker than amyloid, and Congo Red stains for amyloid, which were performed twice, were negative. Dr D'Agati's interpretation was that the findings were those of a fibrillary glomerulopathy referred to as "Congo Red-negative amyloidosis-like glomerulonephropathy" by Sturgil et al.[8] Dr D'Agati pointed out that similar glomerular basement thickening with mesangial sclerosis associated with fibrillosis could be seen in diabetic glomerulosclerosis and also in patients with cryoglobulinemia; however, the patient was not diabetic and there was no evidence of cryoglobulins.

This newly recognized entity has been described by several authors under different titles. Prior to the report by Sturgil and colleagues in 1985, Olesnicky et al[9] at Columbia reported on the finding of two otherwise typical cases of membranous glomerulonephropathy in which there were large clusters of tubular microfibrils measuring 8–12 nm in diameter and small accumulations of larger microfibrils measuring 18–20 nm in the glomerular basement membrane and in the mesangium. The authors noted that microfibrils in glomerular disease have been reported in several systemic disorders including diabetes mellitus, hemolytic-uremic syndrome, and Marfan syndrome.

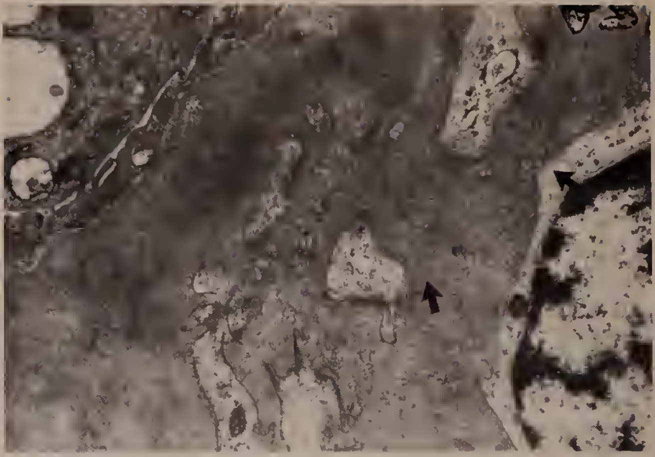


FIGURE 8. Fibrillary depositions in renal mesangium (arrows) (electron micrograph; original magnification × 10,000).

In 1983 Duffy et al[10] reported on eight cases of fibrillary renal deposits with nephritis. In these eight patients, glomeruli contained abundant fibrils in the mesangial matrix and glomerular basement membranes. The fibrils averaged 20 nm in width, about twice the size of amyloid fibrils, and did not react as amyloid does with special stains. Immunofluorescent microscopic studies for immunoglobulins were generally positive with antisera to IgG, often IgM, and in some cases IgA. Korbet et al[11] recently reported 11 other such cases under the title of immunotactoid glomerulopathy. The microtubules were heterogeneous in size and immunoglobulin composition.

During the patient's life, it was the clinical impression that the glomerulonephritis was most probably of membranous type. Renal vein thrombosis, which the patient had during life, is believed to be the result of, rather than the cause of, the nephropathy. Carcinomas are commonly seen in adults with membranous nephropathy; there was no residual carcinoma in this case.

Of special interest is the fact that in the interstitium of the kidney there was also evidence of the cellular infiltrate of myelofibrosis with myeloid metaplasia (Fig 9). Leder esterase stain demonstrated large numbers of myeloid forms.

In summary, our findings are those of advanced myelofibrosis with myeloid metaplasia, with a rare, recently recognized fibrillary glomerulopathy referred to as "Congo Red-negative amyloidosis-like glomerulopathy," with death attributed to a large pulmonary embolus in the right lung associated with pulmonary infarct.

Dr Horner: I wonder, in retrospect, if the finding of dilated cerebral ventricles on the patient's serial CT scans might have been present for a much longer time. I wonder, too, whether the neurologic changes that we saw clinically

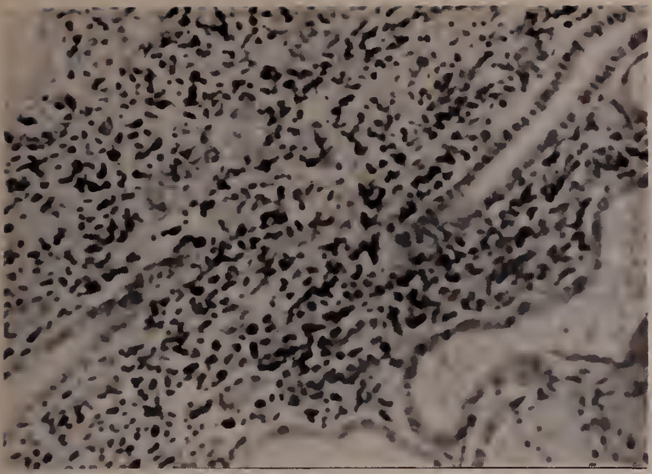


FIGURE 9. Renal interstitial infiltrate of cellular elements of myelofibrosis (hematoxylin-eosin stain; original magnification × 100).

on that one day represented an episode of hypertensive encephalopathy, and not an acute dilation of the ventricles. Besides the ventricular shunt, her blood pressure elevation was aggressively treated with medication.

Anne Nafziger, MD (Medical Resident): The patient was extremely unresponsive to medical treatment of her hypertension at that point, which made us think that her hypertension was due to acute hydrocephalus.

Dr Schwartzman: The response to the shunting was an acute drop in her blood pressure.

Dr Horner: Temporarily, it certainly was.

FINAL DIAGNOSES

Myelofibrosis with myeloid metaplasia (agnogenic myeloid metaplasia) with extramedullary hematopoiesis in the liver, kidneys, right adrenal gland, and lymph nodes.

Venous thrombosis, involving the left femoral vein and vena caval filter, with secondary pulmonary embolus and large recent infarct in the right lung.

Congo Red-negative amyloidosis-like glomerulopathy (immunotactoid glomerulopathy).

Endothelial fibrosis in the right renal vein, residual from healed right renal vein thrombus.

History of colon carcinoma, resected, without metastasis or recurrence.

Minute infarction in the right frontal cortex.

Slight dilation of the cerebral ventricles.

REFERENCES

1. Lichtman MD: Classification of the hematopoietic stem cell disorders, in Williams WJ, et al (eds): *Hematology*, ed 3. New York, McGraw Hill, 1983, pp 144–150.
2. Rosenthal DS, Moloney WC: Occurrences of acute leukemia in myeloproliferative disorders. *Br J Haematol* 1977; 36:373–382.
3. Jacobson RJ, Salo A, Fialkow PJ: Agnogenic myeloid metaplasia—a clonal proliferation of hematopoietic stem cells with secondary myelofibrosis. *Blood* 1978; 51:189–194.
4. Groopman JE: The pathogenesis of myelofibrosis in myeloproliferative disorders [editorial]. *Ann Intern Med* 1980; 92:857–858.
5. Silverstein MN, ReMine WH: Splenectomy in myeloid metaplasia. *Blood* 1979; 53:515–518.
6. Llach F, Arieff AI, Massry SG: Renal vein thrombosis and nephrotic syndrome. A prospective study of 36 adult patients. *Ann Intern Med* 1975; 83:8–14.
7. Wagoner RD, Stanson AW, Holley KE, et al: Renal vein thrombosis in idiopathic membranous glomerulopathy and nephrotic syndrome: Incidence and significance. *Kidney Int* 1983; 23:368–374.
8. Sturgill BC, Bolton WK, Griffith KM: Congo red-negative amyloidosis-like glomerulopathy. *Human Pathol* 1985; 16:220–224.
9. Olesnicky L, Doty SB, Bertani T, et al: Tubular microfibrils in the glomeruli of membranous nephropathy. *Arch Pathol Lab Med* 1984; 108:902–905.
10. Duffy JL, Khurana E, Susin ME, et al: Fibrillary renal deposits and nephritis. *Am J Pathol* 1983; 113:279–290.
11. Korbet SM, Schwartz MM, Rosenberg BF, et al: Immunotactoid glomerulopathy. *Medicine* 1985; 64:228–243.

Readership survey of the *New York State Journal of Medicine*

PASCAL JAMES IMPERATO, MD

The *New York State Journal of Medicine* is now in its 87th year of consecutive publication. It is a peer reviewed scientific medical journal published monthly by the Medical Society of the State of New York. The *Journal* publishes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, and socioeconomic issues in health care. It has a total circulation of 28,297, of which 25,707 copies are distributed in New York State and 2,068 in the rest of the country. Foreign subscriptions total 248 (Table I). Of the total New York State circulation of 25,707, 17,578 copies (68.3%) are sent to subscribers in New York City and the adjacent counties of Nassau, Suffolk, and Westchester.

Readers of the *Journal* come from a broad range of specialties, contrary to the perceptions of some that they are mostly primary care practitioners (Table II). A total of 20,394 (72.1%) subscriptions are from dues-paying members of the society and 4,973 (17.5%) from life members, of whom 3,973 (79.9%) live in New York State. There are 1,320 medical student subscribers, 164 copies are exchanged with other medical journals, and 149 complimentary copies are distributed.

In 1986 the *Journal* was ranked 29th in terms of its impact on the medical and research communities in a study which used the *Science Citation Index* and the *Journal Citation Reports* to determine the frequency with which items were cited from various journals in the medical literature.[1] Approximately 5,000 medical journals are published in the world, of which some 2,400 are indexed in the *Index Medicus*. Of the latter, 112 are indexed by *Index Medicus* in its monthly rapid abridged listing. The *New York State Journal of Medicine* is among the select 112. Its ranking of 29 among a pool of 2,400 journals places it among the top leading medical journals in the world in terms of its impact on the medical community.

In terms of circulation, the *Journal* occupies a middle position with a number of other leading journals. At the very high end of the spectrum are the large circulation journals such as *JAMA* and the *New England Journal of Medicine*, with circulations in excess of 200,000 (Table III). These are followed by journals with circulations between 100,000 and 200,000, and then by those with circulations between 50,000 and 100,000. Those journals with circulations between 20,000 and 50,000 occupy a middle range. As shown in Table III, a number of important medical journals have circulations less than 15,000. There is no relationship between a journal's circulation and its cumulative impact on the medical and research communities as demonstrated by a comparison of current circulation levels with the results of Garfield's study.[1] *The Lancet*,

TABLE I. Geographic Distribution of the Readership of the *New York State Journal of Medicine*

Geographic Location	Number of Subscribers
New York State	25,707
US outside of New York State	2,068
Foreign	248
Total	28,023

TABLE II. Specialties of Selected Groups of *Journal* Readers

Specialty	Number of Subscribers
Allergy	365
Anesthesiology	1,169
Dermatology	493
Emergency Medicine	150
Gastroenterology	399
General Practice	1,625
Internal Medicine	3,947
Legal Medicine	9
Neurology	343
Obstetrics and Gynecology	1,777
Occupational Medicine	191
Ophthalmology	1,179
Otorhinolaryngology	509
Pathology	357
Pediatrics	1,571
Psychiatry	1,582
Pulmonology	266
Radiology	584
General Surgery	1,847
Orthopedic Surgery	956
Plastic Surgery	321
Thoracic Surgery	155
Urology	620

Address correspondence to Dr Imperato, Editor, *New York State Journal of Medicine*, 420 Lakeville Rd, Lake Success, NY 11042.

TABLE III. Comparative Circulations of Selected Medical Journals, 1986*

Journal	Circulation
Journal of the American Medical Association	279,600
New England Journal of Medicine	215,000
Hospital Practice	183,000
Modern Medicine	168,000
Postgraduate Medicine	145,000
Mayo Clinic Proceedings	124,000
Annals of Internal Medicine	91,000
Archives of Internal Medicine	69,200
Western Journal of Medicine	50,000
American Journal of Medicine	44,166
Lancet	42,000
Pediatrics	41,500
Canadian Medical Association Journal	42,000
American Journal of Public Health	35,000
American Journal of Obstetrics & Gynecology	29,435
American Journal of Cardiology	28,905
New York State Journal of Medicine	28,297
Southern Medical Journal	24,120
Circulation	23,000
American Journal of Surgery	20,700
American Journal of Clinical Pathology	15,206
Pennsylvania Medicine	15,100
American Heart Journal	10,589
New Jersey Medicine	10,018
Surgery	9,992
Indiana Medicine	6,600
Mount Sinai Journal of Medicine	6,000
Bulletin of the New York Academy of Medicine	5,000
American Journal of Epidemiology	4,791
Acta Medica Scandinavica	2,900
American Journal of Tropical Medicine and Hygiene	2,800
Bulletin of the History of Medicine	2,100

* From *Ulrich's International Periodicals Directory*, 1986–1987, ed 25, vol 1, New York, R. R. Bowker, 1986.

with a current circulation of 42,000, was ranked as having a greater impact in terms of frequency of citation than either the *New England Journal of Medicine* or *JAMA*, whose circulations are five- and seven-fold greater, respectively.[1] The quality and importance of content as well as the number of years of publication of a journal seem to play important roles in the frequency of citation. This is exemplified by the fact that the *Bulletin of the New York Academy of Medicine*, with a circulation of 5,000, and the *East African Medical Journal*, with a circulation of 1,200, placed 40th and 50th respectively on Garfield's list.[1]

In 1986, the *Journal* published a total of 195 articles authored by 397 authors. The breakdown of these articles by category is given in Table IV. Of the 195 papers published, 35 (17.9%) dealt with socioeconomic, medicolegal, and health administration topics. Some 150 peer reviewers from throughout the state and country reviewed the 375 papers submitted to the *Journal*.[2] Most submissions to the *Journal* are reviewed by two or more outside peer reviewers. In general, accepted papers undergo one or more revisions and re-review by peer reviewers prior to publication. All accepted papers are read and edited by the editor and a consulting editor before being copy edited. The acceptance rate for submissions to the *Journal* in 1986 was 43.7%. In 1983 the *Journal* underwent major changes in both format and the rigor of peer review.

TABLE IV. Categories of Articles Published in the *Journal*, 1986

Category	Number
Case Reports	47
Letters to the Editor	45
Commentaries	32
Research Papers	17
Review Articles	10
Essays in Pediatrics	5
History	6
Special Articles	5
Clinical Conferences	3
Medical Malpractice	13
Miscellaneous	12
Total	195

The needs and views of a medical journal's readership are vitally important to editors in formulating decisions about content, format, and design. In order to solicit the views of our readers, a survey was undertaken from among a sample of 1,550 readers in June and July 1987.

METHODS

The sample of 1,550 readers was proportionately and randomly chosen from among the five board specialty categories of physicians who receive the *Journal* (Table V). The universe of 27,440 included only individual subscribers and excluded institutional ones. Only *Journal* recipients in New York State were included in the sample. A 14-item survey consisting of 13 closed-ended questions and one open-ended one was designed by the *Journal* staff in conjunction with the society's Committee on Publications, Library and Archives and with Health Research Industries of Wilton, Connecticut, which conducted the actual survey. The questionnaire comprised four pages and contained a letter of explanation. A black and white reproduction of the *Journal's* cover was printed on the first page of the questionnaire to aid recall, and a one dollar incentive was included. The completed questionnaires were returned anonymously to Health Research Industries, which conducted the analysis of the results. The questions elicited information about reading frequency, depth of reading, reading time per issue, level of reading for various categories of content, and opinions about the quality of content and format and the positioning of ad-

TABLE V. Survey Groups and Sample Sizes, 1987 Readership Survey

Group	Size	Sample Size	Nth No
General Practice and Family Practice	2,777	241	11
Internal Medicine and its subspecialties	6,207	402	15
Gastroenterology			
Cardiovascular Disease			
Pulmonary Disease			
General Surgery and its subspecialties	3,838	202	18
Pediatrics	1,571	207	7
Other	13,047	498	21
Total	27,440	1,550	

TABLE VI. Reading Frequency and Reading Patterns by Specialty and Age Group

		Specialty					Age Group		
	Total	Family Practice/ General Practice	Internal Medicine	General Surgery	Pediatrics	Other	Under 45	45–65	Over 65
Total responding	707	102	164	107	102	232	203	332	167
Reading frequency									
4 of 4	49%	51%	43%	55%	44%	52%	38%	46%	69%
3 of 4	16%	19%	15%	13%	20%	14%	14%	18%	14%
2 of 4	13%	13%	16%	8%	14%	14%	18%	14%	7%
1 of 4	13%	14%	16%	13%	14%	10%	20%	12%	7%
0 of 4	9%	4%	10%	10%	9%	10%	11%	10%	3%
Reading patterns									
Cover to cover	1%		1%		2%	2%	1%	1%	1%
Read/look	44%	46%	37%	39%	44%	50%	38%	43%	51%
Skim pages	11%	11%	9%	13%	10%	11%	12%	13%	5%
Table of contents and articles of interest	44%	43%	53%	48%	44%	37%	49%	43%	43%

vertising. Respondents were also asked questions about their reading of other medical journals and their saving the *Journal* for future reference. An open-ended item at the end of the questionnaire asked respondents to make suggestions for improvements and to offer any other opinions they had about content and format.

RESULTS

Of the total of 1,550 questionnaires mailed, completed responses were received from 707 (45.7%). The highest level of response was from general surgeons, with 107 of 202 responding (53.0%), and the lowest from internists, with 164 of 402 responding (40.9%). Table VI presents the reading frequency by specialty and age group. Close to half of readers (49%) read four out of four issues and only 9% read none out of four. Fifty-five percent of general surgeons read four out of four issues and 69% of respondents over 65 years of age do so. The majority of individuals in all specialty and age groups read three or more out of every four journal issues (Table VI).

Respondents were asked if they wanted to continue receiving the *Journal*. Overall 584 (85%) responded affirmatively. Ninety-three percent (91/98) of family practitioners/general practitioners responded yes and 145 of 161 internists (90%) said yes. By contrast, 80% (83/104) of general surgeons and 80% (182/228) of respondents in the "other" category replied yes. Among those over 65 years of age, 92% wanted to continue receiving the journal, while among those under age 45, 80% did.

Thirty-three percent of readers spend more than half an hour reading the *Journal* (Table VII). Sixty-seven percent spend less than half an hour. The average reading time level is 32 minutes, with physicians over 65 spending 44 minutes with the average issue. Those under age 45 spend 25 minutes with an average issue (Table VII).

Most readers do not save the *Journal* for future reference (63%). However, 47% of family practitioners/general practitioners do save the *Journal*. Among other groups the rates of saving the *Journal* are as follows: internal medicine (38%), general surgery (32%), pediatrics (38%), and other (34%).

Readers were queried about their receipt of other medi-

TABLE VII. Reading Time for Average Issue by Specialty and Age Group

		Specialty					Age Group		
	Total	Family Practice/ General Practice	Internal Medicine	General Surgery	Pediatrics	Other	Under 45	45–65	Over 65
Total responding	609	92	140	91	87	199	175	284	149
Reading time	100%	100%	100%	100%	100%	100%	100%	100%	100%
1–10 minutes	103	16	24	14	19	30	37	58	8
	17%	17%	17%	15%	22%	15%	21%	20%	5%
11–20 minutes	140	16	34	21	16	53	59	59	22
	23%	17%	24%	23%	18%	27%	34%	21%	15%
21–30 minutes	165	26	40	27	20	52	41	79	44
	27%	28%	29%	30%	23%	26%	23%	28%	30%
31–45 minutes	95	15	25	15	11	29	21	46	28
	16%	16%	18%	16%	13%	15%	12%	16%	19%
46–60 minutes	68	9	10	10	13	26	16	29	23
	11%	10%	7%	11%	15%	13%	9%	10%	15%
Over 60 minutes	38	10	7	4	8	9	1	13	24
	6%	11%	5%	4%	9%	5%	1%	5%	16%
No answer	98	10	24	16	15	33	28	48	18
Average reading time per issue in minutes	32	35	30	32	34	31	25	30	44

cal journals per month. Most (78%) indicated they receive fewer than ten medical journals per month. Twenty-two percent receive eleven or more per month. Among internists, 28% receive 11–20 journals per month, the highest of any specialty group surveyed, followed by family practitioners/general practitioners, of which 24% receive 11–20 journals per month.

Over 90% of respondents spend an hour or more with other journals during an average week. Forty-seven percent of those in the total sample said they spend more than two hours per week reading other journals. Of interest is the fact that those over 65 years of age on average do not spend more time reading other medical journals than most of their younger colleagues, whereas they spend an average of 44 minutes reading the *New York State Journal of Medicine*, much more than younger physicians (Table VII).

Readers were asked a series of questions aimed at eliciting reading levels for the standard categories of features carried in the *Journal's* editorial pages. The results of some of these questions are shown on Tables VIII through XII. Overall they indicate that readers read selectively. Frequency of reading is influenced to some degree by specialty. Overall, book reviews are not read by 53% of the sample, the do-not-read level extending from 44% for the "other" category to 63% for general surgeons.

Specific questions were posed about various recent thematic issues of the *Journal*. Eighty-eight percent of the sample either selectively read or read all/most of the July 1986 issue on medical malpractice. Twelve percent did not read the issue, the highest rates of do-not-read being among internists (17%) and pediatricians (14%). Concerning the three published thematic issues on cigarette smoking (December 1983, July 1985, and September 1986), 27% did not read them. Seventy-three percent read all/most or else read selectively. Readership levels for the issue focusing on geriatrics (December 1986) were much lower, 41% replying that they did not read it. Not surprisingly, 74% of pediatricians stated they did not read this thematic issue, while only 16% of family practitioners/general practitioners responded that they did not. Seventy-six percent of pediatricians compared to 47% of inter-

TABLE VIII. Usual Reading Level of Commentaries in the *Journal* by Specialty

	Total	Family Practice/ General Practice	Internal Medicine	General Surgery	Pediatrics	Other
Total responding	575	90	134	87	85	179
Reading level	100%	100%	100%	100%	100%	100%
Read all/most	96	17	21	13	12	33
	17%	19%	16%	15%	14%	18%
Read selectively	393	60	97	57	53	126
	68%	67%	72%	66%	62	70
Do not read	86	13	16	17	20	20
	15%	14%	12%	20%	24%	11%
No answer	132	12	30	20	17	53

TABLE IX. Usual Reading Level of Research Papers by Specialty

	Total	Family Practice/ General Practice	Internal Medicine	General Surgery	Pediatrics	Other
Total responding	567	87	132	84	83	181
Reading level	100%	100%	100%	100%	100%	100%
Read all/most	41	6	13	6	5	11
	7%	7%	10%	7%	6%	6%
Read selectively	417	67	94	58	56	142
	74%	77%	71%	69%	67%	78%
Do not read	109	14	25	20	22	28
	19%	16%	19%	24%	27%	15%
No answer	140	15	32	23	19	51

TABLE X. Usual Reading Level of Review Articles by Specialty

	Total	Family Practice/ General Practice	Internal Medicine	General Surgery	Pediatrics	Other
Total responding	589	88	138	89	89	185
Reading level	100%	100%	100%	100%	100%	100%
Read all/most	98	14	25	9	15	35
	17%	16%	18%	10%	17%	19%
Read selectively	454	67	108	72	65	142
	77%	76%	78%	81%	73%	77%
Do not read	37	7	5	8	9	8
	6%	8%	4%	9%	10%	4%
No answer	118	14	26	18	13	47

TABLE XI. Usual Reading Level of Clinical Conferences by Specialty

	Total	Family Practice/ General Practice	Internal Medicine	General Surgery	Pediatrics	Other
Total responding	541	84	125	84	80	168
Reading level	100%	100%	100%	100%	100%	100%
Read all/most	63	13	14	7	12	17
	12%	15%	11%	8%	15%	10%
Read selectively	347	56	82	50	51	108
	64%	67%	66%	60%	64%	64%
Do not read	131	15	29	27	17	43
	24%	18%	23%	32%	21%	26%
No answer	166	18	39	23	22	64

TABLE XII. Usual Reading Level of Case Reports by Specialty

	Total	Family Practice/ General Practice	Internal Medicine	General Surgery	Pediatrics	Other
Total responding	563	84	135	85	81	178
Reading level	100%	100%	100%	100%	100%	100%
Read all/most	80	17	18	9	11	25
	14%	20%	13%	11%	14%	14%
Read selectively	374	57	89	53	56	119
	66%	68%	66%	62%	69%	67%
Do not read	109	10	28	23	14	34
	19%	12%	21%	27%	17%	19%
No answer	144	18	29	22	21	54

nists read all/most or selectively read the issue with the sports medicine theme (February 1987). Eighty-eight percent of the total sample selectively read or else read all/most of the May 1987 issue on acquired immunodeficiency syndrome. These levels were high even among surgeons, of whom 88% responded that they read all/most or selectively.

Respondents gave very high ratings to both the current quality and current format of the *Journal* (Tables XIII–XVII). Seventy-eight percent rated the current quality of the content as good or excellent (Table XIII) and 84% rated the current format this way (Table XV). High scores were given to the *Journal* for quality and format improvements over the past few years. Thirty-five percent stated that the improvements in editorial quality were significant and 47% said they were average. Only 15% had not noticed any and 3% rated them low (Table XIV). With regard to format, 14% found it excellent, 70% good, 16% average, and only 1% poor (Table XV). Thirty-two percent thought that the format improvements over the past few years were significant, 50% average, and only 5% low (Table XVI).

The history of medicine section continues to be a popular feature, with 72% replying that they read all/most (17%) or read selectively (55%). Special articles are also widely read, 75% reading selectively and 18% reading all/most. The essays in pediatrics are mostly read by pediatricians (39% read selectively; 53% read all/most). Yet 37% of general surgeons, 19% of internists, and 55% of family practitioners/general practitioners read them selectively. Letters to the editor are selectively read or read at the all/most level by 70% of readers.

The overwhelming majority of readers prefer stacked advertising (ads in the front and back of the *Journal*) as opposed to interspersed advertising (Table XVII). Only 8% of the total sample indicated a preference for interspersed advertising.

Verbatim comments were solicited in a final open-ended question. Some of these follow:

"... everything covered. Like thematic issues. The journal used to bore me, now I like it. Maintain history articles, especially photographically enhanced ones." (FP/GP)

An internist wrote the following: "In general the journal is very much more attractive than five years ago. I like thematic issues which I save for future reference ... More review articles, more special articles."

A general surgeon made the following observations: "I enjoy most of what I read. The book is great ... Front cover—change contents listing—difficult to quickly find. More medical history articles ... Reviews of controversial subjects with pros and cons from authorities in the field. Journal is fine but due to volume of specialty journals time is limited to read it. Need more case presentations."

A pediatrician said: "More pediatric articles and news items. I enjoy reading over-view articles—discuss recent advances in the treatment of common medical problems. History of medicine—pediatrics and the story of past physicians. More pediatric literature."

A reader from the "all other specialties" category said: "The Parkinson review article was particularly good. The reviews are good for me and my house staff. I will continue to read this journal. Material very satisfactory. The magazine is good as it is."

A final comment from a physician in the "all other spe-

TABLE XIII. Rating of the Current Quality of the *Journal* Contents by Specialty

	Total	Family Practice/ General Practice	Internal Medicine	General Surgery	Pediatrics	Other
Total responding	632	99	143	96	90	204
	100%	100%	100%	100%	100%	100%
Excellent	77	13	14	13	10	27
	12%	13%	10%	14%	11%	13%
Good	420	67	88	67	65	133
	66%	68%	62%	70%	72%	65%
Average	126	19	39	15	15	38
	20%	19%	27%	16%	17%	19%
Poor	9		2	1		6
	1%		1%	1%		3%
No answer	75	3	21	11	12	28

TABLE XIV. Rating of Editorial Quality Improvements Made in the *Journal* Over the Past Few Years by Specialty

	Total	Family Practice/ General Practice	Internal Medicine	General Surgery	Pediatrics	Other
Total responding	610	95	140	94	85	196
	100%	100%	100%	100%	100%	100%
Significant	214	28	43	38	35	70
	35%	29%	31%	40%	41%	36%
Average	285	51	65	45	36	88
	47%	54%	46%	48%	42%	45%
Low	17	4	6	2	1	4
	3%	4%	4%	2%	1%	2%
Have not noticed	94	12	26	9	13	34
	15%	13%	19%	10%	15%	17%
No answer	97	7	24	13	17	36

TABLE XV. Rating of the Current Format of the *Journal* by Specialty

	Total	Family Practice/ General Practice	Internal Medicine	General Surgery	Pediatrics	Other
Total responding	628	97	143	96	89	203
	100%	100%	100%	100%	100%	100%
Excellent	85	13	16	15	10	31
	14%	13%	11%	16%	11%	15%
Good	439	68	95	67	69	140
	70%	70%	66%	70%	78%	69%
Average	99	16	32	13	10	28
	16%	16%	22%	14%	11%	14%
Poor	5			1		4
	1%			1%		2%
No answer	79	5	21	11	13	29

TABLE XVI. Rating of Format Improvements Over the Past Few Years by Specialty

	Total	Family Practice/ General Practice	Internal Medicine	General Surgery	Pediatrics	Other
Total responding	597	90	138	90	87	192
	100%	100%	100%	100%	100%	100%
Significant	189	24	36	34	31	64
	32%	27%	26%	38%	36%	33%
Average	296	52	72	45	43	84
	50%	58%	52%	50%	49%	44%
Low	27	4	6	5	1	11
	5%	4%	4%	6%	1%	6%
Have not noticed	85	10	24	6	12	33
	14%	11%	17%	7%	14%	17%
No answer	110	12	26	17	15	40

TABLE XVII. Advertising Format Preference by Specialty

	Total	Family Practice/ General Practice	Internal Medicine	General Surgery	Pediatrics	Other
Total responding	606	90	142	92	87	195
	100%	100%	100%	100%	100%	100%
Prefer interspersed	53	11	6	8	10	18
advertising	8%	12%	4%	9%	11%	9%
Prefer stacked	556	79	137	85	77	178
advertising	92%	88%	96%	92%	89%	91%
No answer	101	12	22	15	15	37

cialties" group demonstrates that some of the *Journal's* readers are of an independent mind: "Sorry, spend much time reading other journals. Your editorial board places too much emphasis on the psychological aspects of clinical results—Too far away from reality."

DISCUSSION

The results of the readership survey clearly indicate that most readers who read the *Journal* enjoy it and highly rate its editorial content and current format. Variations in the reading levels of specific editorial content are often influenced by specialty. Not surprisingly, for example, 53% of pediatricians read all/most of the essays in pediatrics whereas no general surgeons do and only 2% of internists do at this level. The average reading time of 32 minutes for the *Journal* is quite high considering that 53% of respondents replied that they spend less than two hours per week reading other medical journals and that 77% receive 1-10 and 18% receive 11-20 journals per month.

Popular and current thematic issues such as those on medical malpractice and acquired immunodeficiency syndrome are read by most (88% in both instances). Other themes such as geriatrics and sports medicine had greater appeal for those specialists whose practices involve these areas. Special articles and review articles enjoy very high reading levels.

The *Journal* was given very high scores for the quality and format improvements made over the last several years. Among the format changes made in 1983 was a changeover from interspersed to stacked advertising. The overwhelming majority of readers (92%) prefer stacked over interspersed advertising. This is also the preference of many medical editors. Advertisers and advertising agents prefer interspersed advertisements because they are more frequently seen by readers than stacked ones. The supposition that interspersing attracts more advertisements and hence more revenues is not supported by the *Journal's* recent experience. When the *Journal* moved from interspersed to stacked advertising in 1983, advertising revenues significantly increased.

The *Journal* enjoys a wide readership distributed over many specialty groups. The results of this survey indicate that there are very high levels of satisfaction among all these groups with both the *Journal's* current editorial content and its format. Suggestions for improvement include greater coverage of current health administration and health economics issues and the inclusion of diverse opinion pieces on topical controversial issues.

Acknowledgments. Special thanks are extended to Mr Mark Branca, General Manager of Health Industries Research, for conducting the survey and analyzing the results, and to Mrs Elizabeth Somers for preparing the typescript.

REFERENCES

1. Garfield E: Which medical journals have the greatest impact? *Ann Intern Med* 1986; 105:313-320.
2. An appreciation: Our referees. *NY State J Med* 1987; 87:130-131.

A response to the recommendations of the New York State Commission on Graduate Medical Education concerning teaching in ambulatory care settings

ELEANOR Z. WALLACE, MD; LOUIS M. ALEDORT, MD; RICHARD LEVERE, MD; ALVIN I. MUSHLIN, MD

The New York State Commission on Graduate Medical Education links reorganization of administration and funding of residency training with substantive shifts of the content of such training into primary care areas.[1] The basic educational premise in the report of the commission is that "a majority of trainees completing residency must be in training in the primary care specialties (general medicine, family medicine, general pediatrics, general obstetrics/gynecology) by 1990."

The definition of the primary care internist is evolving, with growing national interest in clarifying the roles of the generalist and the internist/subspecialist. Changes in health care delivery and payment have accelerated this process, as internal medicine training programs struggle to prepare their graduates for the reality of the practice world. The definition of primary care must be carefully developed, for it has important implications for undergraduate as well as graduate medical education. An acceptable definition of general internal medicine must incorporate the profession's scholarly and consultative role as well as its health care delivery and gatekeeper functions.

THE NEED FOR PLANNING AND FISCAL SUPPORT

The New York State Chapter of the American College of Physicians (NYSCACP) is in general agreement that changes in internal medicine residency training are necessary in order to incorporate more effective ambulatory experiences and to include areas of learning not sufficiently emphasized in many residency programs (psychosocial skills, preventive care, office skills in nonmedical specialties, and geriatrics, for example). These educational components, already in place in programs with federally funded primary care training tracks, highlight a series of impediments to successful increase of training time in ambulatory settings. It is the concern of the NYSCACP that any mandate by New York State to increase ambulatory training before these disincentives are dealt with will threaten the educational viability of internal medicine training programs. Any ill-implemented set of changes will jeopardize the ability of New York's internal medicine training programs to attract talented young physicians into what is, at this time, the major primary care specialty in this state. This scenario could result in reduced access to care in New York State.

OBSTACLES TO INCREASED TRAINING IN AMBULATORY SETTINGS

Accreditation Issues. The national accrediting body, the Residency Review Committee (RRC) in internal medicine, has not issued guidelines about how much training time in ambulatory settings is appropriate. In the past, the RRC has suggested that no more than 25% of time so allocated is advisable, raising accreditation concerns about programs that exceed this amount.[2] In addition, certain of the commission's recommendations appear to intrude into other areas of accreditation (nature of sites and adequacy of their supervision, for example) and into the domain of the training program director, who must continue to carry the responsibility for the training program curriculum. It is recommended that any major changes in the training program that will result from the commission's recommendations should be reviewed for compliance with RRC guidelines before implementation. Consultation with the RRC may be necessary.

Availability of Suitable Sites. Ambulatory training must be a carefully developed educational experience providing appropriate patient mix, primary care patient responsibility with continuity, good clinical supervision, availability of consultations, and adequate space in an appropriate environment.

Sites that fulfill these needs are not easy to come by if one appropriately requires that medical education in ambulatory care be a rigorous discipline with a stated curriculum and specific skills to be mastered. Training in ambulatory care cannot be perceived as an apprenticeship. As medical schools move to provide education in ambulatory settings for undergraduates, competition for training sites

---

From the Health Delivery Subcommittee of the Health and Public Policy Committee, New York State Chapter, American College of Physicians.

Address correspondence to Dr Wallace, Department of Medicine, Long Island Jewish Medical Center, New Hyde Park, NY 11042.

will become even greater. It is not yet clear whether adequate sites are available in numbers large enough to satisfy internal medicine training needs.

Clinical Supervision in Nonhospital-Based Sites. There are serious concerns about finding appropriate role models to supervise learning in a rigorous ambulatory education program. Only hospital ambulatory sites have the advantage of a full-time faculty who are already part of the educational system, to undertake the shift to increased ambulatory training. The commission's report suggests an advantage to off-site practice settings. We foresee many problems in implementing such a recommendation.

Internists already practicing in the community are not necessarily appropriate for training a new generation of internists to meet the needs of primary care in a changing health care environment. Similar concerns exist about physicians practicing in other sites which are not primary academic affiliates (health maintenance organizations and community health centers).

Strong faculty development and evaluation programs would be needed to aid, monitor, and insure the suitability of those persons undertaking clinical supervision in primary care settings. These teachers will frequently have had no previous experience in residency training.

In defining suitable training settings in hospitals, the commission report specifies a group practice model as the acceptable ambulatory care experience. Many hospital clinics that care for large numbers of patients will not meet the commission's guideline and, therefore, will not qualify as appropriate sites for residency training. Those clinics that could not convert to a group practice model would then be faced with the loss of resident care deliverers. Funding would be required to replace residents with alternate health care deliverers, or conversion of the clinic to a group practice model would be required.

Medical centers historically have varied approaches to ambulatory care delivery. The best of these should be retained as training sites. At the same time, the commission should work with training program directors to better define the resources required by hospital clinics to make these sites acceptable as training centers.

Funding of Ambulatory Training. Educating residents (and medical students) in outpatient settings is labor intensive and expensive. National policy on graduate medical education reimbursement in nonhospital-based settings continues to penalize training programs that utilize outpatient settings through loss of indirect Medicare reimbursement. At the site itself, there is loss of productivity for the clinical supervisors a well as through trainee use of space. If one adds the cost of maintaining an educational environment (overhead, conference space, curricular materials, research in health care delivery), there is a clear net cost to the site. It is unrealistic to assume that these costs will be absorbed by the sites.

The NYSCACP recommends that movement to increased ambulatory training must be adequately financed. It is not clear how the commission's recommendations would be funded. Only when sources of funding are determined can the training programs and training sites evaluate their potential to provide educationally sound training. At that time, negotiations with sites thought to be appropriate for internal medicine residency training can be undertaken.

IMPACT OF THE COMMISSION'S RECOMMENDATIONS ON INPATIENT CARE

At this time, residents in training provide the bulk of care for inpatients. This service function will be sharply curtailed if ambulatory time increases from 10% (current standard) to 25% or more. The appropriate balance between service and education in internal medicine training has not been adequately defined. What is clear is that alternate provision of care for groups of inpatients will be needed if the numbers of residents on inpatient services decrease.

If, as recommended by the commission, care is provided by either full-time physicians or physician extenders, then two levels of patient care will be generated. By most estimates, either of these approaches will be more expensive than care provided by residents. Until appropriate funding for adequate care of inpatients is identified, major movement of residents from inpatient to outpatient settings cannot be accomplished and should not be mandated.

Furthermore, the RRC and most program directors have perceived the inpatient experience as the core of residency and medical student training in internal medicine. Great care must be taken to preserve the educational advantages of the inpatient experience—such as peer interaction, learning from a diversity of faculty and attendings, exposure to other services—while accomplishing the needed increase in primary care ambulatory experience. The transition must be done with adequate lead time for planning and implementation.

CONCLUSIONS

The NYSCACP is concerned about the impact of implementation of the recommendations of the New York State Commission on Graduate Medical Education on residency training in internal medicine.[1] Training in internal medicine must be maintained at the highest level of educational excellence, with appropriate preservation of its acknowledged strengths.

The NYSCACP agrees with the need for increased training in primary care internal medicine, but sees important disincentives to implementing such change at this time. These problems include the following: lack of suitable sites for ambulatory training, lack of suitable clinical supervision in off-hospital sites, and lack of clear methods for funding ambulatory training and for replacing inpatient care currently provided by residents. Educational viability and accreditation of current programs could be jeopardized by untimely, mandated change.

A shift to more primary care training in ambulatory sites could be best accomplished after funding issues are resolved. Then the directors of training programs could best determine how educational excellence can be maintained in the ambulatory and inpatient settings while accomplishing the transition.

REFERENCES

1. *Report of the New York State Commission on Graduate Medical Education*, Albany, NY, State of New York Department of Health, 1986.
2. Wartman SA: The RRC and the "25% rule" [editorial]. *SREPCIM Newsletter*, Jan–Feb 1985, pp 2–3.

Stillbirth following maternal Lyme disease

ALAN B. MACDONALD, MD; JORGE L. BENACH, PHD; WILLY BURGDORFER, PHD

***Borrelia* infection acquired** during gestation may result in death or malformation of the developing fetus.[1-3] This report describes a clinicopathologic investigation of a stillborn fetus that led to a retrospective diagnosis of Lyme disease contracted during the first trimester of pregnancy.

CASE REPORT

A 24-year-old woman enjoyed frequent hikes in the Wasatch mountains outside of Salt Lake City, Utah, in the spring and summer of 1984. In mid-July she was told by her physician that she was approximately eight to ten weeks pregnant. She moved to Southampton, New York, in September 1984. Regular prenatal care was supervised by an obstetrician. Her antepartum course was uneventful until February 9, 1985, when spontaneous labor commenced on the expected day of confinement. Fetal heart sounds were undetectable when she arrived at the hospital, and an ultrasound examination confirmed fetal demise. A 2,500-g stillborn was delivered vaginally. An autopsy of the fetus was requested.

Following completion of the autopsy, the pathologist and obstetrician interviewed the patient for any previous history of signs and symptoms associated with Lyme disease. She reported that following a hike in one of the parks in Wasatch County in May 1984, a red-purple lesion of the skin had developed near the left knee. She presumed that she had been bitten by a small insect. The lesion enlarged to become an annular erythematous patch approximately 14 cm in diameter and then gradually faded and disappeared. Several days after the onset of the skin lesion, pain and swelling developed in the left knee, lasting approximately one week. She did not seek medical attention for these symptoms. With the exception of oral digoxin, 0.5 mg daily, prescribed for mitral valve prolapse, she had taken no medications while pregnant.

A standard autopsy was performed, supplemented by cultures of fetal liver, spleen, kidney, and heart in Barbour Stoenner Kelly medium,[4] which supports the in vitro growth of *Borrelia* spirochetes. Paraffin sections of formalin-fixed autopsy tissues were examined by indirect immunofluorescence (IFA)[5,6] and Warthin Starry silver impregnation. Maternal serum obtained ten days following delivery was divided into three aliquots, and Lyme serologic tests were performed at the Centers for Disease Control (Atlanta, Ga), New York State Department of Health, and Yale University (New Haven, Conn) by IFA and enzyme-linked immunosorbent assay (ELISA) methods.[7,8] *Treponema pallidum* serologic tests were done by the Treponema Research Branch at the Centers for Disease Control.

The fetus showed early cutaneous maceration and no external malformations. Radiographs disclosed no skeletal anomalies. A 4-mm defect in the muscular portion of the basal interventricular septum was classified as an atrioventricular canal ventricular septal defect by a consultant in cardiac pathology (Jesse E. Edwards, MD). No other malformations were identified. The placenta weighed 270 g; the disc measured 17 × 12 × 2 cm; and serial sections revealed no gross abnormalities in the cotyledons, or infarctions, intervillous thrombi, or evidence of abruption.

Spirochetes were recovered from fetal liver, but primary cultures and subcultures were contaminated with bacillus species. The cultured spirochetes bound the mouse IgG monoclonal antibody H5332 (provided by Dr Alan Barbour, Rocky Mountain Laboratory, Hamilton, Mont). Antibody H5332 recognizes the outer surface membrane of *Borrelia burgdorferi*.

Routine sections of fetal tissues showed mild autolysis and no significant inflammation. Placental tissues showed rare plasma cells in isolated villi. Spirochetes were identified by immunofluorescence in the fetal myocardium, adrenal gland, and subarachnoid space of the midbrain; and silver stains disclosed rare spirochetes in the myocardium, placenta, liver, and brain.

Lyme serologic studies on postpartum maternal blood were positive at two of three laboratories by IFA methods and by ELISA methods, but negative by IFA and ELISA at a third laboratory. Serologic tests for syphilis, including the RPR, MHA-TP, and FTA-ABS, were negative at the Centers for Disease Control.

DISCUSSION

Transmission of the spirochete *Borrelia burgdorferi* from mother to fetus during the first trimester of pregnancy was followed by overwhelming spirochetosis in the fetus, with intrauterine death near term. Maternal infection was acquired in the state of Utah, where the tick *Ixodes pacificus* is known to be a vector of *B burgdorferi*.[9,10]

The observations in this case complement those of Schlesinger et al,[11] who described the first case of maternal-fetal transmission of *B burgdorferi* in a young pregnant woman who acquired Lyme disease while camping in Wisconsin. Her infant died 39 hours after birth of complications related to a complex congenital cardiac malformation. Although spirochetes were not found in the myocardium, rare spirochetes were seen in fetal kidney, spleen, and bone marrow. No attempt was made to culture spirochetes from autopsy tissues. Maternal serum was reactive against *B burgdorferi* by IFA and ELISA methods at two laboratories and was falsely nonreactive at a third laboratory.

Symptoms of Lyme disease are protean, and although the characteristic expanding annular erythema followed by arthritis was present in this case, many other clinical presentations are possible in cases in which cutaneous or articular symptoms are absent.[12,13] And, as we describe in this report, even classical symptoms may be overlooked by the patient and her physicians unless

---

From the Department of Pathology, Southampton Hospital, Southampton, NY (Dr MacDonald), the New York State Department of Health and the Department of Pathology, State University of New York at Stony Brook Health Sciences Center School of Medicine, Stony Brook, NY (Dr Benach), and the Laboratory of Pathobiology, Rocky Mountain Laboratory, Hamilton, Mont (Dr Burgdorfer).

Address correspondence to Dr MacDonald, Associate Pathologist, Southampton Hospital, 240 Meeting House Lane, Southampton, NY 11968.

a specific set of questions is included in the patient's review of symptoms. The clinical examination of the patient and the clinical diagnosis of probable Lyme disease must be the "gold standard of diagnosis," because Lyme serologic studies may be nondiagnostic due to interlaboratory variation in detection of serum antibody[14] or due to the delay between primary infection and the production of serum antibody, which is recognized for every serologically defined infectious disease.

Two cases of transplacental transmission of *Borrelia burgdorferi* have been associated with fetal death and cardiac malformation. Different anomalies were found in each case; therefore, a cause and effect relationship cannot be determined between the spirochete in tissue and a specific lesion. We recommend that pathologists search for spirochetes in tissues of stillborn fetuses who show malformations in the cardiovascular system. Clinicians should investigate the possibility of exposure to Lyme disease during the first trimester of pregnancy in such cases, for cardiac organogenesis is essentially complete at the end of the first trimester of gestation.[15]

We believe that evidence is now sufficient to specifically alert women who live in endemic areas for Lyme disease and their physicians to recognize the signs and symptoms of infection by *Borrelia burgdorferi* and related *Borrelia* species. It is our further recommendation that pregnant women with symptoms of Lyme disease be treated immediately with penicillin in doses equivalent to those used for syphilis in pregnancy, and that therapy not be delayed or withheld pending results of serologic studies.

REFERENCES

1. Fihn S, Larson EB: Tick-borne relapsing fever in the Pacific Northwest: An underdiagnosed illness? *West J Med* 1980; 133:203-209.
2. Southern PM Jr, Sanford JP: Relapsing fever: A clinical and microbiological review. *Medicine* 1969; 48:129-149.
3. Markowitz LE, Steere AC, Benach JL, et al: Lyme disease during pregnancy. *JAMA* 1986; 255:3394-3396.
4. Kelly R: Cultivation of *Borrelia hermsi. Science* 1971; 173:443-444.
5. Hunter EF, Greer PW, Swisher BL, et al: Immunofluorescent staining of Treponema in tissues fixed with formalin. *Arch Pathol Lab Med* 1984; 108:878-880.
6. Dorsett, BH, Ioachim HL: A method for the use of immunofluorescence on paraffin-embedded tissues. *Am J Clin Pathol* 1978; 69:66-72.
7. Russell H, Sampson JS, Schmid GP, et al: Enzyme-linked immunosorbent assay and indirect immunofluorescence assay for Lyme disease. *J Infect Dis* 1984; 149:465-470.
8. Benach JL, Coleman JL, Habicht GS, et al: Serological evidence for simultaneous occurrences of Lyme disease and babesiosis. *J Infect Dis* 1985; 152:473-477.
9. Burgdorfer W, Lane RS, Barbour AG, et al: The western black-legged tick, *Ixodes pacificus:* A vector of *Borrelia burgdorferi. Am J Trop Med Hyg* 1985; 34:925-930.
10. Bulletin of the Utah Department of Health, 1982.
11. Schlesinger PA, Duray PH, Burke BA, et al: Maternal-fetal transmission of the Lyme disease spirochete, *Borrelia burgdorferi. Ann Intern Med* 1985; 103:67-68.
12. Schrock CG: Lyme disease: Additional evidence of widespread distribution. Recognition of a tick-borne dermatitis-encephalitis-arthritis syndrome in an area of known ixodes tick distribution. *Am J Med* 1982; 72:700-706.
13. Reik L Jr, Burgdorfer W, Donaldson JO: Neurologic abnormalities in Lyme disease without erythema chronicum migrans. *Am J Med* 1986; 81:73-81.
14. Hedberg CW, Osterholm MT, MacDonald KL, et al: An interlaboratory study of antibody to *Borrelia burgdorferi. J Infect Dis* 1987; 155:1325-1327.
15. Moore KL: *The Developing Human: Clinically Oriented Embryology*. Philadelphia, WB Saunders Co, 1973, pp 239-280.

Visual loss and deafness associated with meningeal carcinoma

MICHAEL GAROFALO, JR, MD; CAROLYN MARTIN, MD; MICHAEL BYKOFSKY, MD;
JOSEPH CASARONA, MD

Meningeal carcinomatosis is a great imitator and can present with headaches, multiple cranial neuropathies,[1] focal neurologic deficits, signs of nerve root compression, and changes in mental status. We report a unique case with the simultaneous onset of blindness and deafness as the major neurologic deficits.

CASE REPORT

Two days after acquiring an upper respiratory infection, a 68-year-old man noted the onset of loss of vision in both eyes, greater in the right than in the left, and bilateral hearing loss. These symptoms progressed during a two-week period. The patient's medical history included treatment of a low-grade bladder carcinoma by cauterization in 1977. In 1979, he was found to have carcinoma of the stomach and was treated with a subtotal gastrectomy. After surgery, the patient refused further therapy; he took "antacids for heartburn."

On initial neurologic examination, the patient was alert and capable of normal speech, but unable to see or hear. There was bilateral blindness, with disc pallor and fixed dilated pupils. Bilateral deafness was also noted. The rest of the neurologic examination was normal. The cerebrospinal fluid (CSF) was clear and colorless, with a normal opening pressure, but with 27 white blood cells per mm^3 and 100% mononuclear leukocytes. The protein level was 115 mg/dL; glucose, 89 mg/dL; IgG, 20 mg/dL. Cultures for bacteria, fungi, and acid-fast bacilli were negative. CSF cytologic studies showed "several mildly atypical cells." An electroencephalogram, chest film, and skull film were all within normal limits. Brainstem auditory evoked potentials revealed no reproducible waveforms, and an audiogram showed bilateral profound sensorineural hearing loss. A pattern shift visual-evoked potential and flash-evoked potential showed no reproducible waveforms. A computed tomographic (CT) scan of the head with contrast revealed no abnormality.

The patient was discharged on tapering doses of prednisone without any change in his clinical course. Seven days later, he was readmitted with an acute change of mental status manifested by severe agitation and paranoid hallucinations. Bladder and bowel incontinence were noted at this time. He remained afebrile, and his vital signs were normal. Repeat laboratory studies of blood and

---

From the Department of Neurology, St Vincent's Hospital and Medical Center of New York.

Address correspondence to Dr Garofalo, Department of Neurology, St Vincent's Hospital and Medical Center of New York, 153 West 11th St, New York, NY 10011.

urine were normal. He was treated with dexamethasone, 40 mg/day, and sedation with haloperidol. A repeat CSF examination revealed no white blood cells at this time; positive cytologic findings showed "scattered single and clustered malignant cells consistent with adenocarcinoma." The spinal fluid protein and glucose levels were 103 mg/dL and 70 mg/dL, respectively, with a serum glucose level of 84 mg/dL. A repeat audiogram was unchanged.

Dysphagia and a mild right peripheral VII nerve weakness then developed, followed one week later by urinary retention and decreased sensation of the perineum. Whole brain radiation and chemotherapy were instituted as palliative measures, but the patient's clinical condition deteriorated, with associated nuchal rigidity, abdominal pain, and hepatomegaly. Increasing lethargy and coma preceded his death, which occurred six weeks after the onset of his first symptoms. The family refused an autopsy.

DISCUSSION

Blindness has been reported in up to 33% of cases of meningeal carcinomatosis,[2] and though usually progressive over several weeks, it has been reported to cause visual loss within 48 hours.[2,3] The exact nature of the pathophysiology has not been elucidated, but increased intracranial pressure, compression, and tumor infiltration have been suggested as the most likely causes.[4,5]

As early as 1900, Saenger[6] described deafness with infiltration of the acoustic nerve in the subarachnoid space by metastatic cancer. In 1974 Olsen et al[7] reported that only 6% of patients with meningeal carcinomatosis presented with hearing loss. The clinical presentation in five cases reported by Alberts and Terrance[8] included unilateral hearing loss associated with tinnitus, and unilateral loss which rapidly progressed to severe bilateral involvement.

Despite the absence of an autopsy in this case, carcinomatous meningitis is the most likely explanation of this patient's clinical picture, given the positive CSF findings. In the series of Olson et al,[7] each of 30 patients with malignant cells in the CSF had meningeal carcinomatosis at autopsy.

A review of the literature disclosed only one other case, reported by Knierim[9] in 1908, of rapid onset blindness and deafness associated with meningeal carcinomatosis from primary gastric carcinoma.

The examination of the spinal fluid is the single most important laboratory study in establishing the diagnosis of carcinomatous meningitis,[10] although the presence of abnormal CSF levels of enzymes has been reported to help in making a diagnosis.[11] This case and others emphasize that initial negative cytologic tests do not rule out the possibility of malignant meningeal involvement.

REFERENCES

1. Rubinstein MK: Cranial mononeuropathy as the first sign of intracranial metastases. *Ann Intern Med* 1969; 70:49-54.
2. Altrocchi PA, Reinhardt PH, Eckman PB: Blindness and meningeal carcinomatosis. *Arch Ophthalmol* 1972; 88:508-512.
3. Terry TL, Dunphy EB: Metastatic carcinoma in both optic nerves simulating retrobulbar neuritis. *Arch Ophthalmol* 1933; 10:611-614.
4. McCrary JA 3d, Patrinely JR, Font RL: Progressive blindness caused by metastatic occult signet-ring cell gastric carcinoma. *Arch Ophthamol* 1986; 104:410-413.
5. Goldstein I, Wexler D: Metastasis in sheath of optic nerve from carcinoma of stomach. *Arch Ophthalmol* 1931; 6:414-419.
6. Saenger A: Uber hirnsymptom bei carcinomatose. *Munch Med Woch;* 1900; 47:341-342.
7. Olson ME, Chernick NL, Posner JB: Infiltration of the leptomeninges by systemic cancer. A clinical and pathologic study. *Arch Neurol* 1974; 30:122-137.
8. Alberts MC, Terrance CF: Hearing loss in carcinomatous meningitis. *J Laryngol Otol* 1978; 92:233-241.
9. Knierim G: Diffuse meningeal carcinomatosis with blindness and deafness in gastric cancer. *Beitr Pathol Anat Allege Pathol* 1908; 44:409-429.
10. Posner JB: Management of central nervous system metastases. *Semin Oncol* 1977; 4:81-91.
11. Hansen M, Pedersen AG: Tumor markers in patients with lung cancer. *Chest* 1986; 89(suppl): 219S-224S.

Microscopic bilateral adenocarcinoma arising in ovarian endometrial cysts

MARY F. A. C. DIMAIO, MD; ELLEN KAHN, MD; A. FENTON, MD

Malignant transformation in ovarian endometriosis was first described by Sampson[1] in 1925. The following criteria were put forth as establishing the diagnosis: the coexistence of endometriosis and carcinoma in the same ovary, bearing a similar histologic relationship to each other, as do benign and malignant endometrial tissue in the uterus; and the presence of carcinoma arising from the endometriosis and not invading it from another source. Scott[2] further qualified these criteria and required microscopic demonstration of continuity between the benign and malignant epithelium.

Ovarian endometriosis can give rise to a variety of tumors.[3] We present a rare case of bilateral microscopic adenocarcinoma arising in smooth-lined ovarian endometrial cysts, which fulfills all criteria and is to our knowledge the second such report in the English literature.[4]

CASE REPORT

This was the second admission to North Shore University Hospital of a 44-year-old woman, gravida 0 para 0, who presented on July 23, 1982, with the chief complaint of pelvic pain and menorrhagia of one week's duration. The physical examination on admission was remarkable for an eight-week size uterus. Laparoscopy demonstrated multiple subserosal leiomyomata of the uterus and adhesion of the left ovary to the posterior leaflet of the broad ligament. The right ovary and both fallopian tubes appeared normal. Endometrial curettage revealed cystic hyperplasia.

The patient continued to have pelvic pain complicated by urinary frequency. A pelvic sonogram in 1984 revealed a myomatous uterus, but poor visualization of the adnexa. In view of the patient's persistent symptoms and increasing uterine size, she was admitted in 1985 for total abdominal hysterectomy and bilateral salpingo-oophorectomy.

From the Departments of Pediatrics (Dr DiMaio), Laboratories (Dr Kahn), and Obstetrics and Gynecology (Dr Fenton), North Shore University Hospital, Manhasset, NY, and Cornell University Medical College, New York, NY.

Address correspondence to Dr DiMaio, 315 E 86th St, Apt 10DE, New York, NY 10028.

At surgery, bilateral ovarian hemorrhagic cysts, adherent to the cul de sac, were noted. Endometriosis involving both uterosacral ligaments and the bladder flap was grossly identifiable. Following diagnosis of a benign hemorrhagic cyst of the right ovary, based on the frozen section, a hysterectomy and bilateral salpingo-oophorectomy were performed. The postoperative course was uneventful.

On gross examination, the uterus and cervix weighed 235 g and measured 11 × 8 × 4.5 cm. There were multiple intramural and subserosal leiomyomas, measuring up to 5 cm in greatest dimension, and demonstrating the typical white, whorled pattern. Adenomyosis was identified within the posterior myometrium. The left ovary measured 3 × 3 cm and contained a gray, smooth-lined cyst that measured 3 cm in diameter. The right ovary demonstrated a smooth-lined 3 × 2-cm cyst, with focal areas of brown discoloration of its internal surface. This cyst contained hemorrhagic fluid.

The ovarian cysts were examined in their entirety. On microscopic evaluation, they had the general characteristics of an endometrial cyst (Fig 1). The stroma was attenuated, and contained hemosiderin-laden macrophages and isolated endometrial glands lined by a predominantly cuboidal epithelium. In addition, multiple foci of a well-differentiated mucus-producing adenocarcinoma were noted in the right ovary (Fig 2), whereas only one focus was found on the left. The carcinoma was characterized by glandular crowding or a cribriforme pattern (Fig 3). The lining cells were frequently stratified and had oval nuclei and at times prominent nucleoli. Segmented leukocytes infiltrated the neoplastic epithelium. The carcinoma was restricted in all sections to the inner lining of the cysts. A transition zone between the non-neoplastic lining and the neoplastic lining was characterized by nuclear crowding and stratification of the epithelial cells, an increase in nuclear size, and the presence of isolated mitoses.

Histologic examination of the uterus revealed a secretory endometrium with focal cystic hyperplasia, adenomyosis, and leiomyomata. In spite of extensive tissue sampling, no endometrial adenocarcinoma was evident. The fallopian tubes were unremarkable.

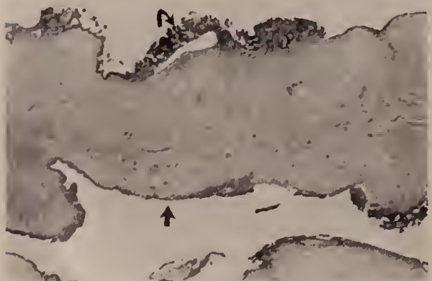


FIGURE 1. Low-power view of the right ovarian endometrial cyst (straight arrow) associated with adenocarcinoma (curved arrow) (hematoxylin-eosin stain; original magnification × 50).

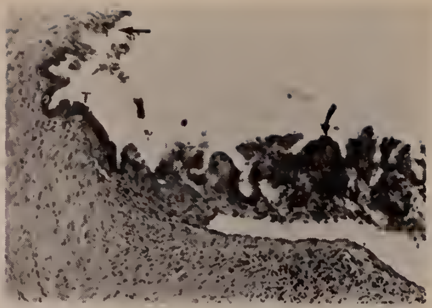


FIGURE 2. Higher magnification of the endometrial cyst depicting the continuity of the non-neoplastic endometrium (straight arrow) with adenocarcinoma (curved arrow). T, transition zone (hematoxylin-eosin stain; original magnification × 200).

Discussion

The endometriotic origin of malignant neoplasms was first recognized by Sampson[1] in 1925, and subsequent reports in the literature have demonstrated that any tumor that arises in uterine endometrium can also arise in endometriotic tissue.[3,5-8] The incidence of this malignant degeneration in ovarian endometriosis approximates 0.56–2%.[9-12] However, the actual frequency of the neoplastic transformation is difficult to determine for several reasons. First, the prevalence of endometriosis in the general population is unknown. Second, a neoplasm may have obliterated the endometriotic tissue from which it originated. Finally, numerous microscopic sections may be necessary to identify small foci of tumor adjacent to benign tissue. The predominant malignancies reported to arise in ovarian endometriosis are carcinomas, although there are sporadic case reports of sarcomas, carcinosarcomas, and mixed mesodermal tumors.[6,7,13] Well-differentiated adenocarcinomas and adenoacanthomas are the most prevalent neoplasms and occur with equal frequency.[11,12] The most common gross pathologic features are abnormally large cysts with the presence of either small papillary or solid areas projecting from the internal surface.[11] Mostoufizadeh[3] states that a diameter of 15 cm is suggestive of the development of carcinoma.

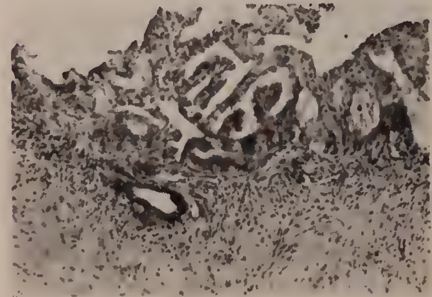


FIGURE 3. Well-differentiated adenocarcinoma with glandular crowding and nuclear stratification (hematoxylin-eosin stain; original magnification × 500).

We have reviewed the literature pertaining to neoplasms that originate within ovarian endometriosis and have included only those cases that strictly fulfill the criteria of Sampson[1] and Scott[2] (Table I). In 54% of the cases, the gross pathologic finding was a cystic structure with either a focal solid nodule or papillary excrescences. The majority of endometrial cysts measured 6 cm or more in greatest dimension, but there were instances of malignant endometrial cysts as small as 1 cm. Adenocarcinomas and adenoacanthomas occurred with equal frequency. The case presented here also demonstrates that carcinoma may occur in small cysts.

There is a paucity of data in the literature pertaining to the clinical presentation, treatment, and follow-up of women with malignant ovarian endometrial cysts. The average age at presentation of women with carcinoma or carcinosarcoma is approximately 40 years.[3,6,12] The most common presenting symptom is pain often associated with abdominal enlargement, bleeding, or urinary frequency. The pain is often described as continuous, unlike the dysmenorrhea of endometriosis. Acute spontaneous rupture of the cyst is a rare occurrence (5%).[11] Tumor metastasis noted at the time of surgery is also rare. In summary, a change in the typical pattern of pain, an enlarging pelvic mass, and/or spontaneous cystic rupture may indicate to the clinician a malignant process within an endometriotic cyst.[11]

Since clinical follow-up was not obtained in most case studies, it is not possible to accurately determine the incidence of recurrence or mortality. Scully[7] and Thompson[12] each reported one case of tumor recurrence during a nine- and ten-year follow-up, respectively. Reported fatalities were attributed to metastasis from squamous cell carcinoma and adenocarcinoma.[5,7,10,14] Three of these cases were associated with cyst rupture at the time of surgical excision, suggesting that cyst rupture can result in tumor recurrence or metastasis.[5,7,10]

If it is conclusively demonstrated that a carcinoma originated from an ovarian endometriosis, the issue of treatment may be problematic. Few articles have sufficient patient follow-up or patient numbers to provide clear-cut guidelines for the clinician. Each case

TABLE I. Reported Cases of Carcinoma Arising in Ovarian Endometriosis

Reference	Case No.	Age (yrs)	Size (cm)*	Consistency	Diagnosis	Treatment	Outcome
Sampson[1]	5	46	NS	C	Adenocarcinoma	TAHBSO	NS
Scott[2]	3	50	NS	C	Adenocarcinoma	TAHBSO	NS
Bacher & Hertzog[4]	—	48	R—1.5	C	Adenocarcinoma	TAHBSO	NS
			L—1.5	C	Adenocarcinoma		
Lele et al[5]	—	36	15 × 12	C	Squamous cell carcinoma	TAHBSO, CT, RT	Died 3 mo with metastasis
Marchevsky & Kaneko[6]	—	44	R—11 × 9 × 5	S/C	Carcinosarcoma	TAHBSO, CT	Alive and well 4 mo
			L—2 × 2	C/P	Adenocarcinoma		
Scully et al[7]	1-4	38-55	NS	C/P(3)	Adenocarcinoma		
				C/S(1)	Adenocarcinoma		
						TAHBSO	Recurred 9 yr later
						TAHBSO	Alive and well 7 yr
						TAHBSO, RT	Alive 9 yr, malignant ascites
						TAHBSO, RT	Died 17 mo with metastasis
Gray & Barnes[9]	1	29	R—8	C/S	Adenoacanthoma	TAHBSO	Alive and well 4 yr
			L—small	C	Adenoacanthoma		
	2	60	15 × 12 × 8	C/S	Clear cell adenocarcinoma	TAHBSO	Alive and well 6 yr
	4	42	15 × 10	C/P	Adenocarcinoma	TAHBSO	NS
	4	46	Small	C	Adenocarcinoma	TAHBSO	NS
Corner et al[10]	4	54	8 × 3 × 3	C/P	Adenocarcinoma	SAHBSO	Died 18 mo with metastasis
	5	50	8	C/P	Adenocarcinoma	TAHBSO, RT	Alive and well 6 yr
Fathalla[11]	2	47	NS	C	Adenoacanthoma	SAHBSO, RT	Alive and well 3 yr
	3	36	Grapefruit	C/P	Adenocarcinoma	TAHBSO	NS
	4	27	15	C/P	Endometrioid carcinoma	TAHBSO	NS
Thompson[12]	1	32	10 × 6 × 3	C	Adenoacanthoma	BSO	Recurred 10 yr later
	3	37	1.7	S	Adenoacanthoma	SAHBSO	Alive and well 8½ yr
	5	27	L—12	C	Adenoacanthoma	SAHBSO	Alive and well 6 yr
			R—12	C	Adenoacanthoma		
	7	37	6	C	Adenoacanthoma	TAHBSO	Alive and well 5 yr
	10	33	Fragments	S	Adenoacanthoma	L-SOP R-partial OP	Alive and well 4 yr
	14	52	Large	C/P	Adenoacanthoma	TAHBSO, RT, omentectomy	Alive and well 5½ yr
	17	28	4	C	Adenoacanthoma	L-OP R-partial OP	Alive and well 2 mo
Cooper[13]	—	53	16 × 10 × 10	C/P	Mixed mesodermal and clear cell carcinoma	TAHBSO	NS
McCullough et al[14]	—	61	Large	C	Squamous cell carcinoma	TAHBSO	Died 3 mo
Mabry[15]	—	25	2.5	C/P/S	Adenoacanthoma	TAHBSO	Alive and well 1 yr
Dockerty[16]	5	34	NS	C/S	Adenocarcinoma	TAHBSO, RT	NS
	6	36	15	C/S	Adenocarcinoma	TAHBSO	Alive and well 14 yr
	7	40	NS	C/S	Adenoacanthoma	TAHBSO	Alive and well 5 yr
	9	40	NS	C/S	Adenoacanthoma	TAHBSO	NS
Stern[17]	—	38	10 × 6 × 4.5	C/S	Adenoacanthoma	L-OP	Alive and well 3½ yr
Wade[18]	—	37	10	C/P	Adenoacanthoma	TAHBSO	NS
Kumar et al[19]	—	39	8.5 × 8 × 4	C/P	Adenocarcinoma	TAHBSO	NS
Plate[20]	—	41	Child's head	C/P	Clear cell adenocarcinoma	L-SOP	NS
Kreiner[21]	—	48	NS	C	Adenocarcinoma	TAHBSO	Alive and well 1 yr
Green & Enterline[22]	1	51	6	C/P	Adenocarcinoma	TAHBSO	NS
Dubrauszky & Niendorf[23]	1	43	Newborn's head	C/S	Adenocarcinoma	TAHBSO	NS
Teilum[24]	—	42	10	C/P	Adenocarcinoma	SAHBSO	NS
Ferreira & Clayton[25]	1	40	NS	C	Adenocarcinoma	SAHBSO	NS

(continued)

Table I continued

Reference	Case No.	Age (yrs)	Size (cm)*	Consistency†	Diagnosis	Treatment‡	Outcome
Hejda & Boháčová[26]	1	38	6	C	Adenoacanthoma	TAHBSO	Alive and well 5 yr
	2	58	NS	C	Adenocarcinoma	TAHBSO	Alive and well 2 yr
Concannon et al[27]	—	38	L—1	C	Adenoacanthoma	TAHBSO	Alive and well 25 mo
			R—NS	C/P	Adenoacanthoma		

* NS, not stated; R, right; L, left.

† C, cystic; S, solid; P, papillary.

‡ TAHBSO, total abdominal hysterectomy and bilateral salpingo-oophorectomy; CT, chemotherapy; RT, radiation therapy; SAHBSO, subtotal abdominal hysterectomy and bilateral salpingo-oophorectomy; BSO, bilateral salpingo-oophorectomy; SOP, salpingo-oophorectomy; OP, oophorectomy.

must be viewed within the context of tumor type and extension, and age and parity of the patient. The surgical approach most often utilized is a total abdominal hysterectomy and bilateral salpingo-oophorectomy, based on a preoperative diagnosis of endometriosis. In most cases, the malignancy is found at the time of pathologic examination. Occasionally, adjuvant therapy is added. Rare aggressive tumors of endometriotic origin, such as squamous cell carcinoma and carcinosarcoma, require a multidisciplinary approach which includes surgery, chemotherapy, and radiation.[5,6] Management is less clear in patients with endometriotic adenocarcinoma. Mabry[15] believes that the most efficacious treatment for endometriotic adenocarcinoma is a total abdominal hysterectomy and bilateral salpingo-oophorectomy. Scully et al[7] state that optimal treatment should include radiation therapy if residual disease is anticipated. Other authorities may elect to treat ovarian adenocarcinoma originating in endometriosis as an ovarian primary. This approach was used in the patient described here.

There are cases in which the possibility of conservative surgical management may be appropriate. Thompson[12] accumulated 17 cases of ovarian adenoacanthomas arising in endometriosis. He noted that this tumor had a low malignant potential and good prognosis, and advocated a limited surgical approach.

The incidence of endometriosis as a significant precancerous condition is less than 2%. However, endometriotic tissue is subject to the same hormonal influences as intrauterine endometrium and thus can undergo malignant degeneration.

We have reported a case of bilateral ovarian adenocarcinoma arising in innocuous endometrial cysts. The malignant transformation occurred in cysts measuring 2–3 cm that were devoid of nodularity or papillary excrescences. We wish to emphasize the necessity of thorough gross and microscopic examination of endometrial cysts in order to avoid overlooking a carcinoma.

REFERENCES

1. Sampson JA: Endometrial carcinoma of the ovary, arising in endometrial tissue in that organ. *Arch Surg* 1925; 10:1–72.
2. Scott RB: Malignant changes in endometriosis. *Obstet Gynecol* 1953; 2:283–289.
3. Mostoufizadeh M, Scully RE: Malignant tumors arising in endometriosis. *Clin Obstet Gynecol* 1980; 23:951–963.
4. Bacher WC, Hertzog AJ: Bilateral ovarian carcinoma arising in endometrial cysts; Report of a case. *Am J Clin Path* 1951; 21:965–968.
5. Lele SB, Piver MS, Barlow JJ, et al: Squamous cell carcinoma arising in ovarian endometriosis. *Gynecol Oncol* 1978; 6:290–293.
6. Marchevsky AM, Kaneko M: Bilateral ovarian endometriosis associated with carcinosarcoma of the right ovary and endometrioid carcinoma of the left ovary. *Am J Clin Pathol* 1978; 70:709–712.
7. Scully RE, Richardson GS, Barlow JF: The development of malignancy in endometriosis. *Clin Obstet Gynecol* 1966; 9:384–411.
8. Zussman WV, Hollander JS: Unilateral carcinoma arising in bilateral ovarian endometriosis. *Am J Obstet Gynecol* 1968; 101:261–263.
9. Gray LA, Barnes ML: Relation of endometriosis to ovarian carcinoma. *Am Surg* 1965; 31:798–806.
10. Corner GW, Hu CY, Hertig AT: Ovarian carcinoma arising in endometriosis. *Am J Obstet Gynecol* 1950; 59:760–774.
11. Fathalla MF: Malignant transformation in ovarian endometriosis. *J Obstet Gynaec Brit Comm* 1967; 74:85–92.
12. Thompson JD: Primary ovarian adenoacanthoma; its relationship to endometriosis. *Obstet Gynecol* 1957; 9:403–416.
13. Cooper P: Mixed mesodermal tumor and clear cell carcinoma arising in ovarian endometriosis. *Cancer* 1978; 42:2827–2831.
14. McCullough K, Froats ER, Falk HC: Epidermoid carcinoma arising in an endometrial cyst of the ovary. *Arch Pathol* 1946; 41:335–337.
15. Mabry EB: Adenocarcinoma of endometriosis of the ovary: A case report. *Am J Obstet Gynecol* 1971; 110:783–784.
16. Dockerty MB: Malignancy complicating endometriosis. Pathologic features in 9 cases. *Am J Obstet Gynecol* 1962; 83:175–179.
17. Stern CA: Adenocarcinoma of the ovary with probable origin in endometrial cyst. *Am J Obstet Gynecol* 1958; 75:282–284.
18. Wade WG: A case of primary ovarian adenoacanthoma arising in an endometrial cyst. *J Obstet Gynaec Brit Emp* 1960; 67:136–137.
19. Kumar D, Anderson W, Van Wyck D: Ovarian carcinoma arising in an endometrial cyst. *J Obstet Gynaec Brit Emp* 1955; 62:375–377.
20. Plate WP: Clear-cell adenocarcinoma in an endometrioid ovarian cyst. *Obstet Gynecol* 1966; 27:428–431.
21. Kreiner E: Adeno-carcinoma in situ in einem Endometrioseherd des Ovars. *Arch Gynaekol* 1978; 225:209–213.
22. Greene JW Jr, Enterline HT: Carcinoma arising in endometriosis. Report of two cases. *Obstet Gynecol* 1957; 9:417–421.
23. Dubrauszky V, Niendorf F: Uber die maligne Entartung heterotoper Korpusschleimhautwucherungen. *Ztsch Geburtsh U Gynak* 1951; 134:213–236.
24. Teilum G: Carcinoma arising in ovarian endometriosis. *Acta Obstet Gynecol Scand* 1945; 25:377–384.
25. Ferreira HP, Clayton SG: Three cases of malignant change in endometriosis, including two cases arising in the recto-vaginal septum. *J Obstet Gynaec Brit Emp* 1958; 65:41–44.
26. Hejda V, Bohácová J: Malignant transformation in ovarian endometriosis. *Neoplasma* 1974; 21:69–74.
27. Concannon PJ, Veprovsky EC, Garrow I: Bilateral adenoacanthoma of the ovary arising in endometriosis. Review of the literature and report of a case. *Am J Obstet Gynecol* 1962; 83:180–184.

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Variations in energy expenditure during physical exercise

TO THE EDITOR: We read with interest the report by Birrer and Daller[1] on "The exercise prescription." We at the Department of Biological Anthropology at Oxford University are also interested in energy expenditure during exercise and various daily activities.

Our method consists of having an experimental subject wear a 24-hour heart rate monitor (Medilog, Oxford Electronic Instruments, Abingdon, UK) and keep a precise diary of activity for the study period. The resultant tape is then analyzed utilizing a computer (Research Machines 380Z, Oxford, UK). During exercise periods, the pulse rate is checked manually and recorded in the diary to verify the results from the monitor. In addition, the subject exercises at three levels of activity (light, medium, and moderate) on a bicycle ergometer (Monark, Stockholm, Sweden) while both the pulse rate and the V_{O_2} level are recorded (the latter using a portable Oxylog, PK Morgan, Ltd, UK).

From this data, a computer is used to draw a regression line for the effect of oxygen consumption on heart rate. The result is then applied to the data from the heart monitor, and the V_{O_2} level (representing energy expenditure) can then be calculated for the activities undertaken during the study period.

Our results for light exercise such as bicycling, walking, and jogging were in close agreement with those that Birrer and Daller presented in Table II.[1] Our measurement of energy expenditure during ice hockey played by one of us (GSM), however, was higher (10.0 METS, compared with the reported value of 7–8 METS [1 MET = 3.5 mL O_2/kg/min]). In addition, some skating activities such as "sprinting" produced energy expenditures of greater than 11.7 METS, compared with Birrer and Daller's reported value of 6–7 METS.

Our results could possibly be explained by differences in methods of measuring energy expenditure, but this is made less likely by our agreement with the data presented by Birrer and Daller on other forms of exercise (eg, cycling). It seems more likely that the data presented by Birrer and Daller on ice hockey were either an underestimate or did not take adequate account of variations in exercise intensity. Our data on ice skating and ice hockey indicate that there is a great deal of inter-individual variation in energy expenditure for a given exercise, and that the practitioner must take this into account when giving an "exercise prescription."

GREGG S. MEYER, MD
VERNON REYNOLDS, PhD
Department of Biological Anthropology
University of Oxford
58 Banbury Rd
Oxford, England 0X2 6QS

---

[1] Birrer RB, Daller JA: The exercise prescription. *NY State J Med* 1987; 87:106–112.

In reply. We would like to thank Drs Meyer and Reynolds for sharing their research data concerning energy expenditure during certain exercises. We concur with their conclusion that there is a significant amount of inter-individual variation in energy expenditure for a given exercise that must be incorporated into the exercise or physical activity prescription. The summary data provided in Table II in our article represent the synthesis of at least seven different studies. The methodologies, measurement techniques, and sample sizes varied significantly among the investigations, thus making some of the data (eg, hockey, certain occupations) less reliable. As more research data becomes available, these figures will be revised accordingly.

RICHARD B. BIRRER, MD
Chairman
Department of Family Medicine
Geisinger Medical Center
Danville, PA 17822

Associate Professor of
Family Medicine
State University of New York
Health Science Center
at Brooklyn

JOHN A. DALLER, MS
Department of Family Practice
State University of New York
Health Science Center
at Brooklyn
Brooklyn, NY 11203

House staff supervision and working hours

TO THE EDITOR: I am writing in appreciation of your excellent commentary in the *Journal* concerning the supervision of house staff and limitations on their working hours.[1]

I dearly hope that the "commendable initiative of reform" will reach fruition in the near future. Again, my compliments on a beautifully written and moving piece. You can be sure it will be widely distributed at my hospital.

EMANUEL E. GARCIA, MD
Resident in Psychiatry
The Institute of Pennsylvania Hospital
111 N 49th St
Philadelphia, PA 19139

---

[1] Imperato PJ: Supervising interns and residents and limiting their working hours. *NY State J Med* 1987; 87:425–427.

TO THE EDITOR: I read your commentary in the *Journal* on the supervision of interns and residents with great interest.[1] I have a tremendous fear that these regulations were issued too rapidly without sufficient thought being given to the practical matter of implementation. The committee should have included major program directors of residencies who are familiar with implementation issues.

Most attending physicians in orthopedic surgery are already putting in 50 to 60 hours a week, much of which is just to meet the costs of liability insurance (an issue which the state has not adequately addressed). Therefore, attending staff physicians really do not have a great deal of free time and often lack the stamina of younger house physicians. A problem also arises about when and how to schedule teaching conferences so that they do not interfere with patient care and operating room schedules and mandatory time off. At the same time one must provide a steady level of service.

What is going to suffer the most is health care in places like large public hospitals, which place the highest demands on the residents and provide a lower level of attending physician reimbursement per hour, at least in my specialty area. Surely this is not the desired effect.

I have suggested that the hospitals institute better support systems. There is no reason why residents at some hospitals (and sometimes the attendings) should have to transport their patients to the x-ray department or to the operating room. Blood drawing and intravenous teams should be available at all times. Individuals who show up in the middle of the night with problems that can wait until the next morning (city prisoners are particularly prone to do this) should be asked or even required to do so after they have been screened by an appropriately trained paramedic or a physician assistant.

Many of the stresses that house officers encounter at night are to a large extent due to unnecessary demands created by a lack of organization and support structure.

I understand what the Commissioner of Health of New York State is trying to accomplish. As far as the emergency room is concerned, his suggestions are excellent. As far as his suggestions for patient care responsibilities are concerned, I think they are impractical. The duties and schedules of these house officers should be the responsibility of trained, accredited educators.

STANLEY L. GORDON, MD
Chairman
Department of Orthopaedic Surgery
and Rehabilitation Medicine
State University of New York
Health Science Center at Brooklyn
450 Clarkson Ave
Brooklyn, NY 11203

---

1. Imperato PJ: Supervising interns and residents and limiting their working hours. *NY State J Med* 1987; 87:425-427.

Minilaparotomy: An approach to postpartum tubal sterilization

TO THE EDITOR: In 1971 Keith[1] reported his experiences with laparoscopic sterilization in the puerperium. The technique that he described does not seem to be the ideal approach. The bulk of the uterus and the thickening of the fallopian tubes make the procedure technically difficult.

At about this time, articles[2,3] began to appear in the literature evaluating minilaparotomy as an approach to interval sterilization. However, the use of the mini-incision immediately postpartum had been neglected.

We thought that because of the height of the uterine fundus, the fallopian tubes could be reached through an incision similar in position to and not much larger than that used for laparoscopy. This approach eliminates the danger of blind entry into the peritoneal cavity. The problems inherent in the use of the large trocar, as described by Keith, are also avoided.[1]

The subumbilical semilunar incision seemed to be the answer, affording the advantage of a small incision while still providing adequate visibility without increasing risk.

Case Series. One hundred eighty-four immediate postpartum sterilizations using the subumbilical incision are reviewed.

All laparotomies were performed by a group of three physicians at the Victory Memorial Hospital during the 11-year period between January 1, 1975, and December 31, 1986. The age of the patients ranged from 22 to 39 years. The median age was 28 years. Parity varied from two to seven. The majority of women were sterilized after the third delivery. The heaviest patient in the series weighed 230 lb.

The Technique. Care must be taken to carefully screen the candidates. A history of previous adnexal surgery may limit mobility and make this incision inadequate. All operations should be carried out within 48 hours after delivery. Waiting longer can result in sufficient uterine involution as to make exteriorization of the fallopian tubes difficult without the added risk of tearing the mesosalpinx.

General anesthesia with adequate relaxation, including bowel immobilization, is necessary. Endotracheal intubation is therefore a requirement in all cases.

With the patient in moderate Trendelenburg position, a 2-cm transverse subumbilical semilunar skin incision is made. The subcutaneous tissue is sharply dissected. The fascia is visualized and grasped with two Allis clamps; an incision is made between the clamps. The peritoneum is then visualized, tented, and entered by sharp dissection, taking care to avoid the underlying bowel.

The fundus of the uterus can immediately be visualized, and should be manually displaced upwards and to the right. The left tube is grasped with a long Babcock clamp. Care is taken not to apply excessive traction. The tube is then followed laterally to the fimbria, confirming the tube's identity.

Sterilization is now performed using the surgeon's method of choice. A Pomeroy ligation was used in all the patients reviewed here.

After the tubal stump has been checked for bleeding, it is gently replaced into the peritoneal cavity. The uterus is then manually displaced upward and to the left. The right tube is then visualized, identified, and ligated in a like manner. The peritoneum is closed with a purse string suture of 00 chromic catgut. The fascia are approximated with a continuous stitch of the same material, and the skin edges are approximated with 000 catgut, using the subcuticular technique.

Discussion. The purpose of this study was to evaluate the mini-incision sterilization method in the immediate puerperium, and to determine if this technique could be consistently performed without increasing the intraoperative or postoperative risk.

In 184 cases, there were no complications directly related to the incision. No immediate or delayed morbidity was noted, and there have been no deaths. There have been no postoperative umbilical hernias, and no pregnancies have occurred in the series.

Only two intraoperative complications occurred. In one case, the ligature slipped from the tubal stump. The stump was grasped without difficulty and was religated. In the other case, the mesosalpinx was torn; this accident was recognized immediately and the proper steps were taken. Neither woman experienced postoperative morbidity or a prolonged hospital stay.

Average blood loss was 10 cc, and the average operating time was 15 minutes. Postoperative pain was minimal, even during the first 24 hours, and a nonnarcotic analgesic was sufficient for pain relief. Discharge from the hospital occurred on day four, which was the normal day of postpartum discharge at the Victory Memorial Hospital.

It can be concluded that in selected cases, the subumbilical semilunar incision provides an ideal approach to female sterilization during the first 48 hours after uncomplicated vaginal delivery.

We feel that the technique described is safe and does not increase the average postpartum stay. The incision provides an adequate operative field and can be performed with minimal blood loss in about 15 minutes.

JOHN M. ESPOSITO, MD
ANIELL T. MASTELLONE, MD
GEORGE RUBINO, MD
Victory Memorial Hospital
9036 Seventh Ave
Brooklyn, NY 11228

---

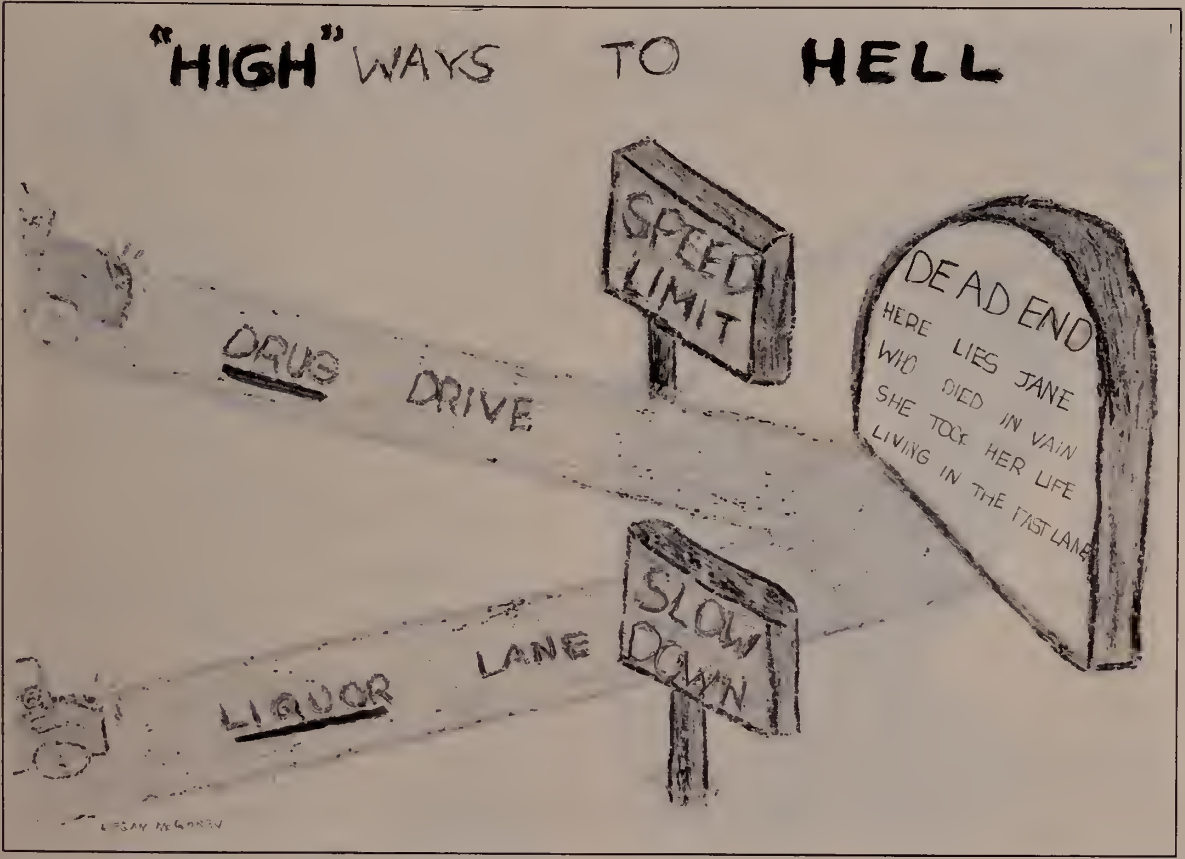
1. Keith L, Houser K, Cohen S, et al: Puerperal laparoscopy at Cook County Hospital. *J Reprod Med* 1971; 6:155-157.
2. Penfield AJ: Minilaparotomy for female sterilization. *Obstet Gynecol* 1979; 54:184-188.
3. Dassenaike GS, Saha A, McCann MF: Female sterilization via minilaparotomy. *J Reprod Med* 1976; 17:119-124.

Poster contest: The hazards of drug abuse

In 1986, the Child and Youth Committee of the Auxiliary to the Medical Society of the County of Queens (MSCQ) sponsored a poster contest for fourth grade through junior high school students in Queens County. The theme of the contest was "The Hazards of Drug Abuse." Over 200 submissions were received by the committee, and winners were chosen in early May. At an awards ceremony held at the headquarters of the MSCQ, prizes were awarded to one first-place winner, one second-place winner, two third-place winners and five fourth-place winners. Financial support for the project was provided in part by the MSCQ.

The Child and Youth Committee of the Auxiliary to the MSCQ, chaired by Mrs Ezra A. Wolff, had previously sponsored essay contests on the subject of drug abuse for school children from the fourth grade through junior high school. During the 14 years that the essay contests were held, over 5,000 essays were submitted. A related project of the Auxiliary, begun in 1985, was a bumper sticker program. To date several thousand stickers—"Child Abuse Is a Criminal Offense" and "Death Awaits Drunken Drivers and Their Victims"—have been distributed to the Community Relations Division of the New York City Police Department.

The four posters published here represent the selected first-, second-, and third-place winners of the 1986 contest.



Regan McGorry, 8th Grade, Sacred Heart School, First Prize

Marc Robinson, 9th Grade, Halsey Junior High School, Second Prize

Duane Adams, 7th Grade, Junior High School 157, Third Prize

Jonathan Geller, 8th Grade, Ryan Junior High School 216, Third Prize

Reprinted from the Summer 1987 issue of Epidemiology Notes *(Vol. 2, No. 5), published by the Division of Epidemiology, New York State Department of Health, Albany, NY.*

Yersiniosis

Yersiniosis is an acute enteric disease manifested by a wide range of clinical symptoms including diarrhea, enterocolitis, acute mesenteric lymphadinitis (which may mimic appendicitis), fever, vomiting, headache, and septicemia. Arthritis (Reiter syndrome), osteomyelitis, and erythema nodosum have also been associated with yersiniosis. The two bacterial species associated with the disease are *Yersinia enterocolitica* and *Y pseudotuberculosis*. Almost all reported cases are due to *Y enterocolitica*.

Yersiniosis was added to the reportable disease list for New York State on January 1, 1986. In 1986, a total of 87 cases were reported among upstate New York residents. Data on yersiniosis cases in New York City are incomplete. Most cases occur sporadically but clusters or outbreaks are not uncommon. Outbreaks described in the recent literature have been associated with contaminated water, chocolate milk, tofu, pets, and an infected food handler.

Clinical Aspects

Infection with *Yersinia* often results in asymptomatic or mild infection. In young children, infection may result in fever, abdominal pain, and diarrhea. In older children and adults, abdominal pain can be severe enough to mimic appendicitis. In one study,[1] of 181 children with gastroenteritis due to *Y enterocolitica*, symptoms were diarrhea (98%), fever (68%), abdominal pain (64%), and vomiting (38%). The mean duration of diarrhea was 14 days (range, 1–46 days), and 25% of the patients noted blood in the stools. Stools remained positive for four to 79 days (mean, 27 days) after resolution of symptoms. In adults, 2–3% may develop accompanying erythema nodosum or arthritis.[2] Other extraintestinal symptoms with yersiniosis include glomerulonephritis, hepatitis, uveitis, and hemolytic anemia.[3]

Diagnosis

The gastrointestinal symptoms associated with yersiniosis cannot be clinically differentiated from those associated with salmonellosis, shigellosis, or campylobacteriosis. The diagnosis is established on the basis of isolating the organism on appropriate culture media from stool, blood, or other body sites. Cold enrichment methods have proven particularly valuable in detecting this agent. Serologic diagnosis has not been demonstrated to be reliable. When stool cultures are negative for common bacterial enteric pathogens (*Salmonella*, *Shigella*, and *Campylobacter*), laboratories should routinely test for *Yersinia*.

Epidemiology

Y enterocolitica has been isolated from a variety of domestic and wild animals, water, milk, ice cream, and meat. However, only a few of the many serotypes have been shown to be pathogenic for humans. Those most often associated with human disease are serotypes 03, 08, and 09. Serotype 08 predominates in New York State, while serotype 03 is found most often in Canada. The reason for the geographic difference in serotypes is unknown.

A review of the 87 cases reported in upstate New York in 1986 revealed a seasonal peak in the summer months similar to campylobacteriosis and salmonellosis. The male-to-female ratio was equal. Twenty-nine percent of the cases occurred in children under age one as compared to 4% for campylobacteriosis, 1% for shigellosis, and 9% for salmonellosis. The predominance of reported infections in young children deserves further study.

While person-to-person spread has been documented, the majority of cases are presumed to be foodborne or waterborne. Prompt reporting and follow-up of cases may yield further information regarding the epidemiology of yersiniosis.

Treatment and Control

As most infections are mild and self-limiting, symptomatic treatment is usually adequate. With cases of septicemia or invasive disease, the antimicrobials of choice are aminoglycosides, TMP-SMX, and possibly certain third generation cephalosporins. The need for adequate handwashing should be emphasized for all patients. During the diarrheal phase of the illness, patients should refrain from food handling, patient care, and close contact with other small children.[4]

References

1. Marks MI, Pai CH, Lafleur L, et al: Yersinia enterocolitica gastroenteritis: A prospective study of clinical, bacteriological, and epidemiologic features. *J Pediatr* 1980; 96:26–31.
2. Vantrappen G, Ponette E, Geboes K, et al: Yersinia enteritis and enterocolitis: Gastroenterological aspects. *Gastroenterology* 1977; 72:220–227.
3. Bottone EJ: Yersinia enterocolitica: A panoramic view of a charismatic microorganism. *CRC Crit Rev Microbiol* 1977; 5:211–241.
4. Yersiniosis, in Benenson AS (ed): *Control of Communicable Diseases in Man*, ed 14. Washington, DC, American Public Health Association, 1985, pp 440–442.

Stomach and colorectal cancers in Puerto Rican-born residents of New York City

Studies of immigrant populations have contributed significantly to our understanding of the epidemiology of various cancers. Comparisons of site-specific cancer rates of immigrant populations in their native country and in their new country have provided much of the scientific evidence linking environmental factors (especially diet) to cancer risk.

If a disease is genetically predetermined, it is presumed that the risk in immigrant populations would remain at the levels of their native countries. For example, in a study of Polish-born emigrants to the United States, stomach cancer mortality rates in the United States reflected the high rates seen in native Poles. However, the traditionally low mortality rate for colon cancer in Poland was not observed in Polish emigrants to the US. The mortality rate for colon cancer in immigrant Poles increased to the level prevailing in US native whites.[1] In a study of Japanese emigrants to Hawaii, stomach cancer risk remained high in immigrants from districts with high risk but risk in offspring of immigrants decreased.[2]

A recent study[3] considered incidence and mortality data from the New York State Cancer Registry and the Cancer Control Program of the Puerto Rico Department of Health to compare stomach and colorectal cancer incidence and mortality rates in Puerto Rican-born residents of New York City (PR-NYC) with Puerto Ricans living in their native country (PR-PR).

Colorectal cancer incidence and mortality rates for PR-NYC residents for 1975–79 were about two times higher than comparable rates among those PR-PR residents and one-half to almost two-thirds higher than the rates for other whites in New York City. From 1958 to 1979, colon cancer mortality rates among PR-NYC residents increased dramatically (ie, 212% in males and 54% in females), whereas among PR-PR residents, rates in males increased only slightly and rates in females remained constant.

For stomach cancer, incidence rates for male and female PR-NYC were slightly but not significantly higher than those for male and female PR-PR residents. In contrast, stomach cancer mortality rates for PR-NYC were lower than those for PR-PR residents throughout the survey period, and mortality decreased substantially in all racial-ethnic groups in New York City and Puerto Rico from 1958 to 1979.

Results of this study indicate that the changes in colorectal and stomach cancer incidence and mortality in Puerto Rican-born residents after migration to New York City, although not as drastic as in immigrants from Japan and eastern Europe, were comparable to those observed for Hispanic emigrants to New Mexico and Los Angeles. The tendency of stomach and colon cancer rates in Puerto Rican emigrants to New York City to move toward levels seen in the overall New York City population adds to the body of evidence suggesting the importance of environmental factors in these diseases.

References

1. Staszewski J, Haenszel W: Cancer mortality among the Polish-born in the United States. *JNCI* 1965; 35:291–297.
2. Haenszel W, Kurihara M, Segi M, et al: Stomach cancer among Japanese in Hawaii. *JNCI* 1972; 49:969–988.
3. Warshauer ME, Silverman DT, Schottenfeld D, et al: Stomach and colorectal cancers in Puerto Rican-born residents of New York City. *JNCI* 1986; 76:591–595.

AIDS Intervention Management System

The October 1986 issue of *Epidemiology Notes* (vol 1, no. 2) included an article describing the designated AIDS center concept of the New York State Department of Health. The following contains a brief review and update on the AIDS centers, and presents new information about the AIDS Intervention Management System (AIMS).

On March 24, 1986, the department issued (through Department of Health Memorandum 86–32) a Request for Application (RFA) regarding AIDS center designation for hospitals. Specifically, the RFA required hospitals to provide care to all subcategories of AIDS patients in discrete units through a case management/continuum of care model. The RFA outlined 13 standards of care and practice that must be provided and/or assured as part of the facility's AIDS program: inpatient, outpatient, emergency room, home care, hospice/residential care, diagnostic and therapeutic radiological services, inservice education, infection control, quality assurance, clinical research, education programs, crisis intervention, and case management.

Through the AIDS care center program, the department proposes to increase access by persons with AIDS to essential health care and community resources so AIDS patients are able to maintain the quality of their lives in a home environment as long as possible. This program is not intended to relieve general hospitals (that are not designated AIDS centers) of their responsibility for the care and treatment of people with AIDS, nor is it intended to inhibit patients' freedom of choice in seeking access to care in facilities not designated as AIDS centers.

In response to the RFA, the department received 26 Certificate of Need applications. To date, one facility has received full AIDS care center designation (St Clare's Hospital, New York City); three have agreed to rate reimbursement schedules but are not yet fully designated (Albany Medical Center, University Hospital at Stony Brook, and St Luke's Roosevelt Hospital); and 15 others have received contingent approval letters.

In conjunction with the development of an AIDS care center program, New York State solicited competitive applications through an RFP process from qualified organizations for the design, implementation, and operation of an oversight system, the AIDS Intervention Management System (AIMS), both to review the performance of the comprehensive AIDS centers and to conduct a comparative review of AIDS patient management in nondesignated hospitals caring for AIDS patients. The organization selected, the New York Statewide Professional Standards Review Council, will ensure that appropriate standards of utilization review, quality assurance, and case management are established and met for patients receiving care under the designated center program. In addition, AIMS will function as a data gathering system and an information resource for the designated AIDS care centers.

The AIMS contractor, in pursuing the general goal of reviewing patient management and compliance with guidelines governing AIDS centers, will address the following specific objectives:

- implement an evaluation of case management and other care models developed by various care centers;
- establish and maintain a utilization review program for persons with AIDS/HIV disease according to the approved program for a designated center;

- develop and conduct a comparative analysis of care programs for patients with AIDS/HIV disease in facilities not designated as care centers;
- design protocols and procedures for assuring quality of care surveillance in a systemic fashion for all groups, including referrals to the Office of Health Systems Management's surveillance staff when appropriate;
- monitor the level, type and amount of services required by patients with AIDS/HIV disease, and identify gaps in service availability and/or accessibility;
- assess and evaluate the effectiveness of the center's discharge planning, and the viability and effectiveness of referral patterns for various levels of care and types of services;
- monitor the availability and accessibility of community and institutional services;
- conduct an ongoing overall evaluation of all centers.

For further information about the AIDS centers or about the AIDS Intervention Management System, contact Ira Feldman, AIMS Coordinator, at (518) 473-7665.

Hypertension and complications of labor/delivery

Hypertension during pregnancy is associated with an increased risk of complications during labor and delivery. For the purpose of this report, women were considered to be hypertensive if they are described as having hypertensive disease or preeclampsia as a complication of pregnancy. Eclamptic women are not included since this condition involves more severe symptoms than elevated blood pressure. Live birth data from 1984 through 1986 were pooled to form one hypertensive and one nonhypertensive population for the period. The number of women in the hypertensive group is 11,132; the nonhypertensive population is 377,187.

Table I shows the frequency and rate of overall complications of labor and delivery, as well as individual complications for hypertensive and nonhypertensive women. The confidence intervals shown in the table were calculated using the method described by Kleinman.[1] If the confidence interval for the difference in two rates does not contain zero, the rates can be said to be significantly different with 95% probability. Those rates with intervals that contain zero cannot be said to be significantly different. With the exception of placenta previa, all complications have significantly higher rates among hypertensive women than among nonhypertensive women.

Looking at individual complications, the largest difference involves hemorrhage, which is more than three times as prevalent among hypertensive women as among nonhypertensive women. Several complications are more than twice as common in the hypertensive group: premature separation of placenta, prolonged labor, cephalopelvic disproportion, contracted pelvis, uterine dysfunction, other dystocia, prolapse of cord, and fetal distress.

Certain additional complications, while less than twice as likely in the hypertensive group, were still significantly more prevalent than in the nonhypertensive group. Other

TABLE I. Rates of Complications of Labor and Delivery* 1984–1986

Complication	Hypertensive Cases	Hypertensive Rate	Nonhypertensive Cases	Nonhypertensive Rate	95% Confidence Interval for Difference in Rates Upper Limit	95% Confidence Interval for Difference in Rates Lower Limit
All complications	6,763	607.50	118,202	313.40	308.70	279.50
Premature separation of placenta	160	14:37	2,598	6.88	9.73	5.25
Placenta previa	59	5.30	1,613	4.27	2.40	−0.34 (NS)
Retained placenta	86	7.73	1,783	4.73	4.64	1.35
Premature rupture of membranes	566	50.84	13,642	36.17	18.90	10.43
Prolonged labor	454	40.78	7,343	19.46	25.10	17.54
Cephalopelvic disproportion	1,307	117.40	17,036	45.16	78.64	65.84
Contracted pelvis	118	10.60	1,460	3.87	8.65	4.81
Uterine dysfunction	290	26.10	3,270	8.99	20.40	14.38
Other dystocia	95	8.53	1,430	3.79	6.47	3.01
Hemorrhage (over 500cc)	261	23.44	2,763	7.32	18.98	13.26
Cord around neck	1,477	132.70	36,749	97.43	42.11	28.43
Prolapse of cord	67	6.02	1,123	2.98	4.49	1.59
Fetal distress	1,422	127.70	22,083	58.55	75.83	62.47
Breech presentation	497	44.65	11,075	29.36	19.25	11.33
Other abnormal presentation	189	16.98	3,380	8.96	10.46	5.58
Other	1,957	175.80	23,480	63.20	120.40	104.70

* Rate per 1,000 women in category.

abnormal presentation is 89% more likely among hypertensives, while retained placenta is 63% more common. Breech presentation shows a 52% increase in the hypertensive group, premature rupture of membranes is 40% more common, and cord around neck is 36% more likely.

While hypertension during pregnancy has long been associated with an increase in maternal and perinatal mortality, these data indicate that hypertension is also a significant risk factor for complications during labor and delivery.

The New York State Department of Health is exploring strategies to reach these high-risk women prior to conception. It is hoped that an interdisciplinary team of obstetrician and internist can jointly work with the patient to achieve blood pressure stabilization and the best possible cardiovascular, renal, and nutritional status in which to begin pregnancy. It would also be important to review the patient's medication regimen before pregnancy and to discontinue any medications that are teratogenic or to prescribe others with known efficacy during pregnancy.

References

1. Kleinman JC: Infant mortality. *Statistical Notes for Health Planners* 1976; (July) 2:1-12.

Injury control program

The Injury Control Program was formed within the Division of Epidemiology in December 1986 to provide a visible focus of injury control activities in epidemiology, preventive services, and education with an aim to reduce the morbidity and mortality from injuries in New York State. The program is organized into two units—the Surveillance and Epidemiology Unit and the Prevention Services Unit.

The Surveillance and Epidemiology Unit is responsible for surveillance of injury mortality and hospitalized injuries; development of community surveillance capability; and conduct of a Governor's Traffic Safety Committee study to evaluate the effectiveness of the New York State seat belt law in the Capital District. The unit also provides guidance and assessment of departmental and external injury control programs.

The Prevention Services Unit is responsible for implementation of statewide programs and development of community programs in local health units. Specific projects include overseeing the Poison Control Network (to fulfill the mandates of the Poison Control Network Act of 1986 to designate, oversee, and evaluate eight regional poison control centers); consultant services in child injury prevention and consumer product safety; and the Child Car Restraint Device Loaner Projects. The 38 loaner projects serve low income populations in 40 counties through funding from the Governor's Traffic Safety Committee. Proposals for an elderly home safety demonstration project in local health units have been solicited and applications are being reviewed.

A major function of the Injury Control Program is education and training. Activities include professional training workshops for local health units, development of public education materials, and development of an academic program within the new Graduate School of Public Health Sciences of the State University of New York at Albany. Additionally, an intradepartmental Injury Prevention Task Force was formed to coordinate the various injury-related activities located in other areas of the department, such as emergency medical services; children's camp safety; child abuse and domestic violence; the head injury program; the home safe home initiative; the healthy neighborhoods project, occupational health; and control of injuries in health care facilities.

The Injury Control Program will serve as a clearinghouse for injury control information in New York State and for the development of a statewide consortium of injury control researchers and practitioners.

For further information about the injury control program, write to Dr Susan Standfast, Injury Control Program, Division of Epidemiology, New York State Department of Health, Corning Tower Room 2202, Empire State Plaza, Albany, NY 12237.

CONTINUING MEDICAL EDUCATION. A PRIMER

Edited by Adrienne B. Rosof and William C. Felch, MD. 213 pp, illustrated. New York, Praeger Publishers, 1986. $29.95 (hardcover)

Adrienne Rosof and Dr Felch are to be congratulated for creating this much needed primer on such an important and difficult subject, continuing medical education. As one might suspect it is not easy to read and I would not recommend it for recreational purposes. However, this book is an invaluable reference source for those involved in the presentation of courses to update medical knowledge for practicing physicians.

I admire the organization of the topics and they are listed here for informational purposes: the arena; educational aspects; managing the CME office; other operational aspects; environmental factors; and new approaches.

The methods of meeting the requirements of accrediting agencies and the evaluation of courses have always been somewhat difficult to grasp. Both are covered in Parts II and V.

The contributors were well chosen, and the book should be included in the library of every director responsible for continuing medical education in hospitals, medical schools, specialty societies, and state associations.

Dr Felch's reputation and experience are well known and Adrienne Rosof's contributions will place her among those to be consulted on the methods and procedures so well presented in their book.

JULIUS E. STOLFI, MD
State University of New York
Health Science Center
at Brooklyn
Brooklyn, NY 11203

BEHAVIORAL COUNSELING IN MEDICINE. STRATEGIES FOR MODIFYING AT-RISK BEHAVIOR

By Michael L. Russell, PhD. 327 pp, illustrated. New York, Oxford University Press, 1986. $29.95 (hardcover)

Patients with chronic medical problems and long-standing self-destructive habits constitute a large part of any physician's practice. Physicians face frustration unless they are able to modify their patients' risk factors such as diet, smoking, lack of exercise, medication noncompliance, and stress. Michael L. Russell has written about just such an approach for physicians and other allied health personnel.

Russell advocates behavioral counseling rather than a more doctor-centered approach. No longer is it enough to diagnose those who do not change voluntary behaviors as "uninformed" or "unmotivated," for those assessments leave the physician with little else to offer. Rather, Russell suggests that physicians and patients collaborate to understand what sustains a particular behavior (no matter how it got started), select a method to reduce the undesirable behavior, and develop new alternatives. This requires that health care professionals consult and guide rather than prescribe or dictate. He acknowledges that this may be an awkward new role for many health professionals, so he tries to build skill and comfort in his readers by conceptualizing the interaction, telling us how to act and what to say.

The first half of the book provides a basic understanding of the behavioral counseling model. It blends basic behavior modification principles with a logical problem-solving model including goal specification, behavioral diagnosis, intervention, feedback, and revised intervention steps. Russell conceptualizes patient difficulty in changing habits in five areas: insufficient knowledge, rejection of medical diagnosis, rejection of medical prescription, lack of self-management skills, and intermittent self-debate due to conflict with other priorities. After a short chapter on basic interviewing skills, he expands on behavioral counseling techniques and skills to maintain long-term change.

Later chapters describe applications to specific habit change problems: nonadherence to medication regimens, adherence to therapeutic diets, weight control, physical activity, smoking cessation, and management of stress reactions. These habits make most physicians' "Top Six" list of unwanted patient behaviors. Russell outlines general strategies for approaching each problem, shows diaries, illustrates behavioral prescription pad assignments to patients, presents patient examples, and outlines interventions for the health care professional. The themes of assigning patients to record their own behavior, analyze records, and develop concrete, achievable goals recur in each chapter. The concluding chapter advises the reader in making a referral to a mental health professional when that is necessary.

Several chapters are very practical. "Patient's Weight Loss Program" is complete, detailed, and ready to use directly from the book. Likewise, the section on physical activity outlines specific target heart rates, a graded walking program, and strategies for solving common problems (such as "lack of time"). Throughout the book there are useful food, exercise, and smoking logs, diaries, stepped care approaches, and wisdom which comes from treating a large number of patients. The book treats other subjects unevenly, however. For example, the chapter on stress management is an overview and the clinician who reads it should not expect to know how to teach relaxation exercises to patients. The major problem is that the book will appeal to a fairly narrow audience consisting of those who are predisposed to collaborate with patients and who enjoy a counseling, consulting relationship. It will appeal to those who want to know step-by-step approaches to problem health behavior over several office visits. It will frustrate those who enjoy shorter interactions and see health behaviors as the responsibility of patients and not a physician concern. I have a hard time picturing any of the family practice residents I work with reading any but the most practical pages because the book is written with only a passing regard for typical physician learning style and practice. Psychologists will have trouble with the book because the presentation of research in the health behavior area is very uneven. For example, the author barely mentions the extensive work on noncompliance and does not seem aware of research on physician behaviors which enhance patients' smoking cessation efforts. Perhaps the book would appeal to beginning mental health professionals in a primary care setting, especially a prevention-oriented HMO.

Dr Russell is to be commended for addressing a major problem facing health care. Lifestyle behaviors influence health and disease, and mental health professionals know the techniques of behavior change. By and large physicians do not, but many people do not want to go to psychologists and are more likely to seek out health care professionals. This book attempts to bridge the two and to teach health care professionals what one psychologist knows. The bridge is needed, but I am not sure how much traffic there will be across this one.

JOHN GERDES, PHD
Geisinger Medical Center
Danville, PA 17822

THE LAST TABOO. MONEY AS SYMBOL AND REALITY IN PSYCHOTHERAPY AND PSYCHOANALYSIS

By David W. Krueger, MD. 262 pp, illustrated. New York, Brunner/Mazel, 1986. $27.50 (hardcover)

There has been a dearth of material on the subject of money and its meaning in a psychotherapeutic context. This book reviews a topic of significant importance through chapter presentations by 25 physicians and psychologists, many of whom are noted in the field of psychotherapy. The 24 chapters are presented in three general categories: the theoretical and symbolic meaning of money; money in the therapeutic-patient relationship; payment structures and management issues. The book's editor, David W. Krueger, MD, clinical associate professor of psychiatry at Baylor College of Medicine, writes about the symbolizations of money in his chapter, "Money Success and Success Phobia." Rosalea A. Schonbar, PhD, chairman of the Department of Clinical Psychology and Psycho-Educational Practice, Teachers College, Columbia University, discusses issues of some controversy regarding the policy of fee setting in her chapter, "The Fee as Focus of Transference and Countertransference in Treatment." G. A. Williston Di Bella, MD, Director of Psychiatric Residency Training and Medical Student Education at Cabrini Medical Center, New York, discusses the various stages of money mastery in her chapter, "Money Issues That Complicate Treatment." Paul Chodoff, MD, past-president of the American Academy of Psychoanalysis, writes about the effect of third party payment on the practice of psychotherapy. Sidney S. Goldenson, MD, Director of Mental Health Services of the Health Insurance Plan of Greater New York (HIP), writes about financial issues in a health maintenance organization (ie, how a third-party payment system affects transference and countertransference). In the sections on payment structure and management issues, Howard Gurevitz, MD, president of the California Psychiatric Association, writes about preferred provider organizations and psychiatric treatment. Arthur Reider, MD, Medical Director of the Center for Mental Health Retardation Services, Inc, Wallerton, Mass, writes about the clinical management of the nonpaying patient. Thomas G. Gutheil, MD, Director of Medical Student Training and Associate Professor of Psychiatry, Harvard Medical School, discusses various issues involved with fee setting in the initial stages of private practice, and John J. McGrath, MD, teaching analyst at Baltimore-District of Columbia Institute for Psychoanalysis, discusses fee setting in a changing health care economy.

In general the chapters in this book are put together in an interesting and informative fashion. Psychiatrists, or in fact any physician who has had to deal with financial issues affecting the physician-patient relationship, will find this book often stimulating in its approach to a difficult subject. Several of the chapters cover material that would be important to the beginning therapist going into private practice and also to the experienced clinician. Terms are clearly defined, and cases are presented with a significant degree of clarity. This book covers an important subject in a very informative style.

ALAN HERTZ, MD
State University of New York
Health Science Center
at Brooklyn
Brooklyn, NY 11203

SEXUAL AVERSION, SEXUAL PHOBIAS, AND PANIC DISORDER

By Helen Singer Kaplan, MD. 185 pages. New York, Brunner/Mazel Publishers, 1987. $25.00 (hardcover)

This is a timely and much needed original contribution to the field of sexual medicine. Coital fear, intense and irrational, is an ancient dilemma for some men and women and is the theme of this pioneering work. Diagnosed under the broad category of inhibited sexual desire, some aversions have responded to techniques of brief psychodynamic behavioral sex therapy, but many have been resistant to treatment.

Dr Kaplan, Professor of Psychiatry and Director of Cornell University Medical Center's Sexual Program, has taken a new look at treatment of resistant cases from among the several hundred seen there since 1970. Blended into a multimodal approach is the use of adequate doses of antipanic medication. The guest author for one chapter is Donald Klein, Professor of Psychiatry at Columbia, a foremost psychopharmacologist who has for a decade successfully pioneered the use of tricyclic antidepressants and other medications for patients suffering from anxiety, phobia, and panic states. With abundant, concise, and well-chosen case examples Dr Kaplan highlights categories of sexual panic disorders, phobias, and aversions, both primary, or lifelong, and secondary, noting that at times they may co-exist (eg, aversion plus panic), with or without a functional genital sexual symptom such as impotence, anorgasmia, etc. In each case she analyzes interpersonal and intrapsychic factors and, where relevant, the impact of important religious-cultural learning that may complicate the sexual problem. She lists over 20 types of sex phobias, describe the possible etiology of each, and provides full details of how each was clinically treated. She also endorses the American Psychiatric Association's inclusion of sexual aversion disorder (302.79) in the revised edition of *Diagnostic Statistical Manual III* (DSM III-R). This disorder is defined as a persistent or recurrent extreme discomfort with or avoidance of all, or almost all, genital sexual contact with a sexual partner.

With care and sensitivity Dr Kaplan points out to afflicted patients that, just as some persons have diabetes, others have a low threshold of tolerance for anxiety. The latter is a critical protective physiological alarm system whose goal is survival from perceived danger. She then includes an explanation of the special protective mother-child alarm system that may trigger reciprocal separation anxiety. This is a core issue which the author hypothesizes is present in many sexual aversions, anxieties, and panic states. The early separation fears have not reached an adult "all clear" state due to the individual's low biological threshold for anxiety, much like a security alarm system that goes off spontaneously, needlessly distressing the occupant. Appropriate medication reduces the anxiety-panic sufficiently to allow therapeutic growth and acceptance of the patient's natural sexual function. This is sufficiently self-reinforcing to permit discontinuation of medication after several months and concurs with work in other sex programs.

The integrated Cornell approach to treatment first consists of careful history-taking and diagnostic trial of sensate focus, nondemanding exercises to recognize resistant cases and to evaluate them for the degree of anxiety, for specific aspects of sexual fantasy or exchange that are discordant with the patient's value system, and also to look for early and current separation anxiety. When these are found, reassurance is given, sex therapy techniques are modified and slowed, and there is judicious introduction of antipanic medications, which the author reports have given new hope and help for recalcitrant cases. The author stresses the importance of therapist awareness of the patient's unresolved psychodynamic issues, but reminds the clinician to focus on the treatment goal of improving sexual functioning as well as the couple's relationship, and not to dwell on unrelated peripheral issues that could then distract and prevent sexual healing. When partner anxiety is aroused with relationship changes during treatment, similar use of psychoanalgesic medication is emphatically offered to the previously nonsymptomatic partner, often with positive results which can serve as a useful bypass when there is resistance to change.

This book is an important contribution to the medical literature and deserves to be read by every practicing gynecologist, primary care physician, psychiatrist, psychoanalyst, and sex therapist in this country and abroad.

DOMEENA C. RENSHAW, MD
Loyola University Medical Center
Maywood, IL 60153

PHYSICIAN ASSISTANTS: PRESENT AND FUTURE. MODELS OF UTILIZATION

Edited by Sarah F. Zarbock, PA-C, Kenneth Harbert, PA-C. 140 pages, illustrated. New York, Praeger Publishers, 1986. $35.00 (hardcover)

In this book, Sarah F. Zarbock, PA-C, and Kenneth Harbert, PA-C, have edited essays that provide answers to commonly asked questions: What is a physician assistant (PA)? How did the profession come into being? Where do physician assistants work? What are their responsibilities? What are their potentials for the future?

The essays are written by authors who are known for their contributions to an emerging profession. Among them are E. Harvey Estes, Jr, MD, Chairman of the Department of Family Medicine at Duke University, where the first PA program emerged under the guidance of Dr Eugene Stead; Richard G. Rosen, MD, Vice-Chairman of the Department of Surgery, Montefiore Hospital and Medical Center in New York, under whose guidance the postgraduate surgical residency program for PAs currently operates; and Rein Tideiksaar, Coordinator of the Department of Geriatrics, Mt Sinai Medical Center, New York, who besides holding a PhD degree is a physician assistant. These and other authors provide much information that will be useful for any hospital or clinic administrator, private practitioner, or personnel director interested in obtaining grassroots information about this profession.

Many examples of job settings are described, such as community and tertiary care hospitals, long term care facilities, industrial health settings, and HMOs. What is most helpful is the breakdown of responsibilities and duties for most of the job assignments, with examples of evaluation formats, patient satisfaction surveys, and surveys dealing with PA productivity and the length of patient visits. Institutional bylaws are also described, with qualifications for employment as a comparative guide for future employers.

Interpersonal relationships are a factor that must be considered when employing a PA. Many authors briefly comment on the subject. However, Barbara Seebold, RN, Vice President of Nursing at Geisinger Medical Center in Danville, Penn, devotes a chapter to the PA/nursing relationship. The initial pitfalls of the relationship are discussed and six points of advice are provided to help ease the transition period when a PA is added to the staff.

The book is divided into two parts, with the first providing an overview of the profession, models of utilization, medical marketing, and nonclinical contingencies to effect change. Part Two covers experiences in different care settings. The writing style varies because of multiple authorship. Better editing would have been helpful in eliminating the repetitive history of the profession and such statements as "My talk will focus on . . . ." On the other hand, most chapters begin at mutual starting points, then expand into the individual topics covered.

Other books written on this subject deal with the specific subtopic of cost effectiveness and patient acceptance, and frequently group PAs into the category of physician extenders. Zarbock and Harbert provide up-to-date information that covers many facets of the PA profession, but the most useful part is the listing of duties and responsibilities which can be used by institutions to meet their requirements.

The answers to many questions are still being sought for this young profession. Through this publication a bridge has been constructed that will provide answers to many of these questions. This book is a must for the reference libraries of those concerned with cost containment, those contemplating hiring physician assistants, and those with physician assistants on their staffs.

ANTHONY MIFSUD, R-PAC
State University of New York
Health Science Center
at Brooklyn
Brooklyn, NY 11203

THE PRACTICING PHYSICIAN'S APPROACH TO HEADACHE

Fourth Edition. Edited by Seymour Diamond, MD, and Donald J. Dalessio, MD. 240 pp, illustrated. Baltimore, Md, Williams and Wilkins, 1986. $38.50 (hardcover)

This is a practical management manual for one of the commonest and often most difficult clinical problems, namely, pain in the head, including facial pain. Coming four years after the previous edition, this is the most extensive revision to date, increasing its size by 64 pages. Ten contributors with expertise in related fields have joined the two editors for the first time, and they are jointly responsible for eight of the 19 chapters. Readers are assured that they are all clinical practitioners with experience in the field. The editors and some of the contributors are also well-established research workers on the subject.

Following a brief outline of the classification and mechanisms of headache, history taking and the physical examination are described. Pictorial profiles of the common varieties of headaches, initially designed by Dr Graham, effectively summarize the salient features of each type. A table of differential diagnosis based on the temporal profile of headache (acute, recurrent, and chronic) should have been included in this section.

The chapter on ancillary tests will produce controversy and needs to be revised. With the ready availability of computed tomographic (CT) scans, routine skull x-rays are difficult to justify. They are not cost effective compared to computed tomography, only occasionally provide clinically useful information, and are now reserved for specific indications. The same could be said for EEG and radioisotope brain scans, and echoencephalograms are hardly ever used now by adult neurologists. The CT scan has replaced these earlier techniques as the prime test to be performed when the cause of the headache is not readily apparent, or the pain is persistent and resists treatment. The section has been updated by an illustrated description of magnetic resonance imaging (MRI).

The common varieties of headache are described in the ensuing chapters, with good clinical descriptions and practical guides to treatment. There are new chapters on headache associated with cerebrovascular, eye, ear, and sinus diseases, as well as a useful one on post-traumatic headache. The book concludes with sections describing headache syndromes in children, psychological aspects, biofeedback and acupuncture techniques, and the organization of a headache clinic. A section of color plates by Dr Frank Netter has been included at the end of the book as it was in the previous two editions. They are very simplistic and do not add to the educational value of the book.

The clinical descriptions are sufficient for the medical practitioner, and the sections on management give detailed instructions, with dosages of recommended drugs. The common side effects and precautions in their use are also mentioned. A balanced description of the use of calcium channel blocking agents attests to the updating of this edition. However, the chapter on migraine lacks a final synthesis outlining the treatment, both abortive and prophylactic, with the authors' preferred sequence in the use of medications.

This is a very useful practical manual which achieves its aim of guiding all physicians who deal with headache patients. There are many larger and more detailed texts on headache which cover the basic science aspects in detail, but none as helpful to the practicing physician.

M. SOMASUNDARAM, MD
State University of New York
Health Science Center
at Booklyn
Brooklyn, NY 11203

THREE FORMS OF SUDDEN DEATH

By Dr F. Gonzalez-Crussi. 207 pages. New York, Harper & Row, 1986. $15.95 (hardcover)

Those who selected a career in medicine imbued with a sense of wonder at things biological sometimes find themselves disappointed. The pace and pressures of medical practice today often leave little time to reflect on the fantastic plasticity of the human being in health and disease. Common enough among burdened medical students and residents, this sense of disappointment strikes established physicians as well in this litigious and regulation-filled period. This disappointment, quelled at times by observation of a classic physical finding or a particularly prompt recovery, is not just a longing for simpler days. Rather, it is a hunger for one of the most fundamental satisfactions of the healing arts.

In this, his second collection of essays, F. Gonzalez-Crussi offers as practical a remedy as we are likely to find short of instituting a 36-hour day. These ten essays, each running 20 pages or so, cover a vast array of subjects. "On Aging," for example, considers not only current concepts of the effects of bodily wear and tear, but also syndromes of premature aging and the limited use of philosophy to console us in the face of our ultimate senescence. The essay that lends its title to the entire volume enumerates the several deadly properties peculiar to the beef hot dog. And in "The Anorectum" we learn of a man of singular talent whose ability to produce an operatic range of "posteriorly generated sounds" packed Parisian theatres early in this century.

Dr Gonzalez-Crussi, a professor of pathology at Northwestern University, extracts his lessons from rich and varied sources. In his piece on the female breast, he reminds his reader of the story of Saint Agatha—a Sicilian martyr punished in the third century AD by amputation of the breasts—to shed light on his own experience with a young woman who suffered from infected breast implants. Elsewhere, he cites with equal facility Greek philosophy (especially the Stoics), contemporary European novelists, and ancient Chinese texts, all with a pathologist's eye for the value of deviance in understanding the normal. For the curious and the inspired, references are amply supplied in 15 pages of scholarly notes.

Stylistically, these essays often reflect their own diverse origins. Apparently clashing images, as when the author describes the inclusion of specific insect fare on "the OK list" of edibles sent down to Moses, seem intended to give the reader pause. Similarly, the use of uncommon terms—horripilation and caducity, coruscating and perspicuous, to name but a few—suggest that this book be read within sight of a heavy dictionary. The trips back and forth will allow time for worthwhile reflection. Overall, the style is well-studied, delicate, deliberate, and precise. The reading is not always easy, but for anyone with an ear for irony and an eye for the miraculous, it is surely profitable.

Truly gifted writers, it has been said, leave us not just their works, but also a way of looking at the world. Through his reflections of the grandeur and misery of the body, Dr Gonzalez-Crussi presents the follies and the perceptiveness of those who toiled in other times. He thereby causes us to examine ourselves and our current tasks. There is strong medicine indeed in that introspection.

ABRAM E. KIRSCHENBAUM
Medical Student
New York University School
of Medicine
New York, NY 10016

ERRATA

The review of *The International Textbook of Cardiology* which appeared in the July 1987 issue of the *Journal* should have noted the inclusion of a chapter on antibodies, not antibiotics, in cardiology.

The review of *Special Care. Medical Decisions at the Beginning of Life* in the September 1987 issue incorrectly identified the reviewer as an MD. Dr Self holds a PhD degree.

BOOKS RECEIVED

AIDS

Sex, Drugs and AIDS. By Oralee Wachter. 76 pp, illustrated. New York, Bantam Books, 1987. $3.95 (paperback)

The Essential AIDS Fact Book. What You Need to Know to Protect Yourself, Your Family, All Your Loved Ones. By Paul Harding Douglas and Laura Pinsky. 63 pp. New York, Simon & Schuster, Inc, 1987. $3.95 (paperback)

Questions and Answers on AIDS. By Lyn Robert Frumkin, MD, and John Martin Leonard, MD. 208 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $19.95 (paperback)

AIDS: Public Policy Dimensions. 308 pp. New York, United Hospital Fund, 1987. $32.50 (paperback)

EMPORIATRIC MEDICINE

Foreign Travel and Immunization Guide. 12th Edition. 86 pp. Oradell, NJ, Medical Economics Books, 1987. $12.50 (paperback)

FAMILY PRACTICE

Urban Family Medicine. Edited by Richard B. Birrer, MD. 287 pp, illustrated. New York, Springer-Verlag New York, Inc, 1987. $65.00 (hardcover)

GERIATRICS

Geriatric Medicine Annual 1987. Edited by Richard J. Ham, MD. 284 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $35.95 (hardcover)

Reality Orientation for the Elderly. Third Edition. By Sylvester Kohut, Jr, PHD, Jeraldine J. Kohut, RN, Joseph J. Fleishman, PHD. 160 pp. Oradell, NJ, Medical Economics Books, 1987. $18.95 (paperback)

Disturbed Behavior in the Elderly. Edited by A. G. Awad, MB, Henry Durost, MD, H. M. Rosemary Meier, MB, and W. O. McCormick, MB. 189 pp. Elmsford, NY, Pergamon Books, Inc, 1987. $40.00 (hardcover)

INTERNAL MEDICINE

Oxford Textbook of Medicine. Second Edition. Volume I and Volume II. Edited by D. J. Weatherall, J. G. G. Ledingham, and D. A. Warrell. New York, Oxford University Press, 1987. $135.00 (2 vols, hardcover)

MEDICAL EDUCATION

Continuing Medical Education. A Primer. Edited by Adrienne B. Rosof and William C. Felch, MD. 213 pp, illustrated. New York, Praeger Publishers, 1986. $29.95 (hardcover)

MISCELLANEOUS

Life-Death Decisions in Health Care. By Lesley F. Degner, PHD, and Janet I. Beaton, PHD. 159 pp. New York, Hemisphere Publishing Corp, 1987. $39.95 (hardcover)

Our Sciences Ruled by Human Prejudice. Humanly Necessary Causal Blindness Persisting Even in Sciences. By D. G. Garan, PHD. 420 pp. New York, Philosophical Library, 1987. $29.95 (hardcover)

Reverse and Prevent High Blood Pressure With the K Factor. By Richard D. Moore, MD, and George D. Webb, PHD. 431 pp, illustrated. New York, Simon & Schuster, Inc, 1986. $4.95 (paperback)

Biological Materials: Structure, Mechanical Properties, and Modeling of Soft Tissues. By Frederick H. Silver, PHD. 228 pp, illustrated. New York, New York University Press, 1987. $42.50 (hardcover)

Every Woman's Guide to Breast Cancer. Prevention, Treatment, Recovery. By Vicki L. Seltzer, MD. 209 pp, illustrated. New York, Viking Penguin, Inc, 1987. $17.95 (hardcover)

Cardiovascular Disease and Behavior. Edited by Jeffrey W. Elias, PHD, and Philip Howard Marshall. 196 pp, illustrated. New York, Hemisphere Publishing Corp, 1987. $32.00 (hardcover)

Webster's New World Medical Word Finder. By George Willeford, Jr, MD. 443 pp. New York, Prentice-Hall Inc, 1987. $11.95 (paperback)

In Africa with Schweitzer. By Dr Edgar Berman. 308 pp, illustrated. Far Hills, NJ, New Horizon Press, 1986. $21.95 (hardcover)

NEUROLOGY

Neurology: Problems in Primary Care. By James J. Bernat, MD, and Frederick M. Vincent, MD. 645 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $39.95 (paperback)

Controversies and Clinical Variants of Migraine. Edited by Joel R. Saper, MD. 127 pp. Elmsford, NY, Pergamon Books Inc, 1987. $29.50 (hardcover)

A Guide to the Understanding of Alzheimer's Disease and Related Disorders. Edited by Anthony F. Jorm. 158 pp, illustrated. New York, New York University Press, 1987. $32.00 (hardcover)

NUTRITION

The Role of Drug Treatments for Eating Disorders. Edited by Paul E. Garfield, MD, and David M. Garner, PHD. 192 pp, illustrated. New York, Brunner/Mazel Publishers, 1987. $20.00 (hardcover)

OBSTETRICS AND GYNECOLOGY

Miscarriage. Sharing the Grief, Facing the Pain, Healing the Wounds. By Walter Williamson. 148 pp. New York, Walker and Co, 1987. $14.95 (hardcover)

Hematologic Problems in Pregnancy. Edited by David Z. Kitay, MD. 420 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $39.95 (softcover)

Protocols for High-Risk Pregnancies. Second Edition. Edited by John T. Queenan, MD, John G. Hobbins, MD. 449 pp, illustrated. Oradell, NJ, Medical Economics Books, 1987. $29.95 (paperback)

Foundations of In Vitro Fertilization. Reproductive Health Technology. Edited by Christopher M. Fredericks, John D. Paulson, and Alan H. DeCherney. 383 pp, illustrated. New York, Hemisphere Publishing Corp, 1987. $64.00 (hardcover)

OCCUPATIONAL MEDICINE

Occupational Pulmonary Disease. Edited by Linda Rosenstock, MD. 215 pp, illustrated. Philadelphia, Hanley & Belfus, Inc, 1987. $28.00 (hardcover)

Health Problems of Health Care Workers. Edited by Edward A. Emmett, MB. 225 pp, illustrated. Philadelphia, Hanley & Belfus Inc, 1987. $28.00 (hardcover)

Adolescent Suicidal Behavior. By David K. Curran, PHD. 208 pp. New York, Hemisphere Publishing Corp, 1987. $39.95 (clothcover)

REHABILITATION MEDICINE

A Primer on Management for Rehabilitation Medicine. Physical Medicine and Rehabilitation. Edited by F. Patrick Maloney, MD. 338 pp, illustrated. Philadelphia, Hanley & Belfus Inc, 1987. $26.00 (hardcover)

SUBSTANCE ABUSE

Cocaine Abuse. New Directions in Treatment and Research. Edited by Henry I. Spitz, MD, and Jeffrey S. Rosecan, MD. 352 pp, illustrated. New York, Brunner/Mazel Publishers, 1987. $35.00 (hardcover)

SURGERY

Surgical Lasers: A Clinical Guide. Edited by Terry A. Fuller, PHD. 237 pp, illustrated. New York, Macmillan Publishing Co, 1987. $75.00 (hardcover)

AMA reports continued decline in US medical school enrollments

The American Medical Association's (AMA) Office of Medical Education Information Analysis reported in the August 28 issue of *JAMA* that first-time enrollment in US medical schools decreased in 1986–1987, continuing a downward trend that began in the 1982–1983 school year. Total enrollment declined for the fourth consecutive year. The report documents the continued increase in the number of women who enroll in and graduate from medical school.

Applications for entry into US medical schools numbered 31,323 in 1986–1987, down 1,570 applications from the previous year. Of these 31,323 applicants, 17,092 were accepted by at least one school.

Of the total population of US medical schools during the 1986–1987 year, about 2.7% of the students were not US citizens. Foreign medical school graduates accounted for about 16% of residents in US training programs in 1986–1987.

At issue: The role of angioplasty in acute myocardial infarction

Balloon angioplasty has become popular in the 1980s as a means of opening stenotic vessels following acute myocardial infarction. Recent reports have suggested that angioplasty produces greater vessel patency than intracoronary streptokinase therapy in patients who present within 12 hours of experiencing symptoms associated with acute infarction. In a recent article in the *New England Journal of Medicine* (1987; 317:581–588), the Thrombolysis and Angioplasty in Myocardial Infarction (TAMI) study group gauged the effectiveness of balloon angioplasty following thrombolytic therapy to establish reperfusion. Commenting on the group's findings in an accompanying editorial, Dr Thomas Ryan (Boston University) concluded that the TAMI investigators may "have provided the first indication that as treatment for acute myocardial infarction, the high-flying balloon of angioplasty is losing some of its air."

The TAMI group documented that following thrombolytic therapy with tissue plasminogen activator, immediate angioplasty performed at the time of hospital admission appeared to be of no benefit in limiting the residual stenosis or lowering the rates of subsequent reocclusion. Elective angioplasty, performed prior to hospital discharge, yielded a slightly higher success rate in the treatment of acute infarction. In addition, 14% of the patients undergoing elective angioplasty demonstrated continued regression of the lesion to the status of a minor lesion. Dr Ryan cited the bleeding complications frequently associated with immediate angioplasty as another argument against the procedure, provided that thrombolytic therapy is successful.

The author takes a wait-and-see attitude regarding the future role of angioplasty in the treatment of patients with acute myocardial infarction, stressing the need to evaluate data from ongoing studies. "For the moment," he concludes, "it would seem prudent to administer thrombolytic therapy early, especially in young patients and those with anterior infarctions; watch closely; and move to invasive procedures at the first clinical indication."

New drug joins the fight against cholesterol

In September, the US Food and Drug Administration (FDA) approved the prescription drug lovastatin (trade name Mevacor®) "as an adjunct to diet for the reduction of elevated total and LDL cholesterol levels in patients with primary hypercholesterolemia (Types IIa and IIb), when the response to diet and other nonpharmacological measures alone has been inadequate." The FDA action followed studies that documented lovastatin's ability to reduce blood levels of certain forms of cholesterol by up to 39%.

There is little doubt in the medical community that a high blood cholesterol level (above 220 mg/dL) can be associated with atherosclerosis, a leading cause of heart attacks. Recent studies indicate that lowering one's cholesterol level to about 200 mg/dL can reduce the risk of atherosclerosis (*Harvard Med Sch Health Let* 1987; 12:1–4). Initial efforts to reduce a patient's cholesterol level should include a restrictive diet and regular exercise. If these measures prove ineffective, various forms of drug therapy are available, although most carry the burden of adverse side effects.

Currently accepted drug treatments to reduce cholesterol levels include niacin, or vitamin B3, in dosages of 3–6 grams/day. Niacin can reduce the level of low-density lipoprotein (LDL)—the harmful form of cholesterol—and can stimulate an increase in the level of high-density lipoprotein (HDL), the more desirable form. Cholestyramine and colestipol are synthetic resins used to bind bile acids, which are derivatives of cholesterol. The drug-bile acid compound then passes out of the body in the feces, thus lowering blood levels of cholesterol by as much as 20–35%.

Lovastatin is the first of a new class of cholesterol-lowering drugs that interfere with the synthesis of cholesterol in the body. Called HMG-CoA reductase inhibitors, these agents act in the liver to block the enzyme 3-hydroxy-3-methylglutaryl-coenzymeA (HMG-CoA) reductase, which converts HMG-CoA to mevalonate—the rate-limiting step in cholesterol synthesis. By inhibiting cholesterol synthesis, lovastatin can lower cholesterol levels by 30–40%. However, since the liver produces only 70% of the body's total cholesterol, lovastatin therapy can only be effective as an adjunct to a low-fat, low-cholesterol diet.

In clinical trials, patient tolerance of lovastatin was good, with fewer than 1% of patients discontinuing the studies because of drug-related adverse effects. Patient reports of side effects included rash, gastrointestinal effects, and headache. In addition, lovastatin demonstrated no clinically significant adverse interactions with commonly prescribed drugs such as beta blockers, diuretics, cholestyramine, calcium channel blockers, digoxin, and warfarin.

Merck, Sharp, and Dohme (West Point, Penn), manufacturers of lovastatin, do recommend certain precaution-

---

NEWS BRIEFS are compiled and written by Vicki Glaser.

ary actions to accompany prescription of the drug. Physicians should perform liver function tests every four to six weeks during the first 15 months of therapy, and periodically thereafter. About 2% of patients demonstrated a reversible increase in liver enzyme levels which required discontinuation of drug treatment. Lovastatin should not be used in patients with active liver disease or who have elevated levels of serum transaminase. The manufacturer also recommends ophthalmic observations with a slit lamp shortly after initiation of lovastatin therapy and once a year thereafter. This action is based on the finding of a high prevalence of baseline lens opacities in patients participating in the clinical trials and the appearance of new opacities during the study. As yet, however, researchers have reported no cause-and-effect relationship between the drug and the ophthalmic findings. Furthermore, lovastatin should not be prescribed during pregnancy and lactation, and the drug should be avoided by women during childbearing years, unless pregnancy is highly unlikely.

The dosing range for lovastatin is 20–80 mg/day, starting with a dose of 20 mg/day taken with the evening meal. Doses should then be adjusted according to the patient's response. When administered together, lovastatin and cholestyramine appear to produce additive cholesterol-lowering effects. The long-term effectiveness and adverse effects of lovastatin, as well as its effects on cardiovascular morbidity and mortality, will only become known after years of widespread use of this new drug.

How safe and effective are generic drugs?

Generic drugs are no longer considered to be second-rate products used by a small minority of consumers. Today, generic drugs are believed to offer a safe, effective, and less costly alternative to brand-name prescription drugs. Their popularity and availability are growing, with more than 1,000 generic drug products on the market in 1986, including generic forms of most of the ten major prescription drugs.

Several factors have led to the rapid increase in the number of generic drugs on the market in recent years. First and foremost was the 1984 Drug Price Competition and Patent Term Restoration Act, which enabled the US Food and Drug Administration (FDA) to expedite the approval of generic versions of brand-name drugs. The law eliminated the need for safety and efficacy tests—which would have been performed on the original, brand-name drug—provided the generic products pass bioequivalence tests. These tests determine whether the generic drug produces the same therapeutic effects as the original drug when given according to the conditions detailed in the labeling.

Responding to the need of physicians for more information about generic drug products, Stuart Nightingale, MD, and James Morrison of the FDA explained the process of FDA review and approval of generic drugs, specifically addressing the issue of therapeutic equivalence, in a recent issue of *JAMA* (1987; 258:1200–1204). The authors concluded that, although a generic drug that is therapeutically equivalent to a brand-name counterpart may not be appropriate for use by a given patient in certain circumstances, "the FDA believes that health professionals can substitute drug products evaluated as therapeutically equivalent with the full expectation that patients will receive products of equal safety and effectiveness."

The authors base this statement on the FDA's rigorous review and approval process for brand-name drugs, and the fact that the FDA maintains the same manufacturing and quality controls for generic versions of prescription products. As with brand-name products, the FDA randomly samples and tests generic drugs and periodically inspects the manufacturing operations of generic drug firms.

To obtain market approval, a generic drug must contain the same active ingredient(s) as the original product and must be identical in strength, dosage form, and route of administration. The generic drug can differ in the following characteristics: color, flavor, shape, packaging, inactive ingredients, and expiration date, note Nightingale and Morrison. Therefore, physicians should be aware of possible allergic reactions to additives such as coloring agents or preservatives.

To be considered bioequivalent, the generic version of a drug must produce equivalent therapeutic effects with no greater risk of adverse effects. Investigators compare the extent of absorption and the rate of absorption of the active ingredient(s) and/or its metabolites for generic and corresponding brand-name products. "Generic versions of pioneer drug products must be bioequivalent to a degree, calculated statistically," note the authors. According to the authors, "there is as yet no documented evidence of bioinequivalence involving a generic drug product approved by the FDA and evaluated as bioequivalent."

However, in another report in the September 4, 1987, issue of *JAMA*, Wyllie et al document the case of an epileptic patient whose frequency of seizures increased on two occasions when her medication was changed from primidone to generic primidone. In this patient, the two forms of primidone were not absorbed at the same rate and to the same extent.

Based on this experience, Wyllie et al concluded that "approval by the FDA does not guarantee bioequivalence." They emphasize that bioequivalence testing can "reduce, but not eliminate, the risk of inappropriate substitution for a specific individual." In their report, Nightingale and Morrison observe that the field of bioequivalence testing based on pharmacokinetic principles is in its infancy, with continued improvements in test design and evaluative methods anticipated.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Dominic S. Ariola, MD, Bayside. Died August 12, 1987; age 76. Dr Ariola was a 1935 graduate of Facolta di Medicina e Chirurgia dell'Universita di Roma, Rome, Italy. He was a member of the American Society of Abdominal Surgeons, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

John Arthur Bannon, MD, Glens Falls. Died July 31, 1987; age 75. He was a 1936 graduate of the University of Vermont College of Medicine, Burlington. Dr Bannon was a member of the Academy of Medicine, the American Academy of Family Practice, the Medical Society of the County of Warren, and the Medical Society of the State of New York.

Virgil Henry F. Boeck, MD, Buffalo. Died August 12, 1987; age 79. He was a 1931 graduate of State University of New York at Buffalo School of Medicine, Buffalo. Dr Boeck was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the Academy of Medicine, the New York State Society of Internal Medicine, the American Geriatrics Society, the Pan American Medical Association, the American Society of Internal Medicine, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Gustav A. Lara, MD, Mattituck. Died July 8, 1987; age 46. Dr Lara was a 1969 graduate of Facultad de Medicina de la Universidad de Zaragoza, Zaragoza, Spain. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Sandor Lorand, MD, Long Beach. Died July 30, 1987; age 95. Dr Lorand was a 1920 graduate of Lekarske Fakulta Univerzita Komenskeho, Bratislava, Czechoslovakia. Dr Lorand was a Fellow of the American College of Physicians, a Diplomate of the American Board of Psychiatry and Neurology, and a member of the Academy of Medicine, the New York Neurological Society, the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Theodore H. Neidengard, MD, New York. Died August 17, 1987; age 48. He was a 1968 graduate of West Virginia University School of Medicine, Morgantown. Dr Neidengard was a member of the American Medical Society for Alcoholism, the New York County Medical Society, and the Medical Society of the State of New York.

Daniel James O'Connor, MD, Lake Mary, Florida. Died August 3, 1987; age 72. Dr O'Connor was a 1940 graduate of the State University of New York Health Science Center at Brooklyn. He was a Qualified Fellow of the International College of Surgeons and a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Hans Oppenheimer, MD, Forest Hills. Died August 9, 1987; age 78. Dr Oppenheimer was a 1933 graduate of Medizinische Fakultaet der Johann-Wolfgang-Goethe Universitaet, Frankfurt-Am-Main, Hessen, Germany. He was a Diplomate of the American Board of Psychiatry and Neurology and a member of the American Psychiatric Association, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Arthur Rubin, MD, Palm Beach, Florida. Died August 9, 1987; age 76. Dr Rubin held Registrable Qualification Granted by the Scottish Conjoint Board in 1939. He was a Fellow of the American Academy of Family Practice and a member of the American Geriatrics Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

William Doughty Scott, MD, Massapequa. Died December 3, 1986; age 71. Dr Scott was a 1943 graduate of Hahnemann Medical College of Philadelphia, Philadelphia. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Amalia Julia Simonetti, MD, Jamaica. Died July 13, 1987; age 91. She was a 1924 graduate of the Medical College of Pennsylvania, Philadelphia. Dr Simonetti was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Charles Aloysius Smith, MD, Albany. Died August 5, 1987; age 73. Dr Smith was a 1940 graduate of Albany Medical College of Union University, Albany. He was a member of the American Academy of Family Practice, the Medical Society of the County of Albany, and the Medical Society of the State of New York.

Charles LeRoy Steinberg, MD, Rochester. Died July 17, 1987; age 84. Dr Steinberg was a 1929 graduate of the University of Louisville School of Medicine, Louisville. He was a Fellow of the American College of Physicians. His memberships included the Academy of Medicine, the New York State Society of Internal Medicine, the American Rheumatism Association, the American Society of Internal Medicine, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Myrtle Wilcox Vincent, MD, Binghamton. Died August 4, 1987; age 79. Dr Vincent was a 1932 graduate of State University of New York at Buffalo School of Medicine, Buffalo. She was a member of the Broome County Medical Society and the Medical Society of the State of New York.

Hyla S. Watters, MD, Tupper Lake. Died August 3, 1987; age 94. Dr Watters was a 1921 graduate of Cornell University Medical College, New York. She was a member of the American Medical Women's Association, the Medical Society of the County of Franklin, and the Medical Society of the State of New York.

MEDICAL SOCIETY OF THE STATE OF NEW YORK
LEGISLATIVE ACCOMPLISHMENTS DURING 1987

Society's Position Prevailed in 9 of 11 Key Issues

●

Key Financial/Political Issues:

Pension Plan Protection. The Governor has signed into law a bill strongly supported by MSSNY which protects approved pension plan assets from the reach of judgment creditors.

Free Excess Insurance Coverage. A bill extending the excess layer of $1 million/$3 million liability insurance protection at no cost to physicians has been signed into law.

Immunity from Damages for Release of Medical Records. Two MSSNY-supported bills providing physicians with immunity for any damages occurring due to the required release of patients' mental health or general medical records have been signed into law.

No-Fault Coverage for Alcohol-Related Accident Victims. Governor Cuomo vetoed a bill strongly supported by MSSNY and approved by the legislature which would have removed the inequity in the No-Fault Law barring physicians and hospitals from receiving payment for treatment rendered to victims of alcohol-related auto accidents. The Governor did, however, direct the Insurance Department to work with health care providers and insurers to devise a solution to the problem, and said he hoped for legislative action on the issue in the 1988 session.

Key Public Health/Educational Issues:

Eligibility for Prenatal Care Services. A bill expanding the number of women eligible for prenatal care services, strongly supported by the MSSNY, has been signed into law.

Presumptive Medicaid Eligibility. The Governor has signed into law a bill making it State policy that patients applying for Medicaid and awaiting transfer to a home health care agency or a long-term care program will be presumed eligible for a 60-day period. The bill was strongly endorsed by the MSSNY.

DNR Procedures. New law supported by MSSNY establishes procedures for physicians issuing Do Not Resuscitate orders which include consulting with patients or specific surrogates. The law grants physicians immunity from both civil and criminal liability, as well as from charges of unprofessional conduct for issuing the DNR orders, if they follow the prescribed procedures.

Physician Drug Dispensing. A bill opposed by MSSNY which would have banned dispensing of prescribed drugs by physicians passed the Assembly but died on the Senate calendar.

Issues of Combined Interest:

Nurse Practitioners. Each house of the legislature passed a different version of a bill to permit nurse practitioners to practice primary care medicine, but they failed to reach agreement, and thus no bill was enacted. MSSNY opposed the bills because they failed to require what Society officials considered the minimally necessary educational level of a master's degree. The assembly bill would have given nurse practitioners the right to diagnose, treat and prescribe with almost complete independence from physicians.

Extension of Liability Study. A bill appropriating funds for a continuation of the study of alternatives to the current tort system for adjudicating medical liability cases has been approved, and extends the study completion date to April 1, 1988.

Medicare/Medicaid Crossover Payments. A bill which would have reinstated Medicaid payment of the co-insurance and deductable amounts under Medicare passed the Senate but died in the Assembly, despite an extensive lobbying effort by MSSNY, the New York State Psychiatric Association, and other provider and consumer groups. Legal challenges by MSSNY to overturn the regulation eliminating such Medicaid payments continue in both the Federal and State Courts.

This year celebrate with Christmas Seals.®

It's a matter of life and breath.®

AMERICAN
LUNG
ASSOCIATION ®
The Christmas Seal People®

e contributed by the publisher as a public service

Residents / Fellows

A $500 AWARD will be presented by the Medical Society of the State of New York to the intern, resident or fellow who submits the best paper on a clinical subject for the Society's 1987 Merit H. Cash Competition.

Entrants need not be members of the MSSNY but must be in training at a hospital within the State of New York.

Entries should be "sponsored" by a faculty member, and all papers submitted should be of sufficient quality to be considered for original publication in the *New York State Journal of Medicine.*

The deadline for receipt of entries has been extended to December 1, 1987, and the prize for the winning paper will be awarded at the MSSNY's annual meeting in April of 1988.

Address all entries to: Division of Scientific Activities
Medical Society of the State of New York
420 Lakeville Road
P.O. Box 5404
Lake Success, NY 11042

Questions? Contact either the Division of Scientific Activities or MSSNY's Resident Physicians Section at (516) 488-6100.

There's never been a better time for her...

and PREMARIN®

Proven benefits beyond relief of vasomotor symptoms

No other estrogen proven effective for osteoporosis

Only conjugated estrogens tablets have established efficacy in both osteoporosis[1] and vasomotor symptoms* at 0.625 mg/day. No other estrogen, oral or transdermal, has established clinical evidence or minimum effective dose in both indications.

No estrogen proven safer

PREMARIN is the most extensively tested estrogen, with an unsurpassed record of long-term safety.
And clinical evidence shows a significantly reduced risk of endometrial hyperplasia when cycled with a progestin.[2]

PREMARIN®
(conjugated estrogens tablets)

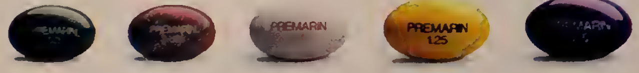
Most trusted for more reasons

*PREMARIN is indicated for moderate-to-severe vasomotor symptoms.

Please see following page for brief summary of prescribing information.

For moderate-to-severe vasomotor symptoms and for osteoporosis

PREMARIN®
(conjugated estrogens tablets)



0.3 mg 0.625 mg 0.9 mg 1.25 mg 2.5 mg

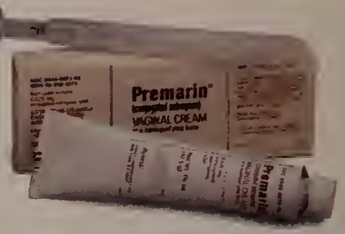
The appearance of these tablets is a trademark of Ayerst Laboratories.

For atrophic vaginitis

PREMARIN®
(conjugated estrogens)

Vaginal Cream

0.625 mg/g



BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION AND PATIENT INFORMATION, SEE PACKAGE CIRCULARS.)*

PREMARIN® Brand of conjugated estrogens tablets, USP
PREMARIN® Brand of conjugated estrogens Vaginal Cream, in a nonliquefying base

> 1. ESTROGENS HAVE BEEN REPORTED TO INCREASE THE RISK OF ENDOMETRIAL CARCINOMA
> Three independent, case-controlled studies have reported an increased risk of endometrial cancer in postmenopausal women exposed to exogenous estrogens for more than one year This risk was independent of the other known risk factors for endometrial cancer. These studies are further supported by the finding that incidence rates of endometrial cancer have increased sharply since 1969 in eight different areas of the United States with population-based cancer reporting systems, an increase which may be related to the rapidly expanding use of estrogens during the last decade. The three case-controlled studies reported that the risk of endometrial cancer in estrogen users was about 4 5 to 13 9 times greater than in nonusers. The risk appears to depend on both duration of treatment and on estrogen dose In view of these findings, when estrogens are used for the treatment of menopausal symptoms, the lowest dose that will control symptoms should be utilized and medication should be discontinued as soon as possible When prolonged treatment is medically indicated, the patient should be reassessed on at least a semi-annual basis to determine the need for continued therapy Although the evidence must be considered preliminary, one study suggests that cyclic administration of low doses of estrogen may carry less risk than continuous administration; it therefore appears prudent to utilize such a regimen. Close clinical surveillance of all women taking estrogens is important. In all cases of undiagnosed persistent or recurring abnormal vaginal bleeding, adequate diagnostic measures should be undertaken to rule out malignancy. There is no evidence at present that "natural" estrogens are more or less hazardous than "synthetic" estrogens at equi-estrogenic doses
> 2 ESTROGENS SHOULD NOT BE USED DURING PREGNANCY.
> The use of female sex hormones, both estrogens and progestogens, during early pregnancy may seriously damage the offspring It has been shown that females exposed in utero to diethylstilbestrol, a nonsteroidal estrogen, have an increased risk of developing, in later life, a form of vaginal or cervical cancer that is ordinarily extremely rare This risk has been estimated as not greater than 4 per 1,000 exposures. Furthermore, a high percentage of such exposed women (from 30% to 90%) have been found to have vaginal adenosis, epithelial changes of the vagina and cervix Although these changes are histologically benign, it is not known whether they are precursors of malignancy Although similar data are not available with the use of other estrogens, it cannot be presumed they would not induce similar changes. Several reports suggest an association between intrauterine exposure to female sex hormones and congenital anomalies, including congenital heart defects and limb-reduction defects. One case-controlled study estimated a 4 7-fold increased risk of limb-reduction defects in infants exposed in utero to sex hormones (oral contraceptives, hormone withdrawal tests for pregnancy, or attempted treatment for threatened abortion). Some of these exposures were very short and involved only a few days of treatment. The data suggest that the risk of limb-reduction defects in exposed fetuses is somewhat less than 1 per 1,000 In the past, female sex hormones have been used during pregnancy in an attempt to treat threatened or habitual abortion. There is considerable evidence that estrogens are ineffective for these indications, and there is no evidence from well-controlled studies that progestogens are effective for these uses If PREMARIN is used during pregnancy, or if the patient becomes pregnant while taking this drug, she should be apprised of the potential risks to the fetus, and the advisability of pregnancy continuation

DESCRIPTION: PREMARIN (conjugated estrogens, USP) contains a mixture of estrogens, obtained exclusively from natural sources, blended to represent the average composition of material derived from pregnant mares' urine It contains estrone, equilin, and 17α-dihydroequilin, together with smaller amounts of 17α-estradiol, equilenin, and 17α-dihydroequilenin as salts of their sulfate esters Tablets are available in 0.3 mg, 0 625 mg, 0 9 mg, 1 25 mg, and 2 5 mg strengths of conjugated estrogens Cream is available as 0.625 mg conjugated estrogens per gram

INDICATIONS AND USAGE: PREMARIN (conjugated estrogens tablets, USP): Moderate-to-severe *vasomotor* symptoms associated with the menopause (There is no evidence that estrogens are effective for nervous symptoms or depression without associated vasomotor symptoms and they should not be used to treat such conditions) Osteoporosis (abnormally low bone mass). Atrophic vaginitis. Kraurosis vulvae. Female castration

PREMARIN (conjugated estrogens) Vaginal Cream is indicated in the treatment of atrophic vaginitis and kraurosis vulvae

PREMARIN HAS NOT BEEN SHOWN TO BE EFFECTIVE FOR ANY PURPOSE DURING PREGNANCY AND ITS USE MAY CAUSE SEVERE HARM TO THE FETUS (SEE BOXED WARNING).

Concomitant Progestin Use: The lowest effective dose appropriate for the specific indication should be utilized Studies of the addition of a progestin for 7 or more days of a cycle of estrogen administration have reported a lowered incidence of endometrial hyperplasia Morphological and biochemical studies of the endometrium suggest that 10 to 13 days of progestin are needed to provide maximal maturation of the endometrium and to eliminate any hyperplastic changes Whether this will provide protection from endometrial carcinoma has not been clearly established. There are possible additional risks which may be associated with the inclusion of progestin in estrogen replacement regimens (See PRECAUTIONS) The choice of progestin and dosage may be important, product labeling should be reviewed to minimize possible adverse effects

CONTRAINDICATIONS: Estrogens should not be used in women (or men) with any of the following conditions: 1 Known or suspected cancer of the breast except in appropriately selected patients being treated for metastatic disease 2. Known or suspected estrogen-dependent neoplasia 3 Known or suspected pregnancy (see Boxed Warning). 4 Undiagnosed abnormal genital bleeding 5. Active thrombophlebitis or thromboembolic disorders. 6 A past history of thrombophlebitis, thrombosis, or thromboembolic disorders associated with previous estrogen use (except when used in treatment of breast or prostatic malignancy).

WARNINGS: Estrogens have been reported to increase the risk of endometrial carcinoma (see Boxed Warning). However, a recent large, case-controlled study indicated no increase in risk of breast cancer in postmenopausal women A recent study has reported a 2- to 3-fold increase in the risk of surgically confirmed gallbladder disease in women receiving postmenopausal estrogens

Adverse effects of oral contraceptives may be expected at the larger doses of estrogen used to treat prostatic or breast cancer or postpartum breast engorgement; it has been shown that there is an increased risk of thrombosis in men receiving estrogens for prostatic cancer and women for postpartum breast engorgement. Users of oral contraceptives have an increased risk of diseases, such as thrombophlebitis, pulmonary embolism, stroke, and myocardial infarction Cases of retinal thrombosis, mesenteric thrombosis, and optic neuritis have been reported in oral contraceptive users An increased risk of postsurgery thromboembolic complications has also been reported in users of oral contraceptives If feasible, estrogen should be discontinued at least 4 weeks before surgery of the type associated with an increased risk of thromboembolism, or during periods of prolonged immobilization Estrogens should not be used in persons with active thrombophlebitis, thromboembolic disorders, or in persons with a history of such disorders in association with estrogen use They should be used with caution in patients with cerebral vascular or coronary artery disease Large doses (5 mg conjugated estrogens per day), comparable to those used to treat cancer of the prostate and breast, have been shown to increase the risk of nonfatal myocardial infarction, pulmonary embolism, and thrombophlebitis When doses of this size are used, any of the thromboembolic and thrombotic adverse effects should be considered a clear risk

Benign hepatic adenomas should be considered in estrogen users having abdominal pain and tenderness, abdominal mass, or hypovolemic shock. Hepatocellular carcinoma has been reported in women taking estrogen-containing oral contraceptives. Increased blood pressure may occur with use of estrogens in the menopause and blood pressure should be monitored with estrogen use. A worsening of glucose tolerance has been observed in patients on estrogen-containing oral contraceptives. For this reason, diabetic patients should be carefully observed Estrogens may lead to severe hypercalcemia in patients with breast cancer and bone metastases.

PRECAUTIONS: Physical examination and a complete medical and family history should be taken prior to the initiation of any estrogen therapy with special reference to blood pressure, breasts, abdomen, and pelvic organs, and should include a Papanicolaou smear. As a general rule, estrogen should not be prescribed for longer than one year without another physical examination being performed. Conditions influenced by fluid retention, such as asthma, epilepsy, migraine, and cardiac or renal dysfunction, require careful observation. Certain patients may develop manifestations of excessive estrogenic stimulation, such as abnormal or excessive uterine bleeding, mastodynia, etc Prolonged administration of unopposed estrogen therapy has been reported to increase the risk of endometrial hyperplasia in some patients. Oral contraceptives appear to be associated with an increased incidence of mental depression. Patients with a history of depression should be carefully observed. Pre-existing uterine leiomyomata may increase in size during estrogen use. The pathologist should be advised of estrogen therapy when relevant specimens are submitted. If jaundice develops in any patient receiving estrogen, the medication should be discontinued while the cause is investigated Estrogens should be used with care in patients with impaired liver function, renal insufficiency, metabolic bone diseases associated with hypercalcemia, or in young patients in whom bone growth is not yet complete If concomitant progestin therapy is used, potential risks may include adverse effects on carbohydrate and lipid metabolism.

The following changes may be expected with larger doses of estrogen:

a. Increased sulfobromophthalein retention.

b. Increased prothrombin and factors VII, VIII, IX, and X; decreased antithrombin 3; increased norepinephrine-induced platelet aggregability

c. Increased thyroid binding globulin (TBG) leading to increased circulating total thyroid hormone, as measured by PBI, T_4 by column, or T_4 by radioimmunoassay Free T_3 resin uptake is decreased, reflecting the elevated TBG, free T_4 concentration is unaltered.

d. Impaired glucose tolerance

e. Decreased pregnanediol excretion.

f. Reduced response to metyrapone test.

g Reduced serum folate concentration.

h. Increased serum triglyceride and phospholipid concentration.

As a general principle, the administration of any drug to nursing mothers should be done only when clearly necessary since many drugs are excreted in human milk.

Long-term, continuous administration of natural and synthetic estrogens in certain animal species increases the frequency of carcinomas of the breast, cervix, vagina, and liver. However, in a recent, large case-controlled study of postmenopausal women there was no increase in risk of breast cancer with use of conjugated estrogens.

ADVERSE REACTIONS: The following have been reported with estrogenic therapy, including oral contraceptives: breakthrough bleeding, spotting, change in menstrual flow; dysmenorrhea; premenstrual-like syndrome; amenorrhea during and after treatment; increase in size of uterine fibromyomata; vaginal candidiasis, change in cervical erosion and in degree of cervical secretion; cystitis-like syndrome; tenderness, enlargement, secretion (of breasts); nausea, vomiting, abdominal cramps, bloating; cholestatic jaundice; chloasma or melasma which may persist when drug is discontinued; erythema multiforme; erythema nodosum; hemorrhagic eruption; loss of scalp hair; hirsutism; steepening of corneal curvature; intolerance to contact lenses; headache, migraine, dizziness, mental depression, chorea; increase or decrease in weight; reduced carbohydrate tolerance, aggravation of porphyria; edema; changes in libido.

ACUTE OVERDOSAGE: May cause nausea, and withdrawal bleeding may occur in females.

DOSAGE AND ADMINISTRATION:

PREMARIN® Brand of conjugated estrogens tablets, USP

1. *Given cyclically for short-term use only.* For treatment of moderate-to-severe *vasomotor* symptoms, atrophic vaginitis, or kraurosis vulvae associated with the menopause (0 3 mg to 1.25 mg or more daily). The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible. Administration should be cyclic (eg, three weeks on and one week off). Attempts to discontinue or taper medication should be made at three- to six-month intervals
2. *Given cyclically:* Osteoporosis. Female castration. Osteoporosis —0 625 mg daily. Administration should be cyclic (eg, three weeks on and one week off). Female castration—1.25 mg daily, cyclically. Adjust upward or downward according to response of the patient. For maintenance, adjust dosage to lowest level that will provide effective control.

Patients with an intact uterus should be monitored for signs of endometrial cancer and appropriate measures taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding

PREMARIN® Brand of conjugated estrogens Vaginal Cream

Given cyclically for short-term use only For treatment of atrophic vaginitis or kraurosis vulvae.

The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible

Administration should be cyclic (eg, three weeks on and one week off).

Attempts to discontinue or taper medication should be made at three- to six-month intervals.

Usual dosage range: 2 g to 4 g daily, intravaginally, depending on the severity of the condition.

Treated patients with an intact uterus should be monitored closely for signs of endometrial cancer and appropriate diagnostic measures should be taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding

References:

1. Lindsay R, Hart OM, Clark OM: The minimum effective dose of estrogen for prevention of postmenopausal bone loss. *Obstet Gynecol* 1984,63:759-763. **2.** Studd JWW, Thom MH, Paterson MEL, et al: The prevention and treatment of endometrial pathology in postmenopausal women receiving exogenous estrogens, in Pasetto N, Paoletti R, Ambrus JL (eds): *The Menopause and Postmenopause* Lancaster, England, MTP Press Ltd, 1980, chap 13.

© 1987 Ayerst Laboratories
All rights reserved

6437/587

Ayerst®
AYERST LABORATORIES
New York, NY 10017

HIV Positive Confirmed. Then What?

AIDS is tough to treat. Ruthless to an increasing patient population, it challenges physicians and nurses as never before in their commitment to quality care.

The Spellman Center for HIV-Related Disease at St. Clare's Hospital and Health Center in New York City, accepts the challenge posed by AIDS and HIV infection. The first facility in the tri-state region to devote its resources solely to the care of AIDS patients, the Spellman Center designates its highest priority as compassionate patient care. A dedicated staff provides comprehensive medical, nursing, psychosocial, and dental treatment in an atmosphere that is warm and understanding.

The Spellman Center—a 60-bed Acute Care Unit, Emergency Service, Outpatient, Ambulatory Infusion and Dental Clinics—accepts referrals of patients with CDC-confirmed AIDS or ARC (AIDS-related complex), and those who are seropositive for HIV. Spellman also reaches out to offer testing, counseling and treatment to persons at risk for AIDS.

If you need consultation concerning your patients, or more information, please call Dr. Debra Spicehandler, the Medical Director, at (212) 459-8409.

The Spellman Center also maintains an information hotline for patients with questions about AIDS: 1-800-433-AIDS.

The Spellman Center
for HIV-Related Disease
A Designated AIDS Treatment Facility
St. Clare's Hospital and Health Center

415 W 51st St
New York, NY 10019

An Affiliate of New York Medical College

The complete journal for family practice physicians

- Reaches 79,000 family physicians monthly
- Presents the most commonly seen patient problems in family practice
- Written by physicians for physicians
- The most current clinical updates in:

Cardiology	Ob/Gyn
Diabetes	Psychiatry
Pediatrics	Gastroenterology

- Provides 20 hours of CME Category 1 Credit

FPR

Pain pathways page 63

Family Practice Recertification®

JULY 1987 VOL 9 NO 7

CLINICAL ARTICLES

Endometrial Cancer: Causes and Patient Evaluation

Pain Management in Primary Care

Controlling Side Effects of Antipsychotic Drugs, Part 2: Extrapyramidal Symptoms

Osteoporosis, Part 2: Prevention and Treatment

KEEPING CURRENT

Assessing Impairment of Elderly Hospitalized Patients

Routine Radiological Testing for Respiratory Illness

Using Ultrasound to Detect Hip Abnormalities

Diagnosing Bone Infection Under Pressure Sores

Slowing Progression of Diabetic Nephropathy

Behavioral Disorders Among Children of Alcoholic Fathers

Catheter Related Septic Central Venous Thrombosis

Withdrawing Patients From Antihypertensive Drug Therapy

Cesarean Section and Infant Survival

Preventing Neonatal Group B Streptococcal Disease

Diagnosing Acute Scrotal Pain

Urinary Tract Infections Among Uncircumcised Infants

Colonoscopy: Detecting Recurrent Colorectal Cancer

Surgical Management of Chronic Intestinal Ischemia

Preventing Travelers' Diarrhea

SPECIAL FEATURE

Willingway: A Fellowship in Alcoholism and Drug Addiction

PRACTICAL ■ CLINICAL ■ EDUCATIONAL ■ CURRENT

(*continued from p 12A*)

10021. Tel: (212) 838-9200, ext 2776.

Dec 5–6. **Vision and the Brain: The Organization of the Central Visual System.** Roosevelt Hotel. Contact: Dr Ivan Bodis-Wollner, Secretary-Treasurer, Association for Research in Nervous and Mental Disease, Inc, Box 1052, Mount Sinai School of Medicine, One Gustave L. Levy Place, New York, NY 10029. Tel: (212) 348-8133.

Dec 7–10. **Neurosurgery NYC/1987.** 22 Cat 1 Credits. Grand Hyatt. *Dec 12–13.* **Advances in Operative Urology.** 16 Cat 1 Credits. NYU Medical Center. *Dec 14–19.* **Computer Tomography Head to Toe.** 37½ Cat 1 Credits. Grand Hyatt. Contact: NYU Medical Center Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Dec 12. **Fluorescein and Laser Workshop.** Manhattan Eye, Ear & Throat Hospital. Contact: Martha Klapp, Manhattan Eye, Ear & Throat Hospital, 210 E 64th St, New York, NY 10021. Tel: (212) 605-3762.

Dec. 12. **The Annual Symposium on Liver Diseases: Clinical Cases, Pathology and Recent Advances.** 6½ Cat 1 Credits. Mount Sinai School of Medicine. Contact: M. A. Gerber, MD, Professor of Pathology, Mount Sinai Medical Center, One Gustave L. Levy Place, New York, NY 10029. Tel: (212) 650-8014.

NASSAU

Dec 2. **A Clinical Day in Ophthalmology: Diabetic Retinopathy.** 7 Cat 1 Credits. Garden City Hotel, Garden City. Contact: Ann J. Boehme, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

WESTCHESTER

Dec 3–6. **Workshop in Reconstructive Surgery of the Hand.** Valhalla. Contact: American Society for Surgery of the Hand, 3025 S Parker Rd, Suite 65, Aurora, CO 80014. Tel: (303) 755-4588.

AROUND THE NATION

CALIFORNIA

Dec 3–5. **Fall Symposium on Back Pain.** 16½ Cat 1 Credits. Fairmount Hotel in San Francisco. Contact: American Back Society, 2647 E 14th St, Suite 401, Oakland, CA 94601. Tel: (415) 536-9929.

Dec 10–11. **Ophthalmic Plastic and Reconstructive Surgery—Past, Present, and Future.** 13 Cat 1 Credits. San Francisco. Contact: Extended Programs in Medical Education, Registration Office, Room 575-U, University of California, San Francisco, CA 94143-0766. Tel: (415) 476-4251.

COLORADO

Dec 3–4. **The First National Fragile X Conference.** Clarion Hotel, Denver. Contact: Expectations Unlimited, Inc, 6897 Paiute Ave, #2, Longmont, CO 80501. Tel: (303) 652-2727.

(*continued on p 28A*)

Patients and
physicians alike prefer

Transderm-Nitro®
nitroglycerin
2.5 mg/24 hr, 5 mg/24 hr, 10 mg/24 hr, 15 mg/24 hr

Almost three quarters of a billion* patches prescribed in the U.S. since 1982

The only patch with a rate-limiting membrane

Familiar, distinctive tan color and unique shape recognized by patients everywhere

There's no substitute for experience

All transdermal nitroglycerin products are being marketed pending final evaluation of effectiveness by the FDA.

CIBA

629-3468-A

(See Brief Summary of Prescribing Information on the next page.)

*Data on file, CIBA Pharmaceutical Co.

Transderm-Nitro® nitroglycerin Transdermal Therapeutic System

BRIEF SUMMARY (FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE INSERT)

INDICATIONS AND USAGE
This drug product has been conditionally approved by the FDA for the prevention and treatment of angina pectoris due to coronary artery disease. The conditional approval reflects a determination that the drug may be marketed while further investigation of its effectiveness is undertaken. A final evaluation of the effectiveness of the product will be announced by the FDA.

CONTRAINDICATIONS
Intolerance of organic nitrate drugs, marked anemia, increased intraocular pressure or increased intracranial pressure.

WARNINGS
In patients with acute myocardial infarction or congestive heart failure, Transderm-Nitro system should be used under careful clinical and/or hemodynamic monitoring

In terminating treatment of anginal patients, both the dosage and frequency of application must be gradually reduced over a period of 4 to 6 weeks to prevent sudden withdrawal reactions, which are characteristic of all vasodilators in the nitroglycerin class.

Transdermal nitroglycerin systems should be removed before attempting defibrillation or cardioversion because of the potential for altered electrical conductivity which may enhance the possibility of arcing, a phenomenon associated with the use of defibrillators.

PRECAUTIONS
Symptoms of hypotension, such as faintness, weakness or dizziness, particularly orthostatic hypotension may be due to overdosage. When these symptoms occur, the dosage should be reduced or use of the product discontinued.

Transderm-Nitro system is not intended for immediate relief of anginal attacks. For this purpose occasional use of the sublingual preparations may be necessary.

ADVERSE REACTIONS
Transient headaches are the most common side effect, especially when higher doses of the drug are used. These headaches should be treated with mild analgesics while Transderm-Nitro therapy is continued. When such headaches are unresponsive to treatment, the nitroglycerin dosage should be reduced or use of the product discontinued.

Adverse reactions reported less frequently include hypotension, increased heart rate, faintness, flushing, dizziness, nausea and vomiting. These symptoms are attributable to the known pharmacologic effects of nitroglycerin, but may be symptoms of overdosage. When they persist the dose should be reduced or use of the product discontinued. In some patients, dermatitis may occur.

DOSAGE AND ADMINISTRATION
Therapy should be initiated with application of one Transderm-Nitro 5 mg/24 hr system to the desired area of skin. Many patients prefer the chest; if hair is likely to interfere with system adhesion or removal, it can be clipped prior to placement of the system. Each system is designed to remain in place for 24 hours, and each successive application should be to a different skin area.

Transderm-Nitro system should not be applied to the distal parts of the extremities.

The usual dosage is one Transderm-Nitro 5 mg/24 hr system. Some patients, however, may require the Transderm-Nitro 10 mg/24 hr system. If a single Transderm-Nitro 5 mg/24 hr system fails to provide adequate clinical response, the patient should be instructed to remove it and apply either two Transderm-Nitro 5 mg/24 hr systems or one Transderm-Nitro 10 mg/24 hr system. More systems may be added as indicated by continued careful monitoring of clinical response. The Transderm-Nitro 2.5 mg/24 hr system is useful principally for decreasing the dosage gradually, though it may provide adequate therapy for some patients when used alone. The optimal dosage should be selected based upon the clinical response, side effects, and the effects of therapy upon blood pressure. The greatest attainable decrease in resting blood pressure that is not associated with clinical symptoms of hypotension especially during orthostasis indicates the optimal dosage. To decrease adverse reactions, the size and/or number of systems should be tailored to the individual patient's needs.

Do not store above 86°F (30°C).

PATIENT INSTRUCTIONS FOR APPLICATIONS
A patient leaflet is supplied with the systems.

HOW SUPPLIED

Transderm-Nitro System Rated Release *in vivo*	Total Nitroglycerin in System	System Size	Carton Size
2.5 mg/24 hr	12.5 mg	5 cm^2	30 Systems...NDC 0083-2025-26
			*30 Systems...NDC 0083-2025-26
			*100 Systems...NDC 0083-2025-30
5 mg/24 hr	25 mg	10 cm^2	30 Systems...NDC 0083-2105-26
			*30 Systems...NDC 0083-2105-26
			*100 Systems...NDC 0083-2105-30
10 mg/24 hr	50 mg	20 cm^2	30 Systems...NDC 0083-2110-26
			*30 Systems...NDC 0083-2110-26
			*100 Systems...NDC 0083-2110-30
15 mg/24 hr	75 mg	30 cm^2	30 Systems...NDC 0083-2115-26
			*30 Systems...NDC 0083-2115-26
			*100 Systems...NDC 0083-2115-30

*Institutional Pack

C87-23 (Rev. 7/87)

Dist. by:
CIBA Pharmaceutical Company
Division of CIBA-GEIGY Corporation
Summit, New Jersey 07901

© 1987, CIBA (9/87) 629-3468-A CIBA

"SELLING PRACTICES IS OUR BUSINESS"

Want to maximize the return on your investment? Before you buy or sell, call for an appraisal to assure the best financial and transfer terms. We are a full service practice broker, not simply a listing service. Since 1981, Countrywide has been instrumental in helping hundreds of doctors like yourself buy and sell their practices. We guide you through the entire sales process from initial meeting to closing. Our offices serve New York, New Jersey and New England.

Countrywide Business Brokerage, Inc.
319 East 24th Street, Suite 23-G, New York, NY 10010
(212) 686-7902 (203) 868-3666 (617) 232-1075

REAL ESTATE FOR SALE OR RENT—CONT'D

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

THE CONTINENTAL—Professional Co-op office space for sale. 70-20 108 St. Premier; prestigious building in the heart of Forest Hills, Queens, NY. Superb location, subway and buses at your door. 24 hour attended garage, central A/C, separate professional wing. 370–1600 sq. ft. possible to combine. Call: Mr. Marvin Weingart, (718) 793-1081.

EAST SIXTIES, PROFESSIONAL BUILDING. The preferred location for internists, cardiologist, rheumatologist. 7 treatment rooms, 2 reception areas to share with three other internists. Share NYC licensed lab, radiology, thermography. Full or part time. (212) 838-2860.

OFFICE TO SHARE N.Y.C., East 82nd Street. Newly renovated, architect designed, state-of-the-art, medical office. 3 consultation rooms, 5 examining rooms, large business office, etc. Fully furnished and equipped, including x-ray and lab. Street entrance. Call Dr. Siegel, office (212) 517-4770, home (212) 628-4696.

MANHATTAN'S GREENWICH VILLAGE AREA, prime medical space for rent/sale. Private street entrance with 2500 sq. ft. of ground floor space and 1300 sq. ft. of lower level space. Ideal for labs, x-ray facility and/or suite doctor's office. Immediate occupancy. Will build to suit. No brokers. No fee. Call owner (516) 487-1714.

1500 SQUARE FOOT SUITE left in our multi-specialty medical center in Syosset, N.Y., a north shore Long Island community, 50 minutes from the heart of New York City. This area needs a neurologist or a proctologist. Since none are at our local hospitals, it is a great opportunity for the right person to live in suburban Long Island and have immediate hospital privileges. Please contact Howard Kantor, M.D. at 50 Underhill Blvd., Syosset, N.Y. 11791.

5 BOROUGHS NASSAU/SUFFOLK. Specialist in professional office space and income properties. Over 500 listings, apartment buildings, office buildings, shopping centers and vacant land. Sales, rentals all sizes and locations. Leidl & Coffey Real Estate (516) 747-2568.

REAL ESTATE FOR SALE OR RENT—CONT'D

CRANDALL REAL ESTATE presents a 6,000 sq. ft. contemporary home on 6.5 acres commanding a dramatic view of the Shawangunk Mountains (near New Paltz). 20 rooms include 7 bedrooms, 4 baths. A massive stone fireplace and 3 car garage plus 2 room guest suite are a few of the amenities. Price $695,000. Additional 27 acres include a 6 acre lake. Total package 1 million dollars. Call (914) 679-2232.

CRANDALL REAL ESTATE presents a luxurious 4 bedroom, 4 bath home with beautiful winter views of Bayville Long Island Sound. Amenities include 3 fireplaces, walnut parquet floor in living room, walnut cabinets and ceramic tile floor in kitchen, and guest apartment. An outdoor deck overlooks the heavily wooded private setting. Walk to 5 beaches, village dock, stores. $475,000. Call (914) 679-2232.

MISCELLANEOUS

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuarial certification and employee statements of participation—partnership agreements and professionals incorporation—No insurance required—references upon request—Wachstock and Dienstag Attorneys at Law, 122 Cutter Mill Road, Great Neck, NY 11021 (516) 773-3322.

1988 CME CRUISE/CONFERENCES on Medicolegal Issues & Risk Management—Caribbean, Mexico, Alaska, China/Orient, Europe, New England/Canada, Trans Panama Canal, South Pacific. Approved for 24-28 CME Cat. 1 Credits (AMA/PRA) and AAFP prescribed credits. Distinguished lecturers. Excellent Group Rates on Finest Ships. Registration limited. Prescheduled in compliance with IRS requirements. Information: International Conferences, 189 Lodge Ave., Huntington Station, NY 11746. (516) 549-0869.

SIGNATURE LOANS—$5,000 to $60,000

DOCTORS ONLY. No collateral, simple interest, 6 years to repay. No points, no fees, no prepay penalties. Use for any reason. Residents, D.O.'s, start-up welcome. For appointment and info call:

(NY) NORTHSTAR FUNDING (212) 323-8076
or MEDIVERSAL 1-800-331-4952 Dept. #135

EQUIPMENT LEASING—$10K-$10 MILLION
NORTHSTAR FUNDING (212) 323-8076 Apply by Phone

MISCELLANEOUS—CONT'D

HAVE YOU RECEIVED ANY COMMUNICATION FROM THE OFFICE OF PROFESSIONAL MEDICAL CONDUCT? If affirmative, contact Susan Kaplan, Attorney-at-Law, (212) 877-5998. Practice limited to the crucial legal problems directly affecting your license to practice medicine—with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professions), and as an Assistant District Attorney in Nassau County. Susan Kaplan, Esq., 165 West End Avenue, Suite 27P, New York, N.Y. 10023.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta, GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

ADVICE, COUNSEL & REPRESENTATION of physicians and other health care professionals in licensure and professional conduct matters, audit preparations, reimbursement issues and practice-related litigation. David E. Ruck, Esq., former Chief, Criminal Division, Office of the New York State Special Prosecutor for Medicaid Fraud Control and Alain M. Bourgeois, Esq., former Acting Justice, Supreme Court of the State of New York. Bourgeois & Ruck, 101 Park Avenue, New York, New York 10178. Telephone 212-661-8070.

PORTABLE RADIOLOGY SERVICE: abdominal + pelvic ultrasound, cardiac echos including 2D + m-mode portable x-rays and portable osteoporosis bone densitometry screening. NYC and suburbs. Schedule weekly/bi-weekly in your office or visits to your patient's home. New portable hi-tech equipment. Hi-quality like standard office images, far superior to hospital portable images. No lead shielding needed for x-rays. University Radiology Service, (212) 265-7392 or (718) 339-5363.

PROFESSIONAL MISCONDUCT ATTORNEYS. William L. Wood, Jr., formerly Executive Director of the New York State Office of Professional Discipline and Anthony Z. Scher, formerly Director of Prosecutions. Our recent tenure as chief enforcement officers for the regulation of New York State's one-half million licensed professionals has given us experience which allows us to represent physicians in professional misconduct proceedings, malpractice, license restoration, controlled drug proceedings, insurance company reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practitioners. Wood & Scher, Attorneys at Law, One Chase Road, Scarsdale, New York 10583. Telephone (914) 723-3500.

SARANTOS
&COMPANY

S. Randy Sarantos, President

THE DOCTORS' MONEY DOCTOR

1 *Investment Management Services*
Pension, Personal and Childrens' Educational Asset Management Programs

2 *Total Financial Planning Services*
Wide selection of 10 different financial plans for every budget level

3 *Insurance Advisory Services*
Custom-designed Insurance and Risk Management programs

4 *Practice Management Services*
Buy/Sell, Associate Advisory Services, Practice Evaluations, Cash-Flow Analysis

5 *Seminar Education Services*
Special Financial Seminar programs for professional societies, groups & institutions

Exclusive Marketing Agents For:

Pro-Tech Practice Evaluations
Low-cost Expanded Option Analysis, including 180 values for your practice.

National Association of Doctors (NAD)
Exclusive agents in Eastern PA, NJ and CT, providing low-cost, high quality financial services.

YES: I would like to know more about Professional Financial Services. Please send me a free brochure.

Name ______

Address ______

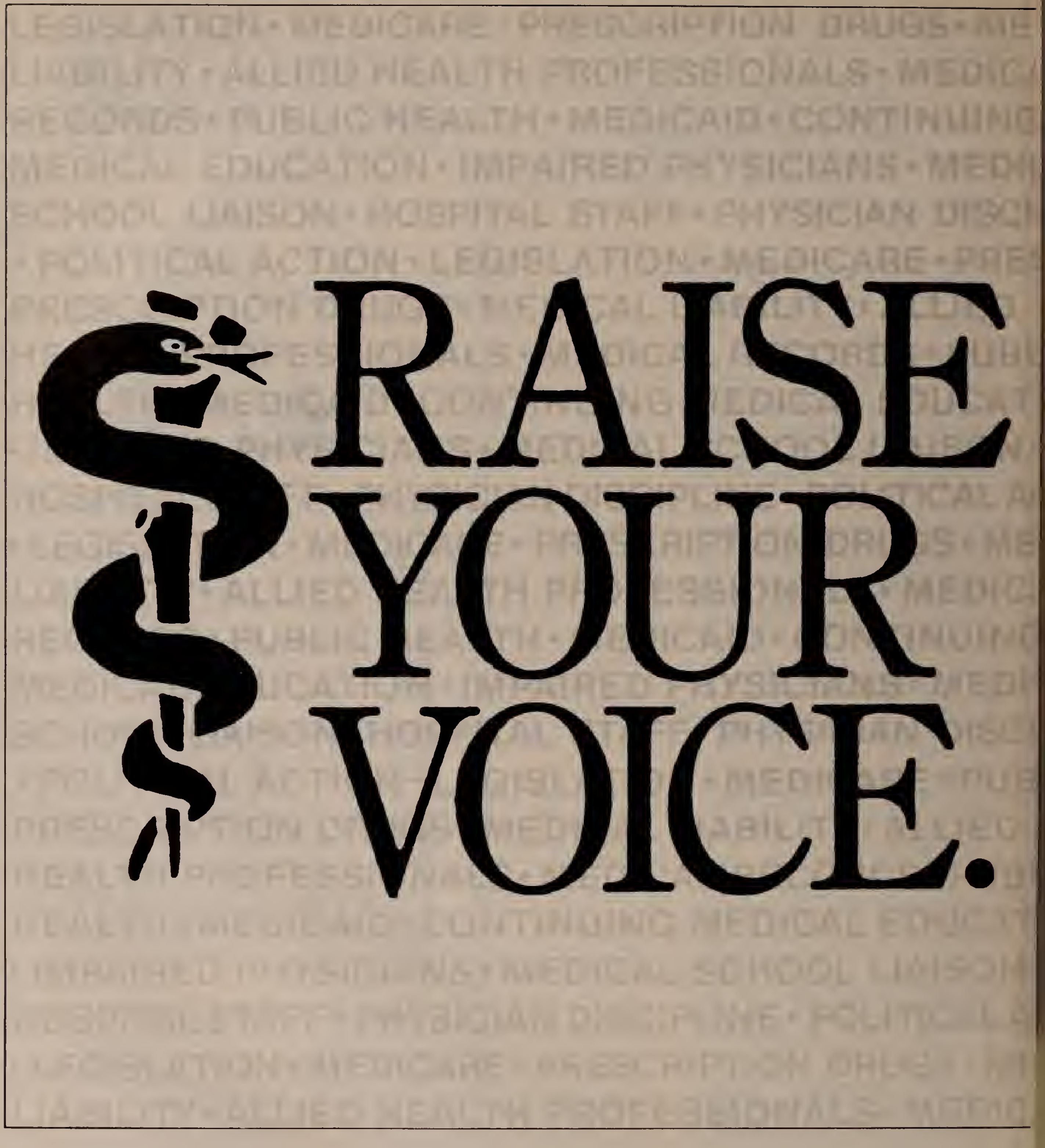
City ______ State ______ Zip ______

Sarantos & Company, Inc. 240 Cedar Knolls, Rd., Suite 310
Cedar Knolls, NJ 07927 (201) 539-4000

— *OR CALL 1-800-223-0164* —

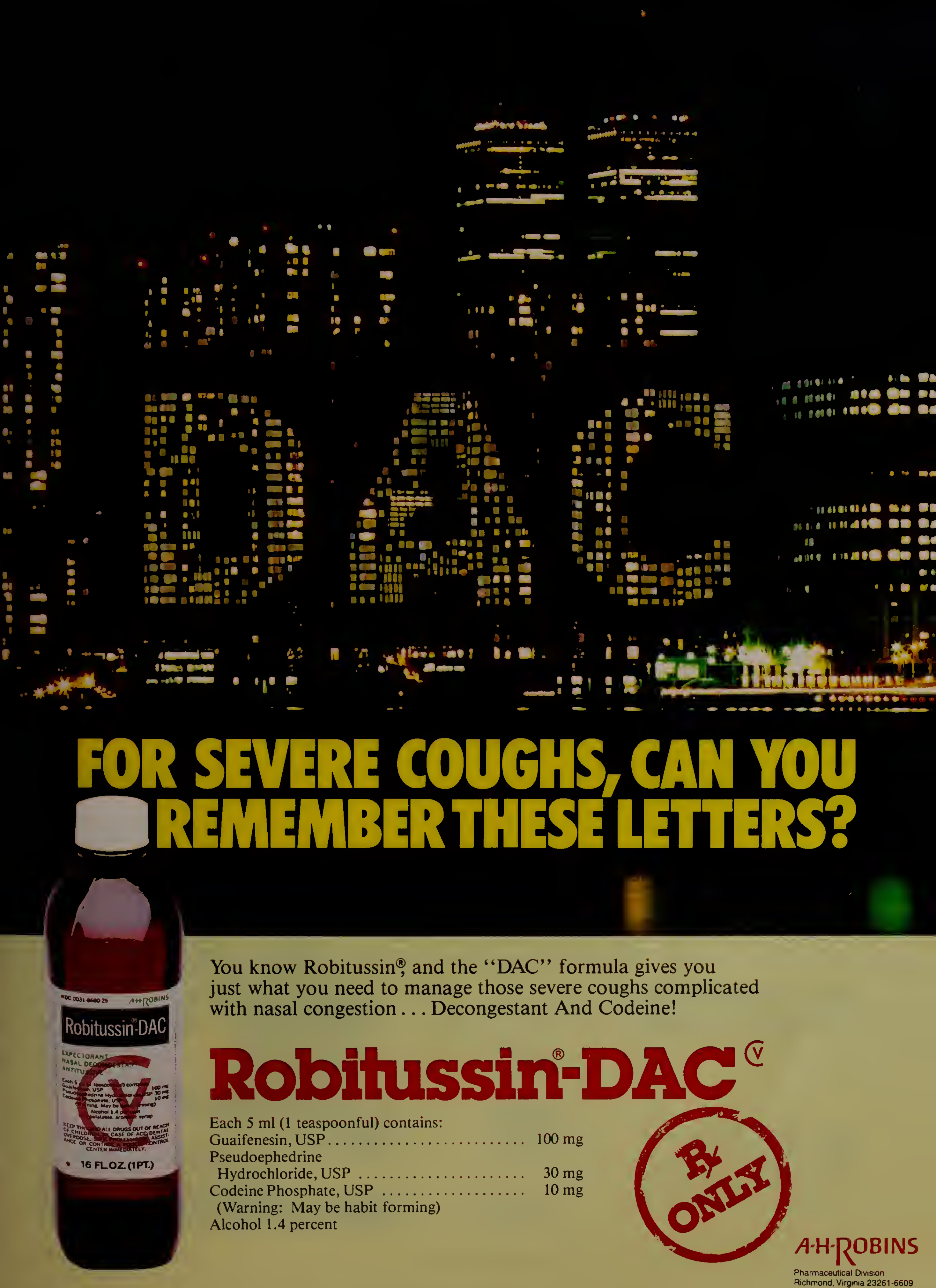
The principals of Sarantos & Company, Inc., a Registered Investment Advisor, are also representatives of Integrated Resources Equity Corp., Member NASD & SIPC, and a registered broker dealer

NYMPAC


LEGISLATION • MEDICARE • PRESCRIPTION DRUGS • ME
LIABILITY • ALLIED HEALTH PROFESSIONALS • MEDIC
RECORDS • PUBLIC HEALTH • MEDICAID • CONTINUING
MEDICAL EDUCATION • IMPAIRED PHYSICIANS • MEDI
SCHOOL LIAISON • HOSPITAL STAFF • PHYSICIAN DISC
• POLITICAL ACTION • LEGISLATION • MEDICARE • PRE
RAISE
YOUR
VOICE.

New York Medical Political Action Committee
An Arm of the Medical Society of the State of New York
420 Lakeville Road, Lake Success, NY 11042 • 516-488-6100

Political Action for Physicians



You know Robitussin®, and the "DAC" formula gives you just what you need to manage those severe coughs complicated with nasal congestion . . . Decongestant And Codeine!

Robitussin®-DAC Ⓥ

Each 5 ml (1 teaspoonful) contains:

Guaifenesin, USP	100 mg
Pseudoephedrine Hydrochloride, USP	30 mg
Codeine Phosphate, USP	10 mg

(Warning: May be habit forming)
Alcohol 1.4 percent

℞ ONLY

A-H-ROBINS
Pharmaceutical Division
Richmond, Virginia 23261-6609

"This year celebrate with Christmas Seals.®"

It's *Christmas Seals' 80th birthday! So join the celebration by putting Christmas Seals on all your gifts and cards. You'll be saying "Happy Birthday!" to Christmas Seals and at the same time helping the American Lung Association fight all lung diseases. This 80th year is the perfect time to send your friends and family healthy holiday wishes with Christmas Seals from the American Lung Association.

It's a matter of life and breath.®

AMERICAN LUNG ASSOCIATION®
The Christmas Seal People®

Christmas Seals is a registered trademark
Space contributed by the publisher as a public service

Dec 19–26. **Current Issues in Medicine.** Marriott's Mark Resort, Vail. Contact: Professional Seminars/University of Miami, PO Box 012318, Miami, FL 33101. Tel: (305) 547-6411.

ILLINOIS

Dec 8–10. **Cardiology Update.** 12 Cat 1 Credits. McCormick Center Hotel, Chicago. Contact: Lisa Krehbiel, Institute for Medical Studies, 30131 Town Center Dr, Suite 215, Laguna Niguel, CA 92677. Tel: (714) 495-4499.

KENTUCKY

Dec 16–17. **Nineteenth Family Medicine Review—Session 1.** Hyatt Regency Hotel, Lexington. *Dec 18–19.* **Trauma Update 1987.** Hyatt Regency Hotel, Lexington. Contact: Joy Greene, Continuing Medical Education, 132 College of Medicine Office Building, University of Kentucky, Lexington, KY 40536-0086. Tel: (606) 233-5161.

TEXAS

Dec 1–3. **Cardiology Update.** 12 Cat 1 Credits. Hotel Inter-Continental, Houston. Contact: Lisa Krehbiel, Institute for Medical Studies, 30131 Town Center Dr, Suite 215, Laguna Niguel, CA 92677. Tel: (714) 495-4499.

VIRGINIA

Dec 7–11. **Noninvasive Vascular Diagnosis by Doppler Ultrasound.** 30 Cat 1 Credits. Alexandria. Contact: Lisa Krehbiel, Institute for Medical Studies, 30131 Town Center Dr, Suite 215, Laguna Niguel, CA 92677. Tel: (714) 495-4499.

AROUND THE WORLD

CARIBBEAN CRUISE

Dec 5–12. **Post-RSNA Imaging at Sea.** Contact: Medical Seminars International, Inc, 21915 Roscoe Blvd, Suite 222, Canoga Park, CA 91304. Tel: (818) 340-0580, ext 280.

MEXICO

Dec 3–5. **Advanced Colposcopy.** Acapulco Princess, Acapulco. Contact: Registrar, American College of Obstetricians and Gynecologists, 600 Maryland Ave, SW, Suite 300 E, Washington, DC 20024-2588. Tel: 1-800-633-8811. ☐

Exercise?
Take the Time!

Back pain and other tensions from stress and overwork can be eased by walking briskly at lunchtime, stretching at your desk, or a good workout after work.

For more information, write to: Fitness, Dept. 83, Washington, DC 20001.

Fitness is feeling GREAT!

The President's Council on Physical Fitness and Sports

Classified Advertising

New York State Journal of Medicine

GENERAL INFORMATION

All classified advertisements are payable in advance, except for government agencies and advertising agencies; a 15% commission to recognized advertising agencies.

Frequency: Monthly
Mailing date: 7th of the month
Closing date: 30 days before mailing date

Circulation: 28,000 (mainly physicians & medical libraries)

DISPLAY ADVERTISING

	1 time	*6 time*	*12 time*	*Width*		*Depth*
1/4-page	$600	$550	$500	3-1/2″	×	4-7/8″
1/6-page	302	279	254	2-1/8″	×	4-7/8″
1/8-page	278	254	230	3-1/2″	×	2-3/8″
1/16-page	164	152	140	3-1/2″	×	1-1/8″
1/18-page	134	122	110	2-1/8″	×	1-1/2″

PARAGRAPH ADVERTISING

The first several words are set in bold face caps. **Rates:** $40.00 per insertion for 50 words or less; additional words are 50 cents each. On request, Dept. numbers will be assigned by the Journal for replies to be forwarded to the advertiser. Dept. numbers are 75 cents per insertion. There is no frequency discount for paragraph ads.

Counting Words: Two initials, each abbreviation, figures consisting of 5 numerals or less, are counted as separate words. For replies, your name and address, or telephone number should appear at the end of the ad and be counted in the total number of words.

For further information, please call the Advertising Department at 516-488-6100.

CLASSIFIED ADVERTISING ORDER FORM

New York State Journal of Medicine
Attn: Classified Advertising
420 Lakeville Rd., P.O. Box 5404
Lake Success, N.Y. 11042

Date______________________

Name____

Address____

City/State/Zip____________________________________

Payment enclosed: $______________

Number of Insertions: ____________

Assign Dept. Number: ____________

Please make check payable to New York State Journal of Medicine.

____

____

____

____

____

____

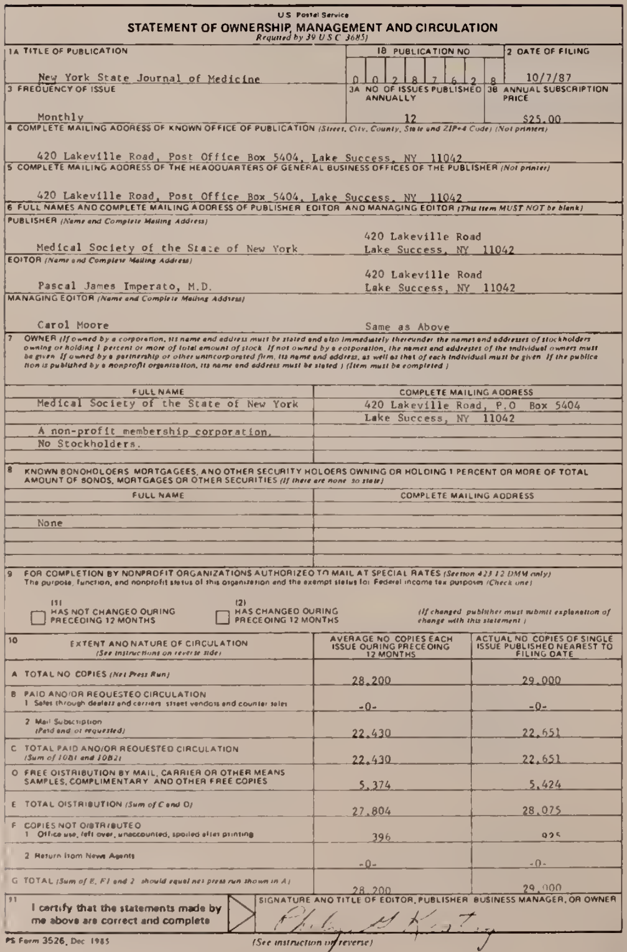
MISCELLANEOUS—CONT'D

SIGNATURE LOANS—$5,000 to $60,000 doctors only. No collateral, simple interest, 4, 5, or 6 year terms. No points, no fees, no prepay penalties. No use restrictions. Residents, D.O.'s, new practitioners welcome. For application and info call: (NY) Northstar Funding (212) 323-8076 or Mediversal 1-800-331-4952 Dept. #135.

PROFESSIONAL MISCONDUCT ADVOCACY with dignity and concern. George Weinbaum, Esq. former deputy director of the New York State Attorney General's office for Medicaid Fraud Control and also former Director of Medicare and private insurance investigations for Empire Blue Cross/Blue Shield is pleased to announce the opening of his office for the practice of health law. Attention is devoted to misconduct avoidance, disciplinary proceedings, reimbursement maximization, controlled drug issues and medical staff representation among other physician needs. George Weinbaum, Esq., 3 Barker Avenue, White Plains, New York 10601. Telephone (914) 686-9310 office hours, (212) 621-7776-24 hours.

EB TRANSCRIPTION SPECIALISTS offering expert work with professional service to fit your needs. All medical specialties. Letters and correspondence, patients charts and manuscript typing. Fast turn around time, free pick up and delivery in the New York area and competitive rates. For additional information call Emily at (718) 894-1987.

FIRM OF GERALD GOLDBERG, CPA-ATTORNEY, formerly with IRS, announces his availability to prepare taxes and plan under changing tax environment, accounting, tax appeals, legal representation or practice transfers. (212) 967-1404 and (516) 565-6260.



U.S. Postal Service

STATEMENT OF OWNERSHIP, MANAGEMENT AND CIRCULATION

Required by 39 U.S.C. 3685)

1A TITLE OF PUBLICATION New York State Journal of Medicine	1B PUBLICATION NO 0 0 2 8 7 6 2 8	2 DATE OF FILING 10/7/87
3 FREQUENCY OF ISSUE Monthly	3A NO OF ISSUES PUBLISHED ANNUALLY 12	3B ANNUAL SUBSCRIPTION PRICE $25.00

4 COMPLETE MAILING ADDRESS OF KNOWN OFFICE OF PUBLICATION *(Street, City, County, State and ZIP+4 Code) (Not printers)*

420 Lakeville Road, Post Office Box 5404, Lake Success, NY 11042

5 COMPLETE MAILING ADDRESS OF THE HEADQUARTERS OF GENERAL BUSINESS OFFICES OF THE PUBLISHER *(Not printer)*

420 Lakeville Road, Post Office Box 5404, Lake Success, NY 11042

6 FULL NAMES AND COMPLETE MAILING ADDRESS OF PUBLISHER, EDITOR, AND MANAGING EDITOR *(This item MUST NOT be blank)*

PUBLISHER *(Name and Complete Mailing Address)*	Medical Society of the State of New York	420 Lakeville Road Lake Success, NY 11042
EDITOR *(Name and Complete Mailing Address)*	Pascal James Imperato, M.D.	420 Lakeville Road Lake Success, NY 11042
MANAGING EDITOR *(Name and Complete Mailing Address)*	Carol Moore	Same as Above

7 OWNER *(If owned by a corporation, its name and address must be stated and also immediately thereunder the names and addresses of stockholders owning or holding 1 percent or more of total amount of stock. If not owned by a corporation, the names and addresses of the individual owners must be given. If owned by a partnership or other unincorporated firm, its name and address, as well as that of each individual must be given. If the publication is published by a nonprofit organization, its name and address must be stated.) (Item must be completed.)*

FULL NAME	COMPLETE MAILING ADDRESS
Medical Society of the State of New York	420 Lakeville Road, P.O Box 5404
	Lake Success, NY 11042
A non-profit membership corporation,	
No Stockholders.	

8 KNOWN BONDHOLDERS, MORTGAGEES, AND OTHER SECURITY HOLDERS OWNING OR HOLDING 1 PERCENT OR MORE OF TOTAL AMOUNT OF BONDS, MORTGAGES OR OTHER SECURITIES *(If there are none, so state)*

FULL NAME	COMPLETE MAILING ADDRESS
None	

9 FOR COMPLETION BY NONPROFIT ORGANIZATIONS AUTHORIZED TO MAIL AT SPECIAL RATES *(Section 423.12 DMM only)*
The purpose, function, and nonprofit status of this organization and the exempt status for Federal income tax purposes *(Check one)*

(1) ☐ HAS NOT CHANGED DURING PRECEDING 12 MONTHS

(2) ☐ HAS CHANGED DURING PRECEDING 12 MONTHS

(If changed, publisher must submit explanation of change with this statement.)

10 EXTENT AND NATURE OF CIRCULATION *(See instructions on reverse side)*	AVERAGE NO. COPIES EACH ISSUE DURING PRECEDING 12 MONTHS	ACTUAL NO. COPIES OF SINGLE ISSUE PUBLISHED NEAREST TO FILING DATE
A TOTAL NO. COPIES *(Net Press Run)*	28,200	29,000
B PAID AND/OR REQUESTED CIRCULATION 1 Sales through dealers and carriers, street vendors and counter sales	-0-	-0-
2 Mail Subscription *(Paid and/or requested)*	22,430	22,651
C TOTAL PAID AND/OR REQUESTED CIRCULATION *(Sum of 10B1 and 10B2)*	22,430	22,651
D FREE DISTRIBUTION BY MAIL, CARRIER OR OTHER MEANS SAMPLES, COMPLIMENTARY, AND OTHER FREE COPIES	5,374	5,424
E TOTAL DISTRIBUTION *(Sum of C and D)*	27,804	28,075
F COPIES NOT DISTRIBUTED 1 Office use, left over, unaccounted, spoiled after printing	396	925
2 Return from News Agents	-0-	-0-
G TOTAL *(Sum of E, F1 and 2 - should equal net press run shown in A)*	28,200	29,000

11 I certify that the statements made by me above are correct and complete

SIGNATURE AND TITLE OF EDITOR, PUBLISHER, BUSINESS MANAGER, OR OWNER

PS Form 3526, Dec. 1985 *(See instruction on reverse)*

ORGANIZED 1807

Doctor . .

Do you, or a physician you know, have a problem with alcohol, drugs, or emotional illness?

If so, please contact the Medical Society of the State of New York's Committee for Physicians' Health, whose function is to assist, *confidentially.*

Telephone: 516-488-7777.

Index to Advertisers

Ayerst Laboratories 18A, 19A, 20A
American Air Lines 8A
Bill of Health Services 6A
CIBA Pharmaceuticals 23A, 24A
St. Clare's Hospital 21A
Classified Advertising 4A, 10A, 24A, 25A, 30A
Family Practice Recertification Magazine 22A
Knoll Pharmaceuticals 3A, 4A
Eli Lilly and Company 1A
Marion Laboratories 9A, 10A, 31A, 32A
Milstein Properties Corp. 6A
A. H. Robins Company 11A, 27A
Roche Laboratories . . . . . . 6A, 7A, 3rd & 4th Cover
Sarantos and Company, Inc. 25A
SK&F Company 13A
Upjohn Company . . . . . . 2nd Cover
U.S. Air Force 12A

The benefit of antianginal protection plus safety...

CARDIZEM®: A FULLER LIFE

diltiazem HCl/Marion

- **A remarkable safety profile**[1-6]
  The low incidence of side effects with Cardizem allows patients to feel better.
- **Protection against angina attacks**[1,5,7-9]
  The predictable efficacy of Cardizem in stable exertional* and vasospastic angina allows patients to do more.
- **A decrease in myocardial oxygen demand**
  Resulting from a lowered heart rate-blood pressure product.[5]
- **Compatible with other antianginals**[6]†
- **Safe in angina with coexisting hypertension, COPD, asthma, or PVD**[1,3,5,6]

90 mg GREATER DOSAGE FLEXIBILITY
NEW 120 mg

*CARDIZEM® (diltiazem HCl) is indicated in the treatment of angina pectoris due to coronary artery spasm and in the management of chronic stable angina (classic effort-associated angina) in patients who cannot tolerate therapy with beta-blockers and/or nitrates or who remain symptomatic despite adequate doses of these agents.

†See Warnings and Precautions.

Please see brief summary of prescribing information on the next page.

CARDIZEM® diltiazem HCl/Marion ANTIANGINAL PROTECTION PLUS SAFETY

℞
Cardizem®
(diltiazem HCl)

☐ 60 mg ☐ 90 mg
☐ 120 mg

Sig: tid

Usual maintenance dosage range: 180-360 mg/day

Brief Summary
Professional Use Information

CARDIZEM®
(diltiazem HCl) 30 mg, 60 mg, 90 mg, and 120 mg Tablets

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. ***Cardiac Conduction.*** *CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1,243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.*
2. ***Congestive Heart Failure.*** *Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt).*
   Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. ***Hypotension.*** *Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.*
4. ***Acute Hepatic Injury.*** *In rare instances, significant elevations in enzymes such as alkaline phosphatase, CPK, LDH, SGOT, SGPT, and other symptoms consistent with acute hepatic injury have been noted. These reactions have been reversible upon discontinuation of drug therapy. The relationship to CARDIZEM is uncertain in most cases, but probable in some. (See PRECAUTIONS.)*

PRECAUTIONS

General. *CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.*

Drug Interaction. *Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)*

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. *A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.*

Pregnancy. *Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.*

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. *Diltiazem is excreted in human milk. One report suggests that concentrations in breast milk may approximate serum levels. If use of CARDIZEM is deemed essential, an alternative method of infant feeding should be instituted.*

Pediatric Use. *Safety and effectiveness in children have not been established.*

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences as well as their frequency of presentation are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%). In addition, the following events were reported infrequently (less than 1%).

Cardiovascular:	*Angina, arrhythmia, AV block (first degree), AV block (second or third degree — see conduction warning), bradycardia, congestive heart failure, flushing, hypotension, palpitations, syncope.*
Nervous System:	*Amnesia, gait abnormality, hallucinations, insomnia, nervousness, paresthesia, personality change, somnolence, tinnitus, tremor.*
Gastrointestinal:	*Anorexia, constipation, diarrhea, dysgeusia, dyspepsia, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH (see hepatic warnings), vomiting, weight increase.*
Dermatologic:	*Petechiae, pruritus, photosensitivity, urticaria.*
Other:	*Amblyopia, dyspnea, epistaxis, eye irritation, hyperglycemia, nasal congestion, nocturia, osteoarticular pain, polyuria, sexual difficulties.*

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: alopecia, gingival hyperplasia, erythema multiforme, and leukopenia. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

Issued 9/86

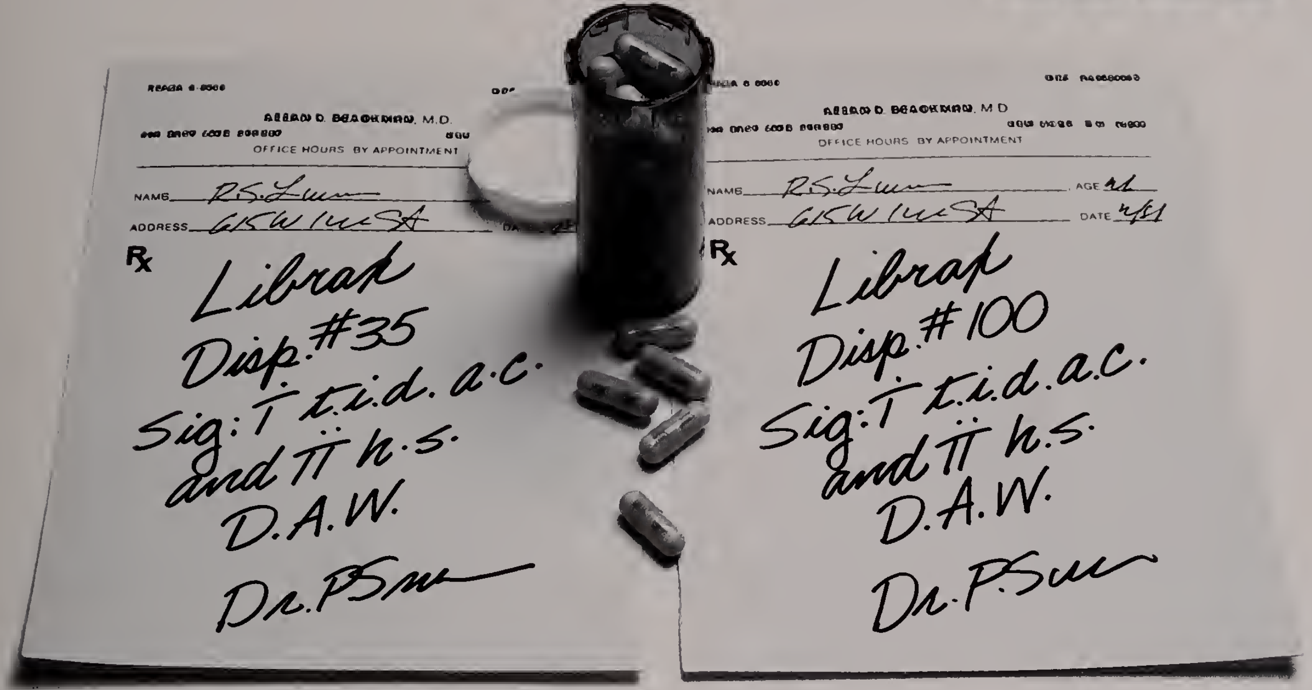
See complete Professional Use Information before prescribing.

References: 1. Schroeder JS: Mod Med 1982;50(Sept):94-116. **2.** Cohn PF, Braunwald E: Chronic ischemic heart disease, in Braunwald E (ed): Heart Disease: A Textbook of Cardiovascular Medicine, ed 2. Philadelphia, WB Saunders Co, 1984, chap 39. **3.** O'Rourke RA: Am J Cardiol 1985;56:34H-40H. **4.** McCall D, Walsh RA, Frohlich ED, et al: Curr Probl Cardiol 1985;10(8):6-80. **5.** Frishman WH, Charlap S, Goldberger J, et al: Am J Cardiol 1985;56:41H-46H. **6.** Shapiro W: Consultant 1984;24(Dec):150-159. **7.** O'Hara MJ, Khurmi NS, Bowles MJ, et al: Am J Cardiol 1984;54:477-481. **8.** Strauss WE, McIntyre KM, Parisi AF, et al: Am J Cardiol 1982;49:560-566. **9.** Feldman RL, Pepine CJ, Whittle J, et al: Am J Cardiol 1982;49:554-559.

Another patient benefit product from
PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC.
KANSAS CITY, MO 64137

0034M7

Specify
Adjunctive
LIBRAX®



NAME R.S. Lum
ADDRESS 615 W 1st St
℞
Librax
Disp. #35
Sig: T t.i.d. a.c.
and TT h.s.
D.A.W.
Dr. P.S.

NAME R.S. Lum
AGE 41
ADDRESS 615 W 1st St
DATE 4/81
℞
Librax
Disp. #100
Sig: T t.i.d. a.c.
and TT h.s.
D.A.W.
Dr. P.S.

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide.

Please consult complete prescribing information, a summary of which follows:

* **Indications:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:
"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl/Roche) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.
Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

P I. 0186

ROCHE®
Roche Products Inc.
Manati, Puerto Rico 00701

To insist on the brand, be sure to specify "Dispense as Written."
When brain and bowel conflict...

It's time for the Peacemaker.

In irritable bowel syndrome* anxiety can aggravate intestinal symptoms, which may further intensify anxiety — a distressing cycle of brain/bowel conflict. Librax intervenes with two well-known compounds. The Librium® (chlordiazepoxide HCl/Roche) component safely relieves anxiety. And Quarzan® (clidinium bromide/Roche) provides antisecretory and antispasmodic action to relieve discomfort associated with intestinal hypermotility.

Dual action — for peace between brain and bowel. Because of possible CNS effects, caution patients about engaging in activities requiring complete mental alertness. Specify Adjunctive

LIBRAX®

Each capsule contains 5 mg chlordiazepoxide HCl
and 2.5 mg clidinium bromide

*Librax has been evaluated as possibly effective as adjunctive therapy in the treatment of peptic ulcer and the irritable bowel syndrome.

Copyright © 1987 by Roche Products Inc. All rights reserved. Please see summary of prescribing information on adjacent page.

STACKS

NEW YORK STATE JOURNAL OF MEDICINE

OCTOBER 1987 • Volume 87, Number 10

MEDICAL SOCIETY OF THE STATE OF NEW YORK ORGANIZED 1807

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA
OCT 14 1987
M. D. S.

Contents

COMMENTARIES

The myth called Medicare 531
JOHN T. PRIOR, MD

A view of the proposed New York State regulations pertaining to the activities of medical students 533
ASSOCIATED MEDICAL SCHOOLS OF NEW YORK

The "exaggerated death" of peritoneal lavage in the management of trauma patients 534
CARL A. SODERSTROM, MD, PHILIP R. MILITELLO, MD

Nonoperative management of blunt abdominal trauma: The cost/benefit margin 536
JOHN A. MARX, MD, ERNEST E. MOORE, MD

IN MEMORIAM

Joseph F. Shanaphy, MD 538

RESEARCH PAPERS

Physicians' attitudes toward and knowledge about generic drug substitution 539
DANIEL FRIEDMAN, MD; ARNOLD JAFFE, PHD; SUSAN STEINHARDT, MPA

The diagnostic significance of free erythrocyte protoporphyrin and the FEP/hemoglobin ratio in plumbism 542
SOLOMON RAJKUMAR, MD; VALERIE GEIBEL, PHD; BHASKAR DEVANAGONDI, MD; BALAKRISHNA KAUL, PHD; AUDREY K. BROWN, MD

HISTORY

The role of physicians in the life insurance industry in the 19th century 546
JOHN S. HALLER, JR

SPECIAL ARTICLE

The physician-patient relationship in interesting times 553
JOSEPH A. STANKAITIS, MD, MPH

MSSNY WHITE PAPER

Mandatory Medicare assignment 560
MARIO V. MENGHINI

CASE REPORTS

Bile peritonitis following expectant management of liver fracture 565
STEPHEN L. BARKER, MD, DAVID FROMM, MD

Modified radical neck dissections necessitated by a retropharyngeal abscess 567
JAY H. KLARSFELD, MD; STEVEN H. SACKS, MD; ROBERT P. GREEN, MD

Fulminant lymphocyte-depleted Hodgkin disease in a homosexual man with HIV infection 570
DONALD COLBOURN, MD; HARRY STASZEWSKI, MD; STEVEN GOLDENBERG, MD; VIRGINIA DONOVAN, MD; FRANK RAIO, MD

Hypersensitivity reaction to anticonvulsants following head injury 571
JAMES M. HASSETT, JR, MD; THOMAS DOOLITTLE, MD; MICHAEL MOLLOY, BS; DAVID LALKA, PHD

LETTERS TO THE EDITOR

Responses to "Death of a dentist" 574
MAURICE TULIN, MD; ALFRED P. INGEGNO, MD; CHARLES S. HIRSCH, MD; CHARLES A. PERERA, MD; BARRY M. ZIDE, DMD, MD

Moses Maimonides and air pollution 574
FRED ROSNER, MD

Rare *Streptococcus pneumoniae* soft tissue abscess 574
LAKSHMANAN SATHYAVAGISWARAN, MD; P. C. T. DICKINSON, MD; JAY DOBKIN, MD

Isolated endometriosis in a Bartholin gland 575
STEPHEN MATSEOANE, MD; TOMMY HARRIS, MD; EDWARD MOSCOWITZ, MD

Tetracycline reduces the need for insulin 576
VINCENT P. GARBITELLI, MD

BOOK REVIEWS 577
NEWS BRIEFS 581
OBITUARIES 583
MEDICAL MEETINGS AND LECTURES 4A

©1986 The Upjohn Company

J-6138 January 1986

HIV Positive Confirmed. Then What?

AIDS is tough to treat. Ruthless to an increasing patient population, it challenges physicians and nurses as never before in their commitment to quality care.

The Spellman Center for HIV-Related Disease at St. Clare's Hospital and Health Center in New York City, accepts the challenge posed by AIDS and HIV infection. The first facility in the tri-state region to devote its resources solely to the care of AIDS patients, the Spellman Center designates its highest priority as compassionate patient care. A dedicated staff provides comprehensive medical, nursing, psychosocial, and dental treatment in an atmosphere that is warm and understanding.

The Spellman Center—a 60-bed Acute Care Unit, Emergency Service, Outpatient, Ambulatory Infusion and Dental Clinics—accepts referrals of patients with CDC-confirmed AIDS or ARC (AIDS-related complex), and those who are seropositive for HIV. Spellman also reaches out to offer testing, counseling and treatment to persons at risk for AIDS.

If you need consultation concerning your patients, or more information, please call Dr. Debra Spicehandler, the Medical Director, at (212) 459-8409.

The Spellman Center also maintains an information hotline for patients with questions about AIDS: 1-800-433-AIDS.

The Spellman Center
for HIV-Related Disease
A Designated AIDS Treatment Facility
St. Clare's Hospital and Health Center

415 W 51st St
New York, NY 10019

An Affiliate of New York Medical College

NEW YORK STATE JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
ELIZA H. CALDWELL, MD
JOSEPH F. MURATORE, MD
JOHN T. PRIOR, MD
GITA S. SINGH*
STANFORD WESSLER, MD

**Medical student*

Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Consulting Editor and Book Review Editor	RICHARD B. BIRRER, MD
Consulting Editor	NAOMI R. BLUESTONE, MD, MPH
Consulting Editor	CARL POCHEDLY, MD
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Managing Editor	CAROL L. MOORE
Advertising Director	ROBERT MILLER
Consulting Medical Writer	VICKI GLASER
Editorial Assistant	MILDRED J. AREMANN
Secretary	ELIZABETH J. SOMERS
Librarian	ELLA ABNEY
Assistant Librarian	ELEANOR BURNS

ASSOCIATE EDITORIAL BOARD 1987

MICHAEL E. BERLOW, MD
RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MYLES S. GOMBERT, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
JAMES M. MORRISSEY, ESQ
STEPHEN NORDLICHT, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. United Media Associates, Inc, 16 Bruce Park Ave, Greenwich, CT 06830. Telephone: (203) 661-9702.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

SAMUEL M. GELFAND, MD,	*President*
JOHN A. FINKBEINER, MD,	*Past-President*
CHARLES D. SHERMAN, JR, MD,	*President-Elect*
DAVID M. BENFORD, MD,	*Vice-President*
JOHN H. CARTER, MD,	*Secretary*
GEORGE LIM, MD,	*Assistant Secretary*
MORTON KURTZ, MD,	*Treasurer*
ROBERT A. MAYERS, MD,	*Assistant Treasurer*
CHARLES N. ASWAD, MD,	*Speaker*
SEYMOUR R. STALL, MD,	*Vice-Speaker*

Councilors

Term Expires 1988

JAMES H. COSGRIFF, JR, MD,	Erie
RICHARD A. HUGHES, MD,	Warren
ANTONIO F. LASORTE, MD,	Broome
SIDNEY MISHKIN, MD,	Nassau

Term Expires 1989

ROBERT E. FEAR, MD,	Suffolk
STANLEY L. GROSSMAN, MD,	Orange
THOMAS D. PEMRICK, MD,	Rensselaer
RALPH E. SCHLOSSMAN, MD,	Queens

Term Expires 1990

STUART I. ORSHER, MD, (*elected to serve until 1988*)	New York
DUANE M. CADY, MD,	Onondaga
WILLIAM A. DOLAN, MD,	Monroe
ROBERT E. GORDON, MD,	Kings

Resident Councilor (*representing the resident physician membership*)

KATHLEEN E. SQUIRES, MD,	New York

Student Councilor (*representing the medical student membership*)

MICHAEL PACIOREK,	Onondaga

Trustees

RICHARD D. EBERLE, MD, (*Chairman*)	Onondaga
EDGAR P. BERRY, MD,	New York
JAMES M. FLANAGAN, MD,	Wayne
ALLISON B. LANDOLT, MD,	Westchester
DANIEL F. O'KEEFFE, MD,	Warren
BERNARD J. PISANI, MD,	New York
VICTOR J. TOFANY, MD,	Monroe

Executive Vice-President	DONALD F. FOY
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Vice-President Emeritus	GEORGE J. LAWRENCE, JR, MD
Director, Division of Policy Coordination	IRMA A. ERICKSON
Director, Division of Scientific Publications	PASCAL JAMES IMPERATO, MD
Director, Division of Communications	EDWARD A. HYNES
Director, Division of Computer Services	IVAN H. NAJMAN
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Governmental Affairs	GERARD E. CONWAY, ESQ
General Counsel	DONALD R. MOY, JD
Director, Division of Management and Meeting Services	ROBERT MILLER
Director, Division of Membership Support Services	EUNICE M. SKELLY
Director, Division of Scientific and Educational Activities	PETER B. FARNSWORTH, MD
Director, Division of Socio-Medical Economics	MARIO V. MENGHINI
Medical Director, Committee for Physicians' Health	BARTON A. HARRIS, MD
Consultant	EDWARD SIEGEL, MD

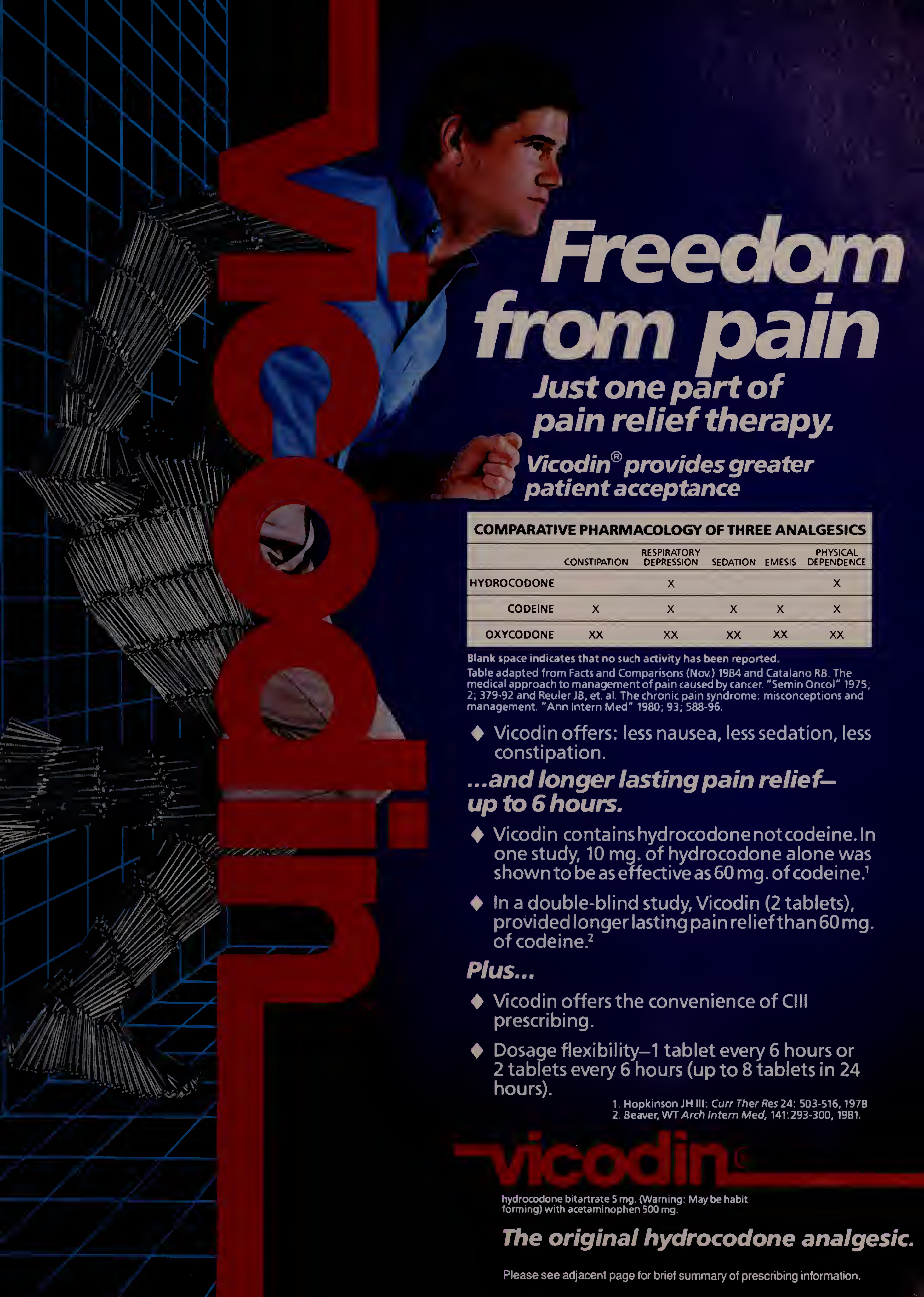
The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York.

Copyright © 1987, Medical Society of the State of New York. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.



Freedom from pain

Just one part of pain relief therapy.

Vicodin® provides greater patient acceptance

COMPARATIVE PHARMACOLOGY OF THREE ANALGESICS

	CONSTIPATION	RESPIRATORY DEPRESSION	SEDATION	EMESIS	PHYSICAL DEPENDENCE
HYDROCODONE		X			X
CODEINE	X	X	X	X	X
OXYCODONE	XX	XX	XX	XX	XX

Blank space indicates that no such activity has been reported.

Table adapted from Facts and Comparisons (Nov.) 1984 and Catalano RB. The medical approach to management of pain caused by cancer. "Semin Oncol" 1975; 2; 379-92 and Reuler JB, et. al. The chronic pain syndrome: misconceptions and management. "Ann Intern Med" 1980; 93; 588-96.

- Vicodin offers: less nausea, less sedation, less constipation.

...and longer lasting pain relief– up to 6 hours.

- Vicodin contains hydrocodone not codeine. In one study, 10 mg. of hydrocodone alone was shown to be as effective as 60 mg. of codeine.[1]
- In a double-blind study, Vicodin (2 tablets), provided longer lasting pain relief than 60 mg. of codeine.[2]

Plus...

- Vicodin offers the convenience of CIII prescribing.
- Dosage flexibility–1 tablet every 6 hours or 2 tablets every 6 hours (up to 8 tablets in 24 hours).

1. Hopkinson JH III: *Curr Ther Res* 24: 503-516, 1978
2. Beaver, WT *Arch Intern Med,* 141:293-300, 1981.

vicodin®

hydrocodone bitartrate 5 mg. (Warning: May be habit forming) with acetaminophen 500 mg.

The original hydrocodone analgesic.

Please see adjacent page for brief summary of prescribing information.

vicodin®

hydrocodone bitartrate 5mg (Warning May be habit forming), and acetaminophen 500mg

INDICATIONS AND USAGE: For the relief of moderate to moderately severe pain.
CONTRAINDICATIONS: Hypersensitivity to acetaminophen or hydrocodone
WARNINGS
Drug Abuse and Dependence: VICODIN is subject to the Federal Controlled Substances Act (Schedule III). Psychic dependence, physical dependence and tolerance may develop upon repeated administration of narcotics, therefore, VICODIN should be prescribed and administered with the same caution appropriate to the use of other oral-narcotic-containing medications.
Respiratory Depression: At high doses or in sensitive patients, hydrocodone may produce dose-related respiratory depression by acting directly on brain stem respiratory centers. Hydrocodone also affects centers that control respiratory rhythm, and may produce irregular and periodic breathing
Head Injury and Increased Intracranial Pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a preexisting increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries
Acute Abdominal Conditions: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions.
PRECAUTIONS
Special Risk Patients: VICODIN should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addisan's disease, prostatic hypertrophy or urethral stricture.
Information for Patients: VICODIN, like all narcotics, may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery; patients should be cautioned accordingly
Cough Reflex: Hydrocodone suppresses the cough reflex, caution should be exercised when VICODIN is used postoperatively and in patients with pulmonary disease.
Drug Interactions: The CNS-depressant effects of VICODIN may be additive with that of other CNS depressants. When combined therapy is contemplated, the dose of one or both agents should be reduced. The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone. The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus
Usage in Pregnancy: Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose. There are no adequate and well-controlled studies in pregnant women. VICODIN should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus
Nonteratogenic Effects: Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent The intensity of the syndrome does not always correlate with the duration of maternal opioid use or dose.
Labor and Delivery: Administration of VICODIN to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used
Nursing Mothers: It is not known whether this drug is excreted in human milk; therefore, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.
Pediatric Use: Safety and effectiveness in children have not been established.
ADVERSE REACTIONS
Central Nervous System: Sedation, drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, dizziness, psychic dependence, mood changes
Gastrointestinal System: Nausea and vomiting may occur, they are more frequent in ambulatory than in recumbent patients Prolonged administration of VICODIN may produce constipation.
Genitourinary System: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported
Respiratory Depression: (See WARNINGS.)
DOSAGE AND ADMINISTRATION: Dosage should be adjusted according to the severity of the pain and the response of the patient. However, tolerance to hydrocodone can develop with continued use, and the incidence of untoward effects is dose related The usual dose is one tablet every six hours as needed for pain (If necessary, this dose may be repeated at four-hour intervals) In cases of more severe pain, two tablets every six hours (up to eight tablets in 24 hours) may be required
Revised January, 1987 MR 1987 5786

Knoll Pharmaceuticals
A Unit of BASF K&F Corporation
Whippany, New Jersey 07981

BASF Group

knoll®

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

NOVEMBER 1987

AROUND THE STATE

MANHATTAN

Nov 3–5. **Structure and Activity of Heparin and Related Polysaccharides.** Vista International Hotel. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd St, New York, NY 10021.

Nov 4. **Involuntary Hospitalization and Treatment in New York 1987. The State of Debate.** Beth Israel Medical Center. Contact: Beverly R. Baptiste, Medical Education Office, Beth Israel Medical Center, First Ave at 16th St, New York, NY 10003. Tel: (212) 420-2849.

Nov 6. **New Dimensions in the Management of Facial, Head and Neck Pain.** Conference Room, New York Eye and Ear Infirmary. Contact: Barry C. Cooper, DDS, Conference Coordinator, 310 East 14th St, New York, NY 10003. Tel: (212) 598-1600.

Nov 6. **New York Allergy Society—Annual Teaching Day.** 5 Cat 1 Credits. Cornell Medical Center. Contact: Ms Noell Sexton, The New York Allergy Society, c/o St Lukes-Roosevelt Hospital Center, 428 West 59th St, New York, NY 10019. Tel: (212) 554-7192.

Nov 7–8. **The Cardiac Patient for Non-Cardiac Surgery—The Experts Opine.** Mount Sinai Medical Center. Contact: Department of Anesthesiology, Mount Sinai Medical Center, One Gustave L. Levy Place, Box 1010, New York, NY 10029. Tel: (212) 650-7630.

Nov 7–8. **New York Otology: '87.** Manhattan Eye, Ear and Throat Hospital. Contact: Joanne Lupetin, Administrator, Department of Otolaryngology, Manhattan Eye, Ear and Throat Hospital, 210 East 64th Street, New York, NY 10021. Tel: (212) 838-9200, ext 2448.

Nov 19–21. **Obstetric and Gynecologic Emergencies.** 16 Cat 1 Credits. Grand Hyatt Hotel. Contact: Registrar, The American College of Obstetricians and Gynecologists, 600 Maryland Ave, SW, Suite 300 East, Washington, DC 20024-2588. Tel: 1-800-633-8811.

Nov 20. **First International Breast Cancer Chemoprevention Workshop-Conference.** Essex House. Contact: Jaclyn Silverman, Chemoprevention Workshop Coordinator, Division of Medical Oncology, Mount Sinai Medical Center, One Gustav L. Levy Place, New York, NY 10029. Tel: (212) 369-5440.

AROUND THE NATION

ARIZONA

Nov 2–5. **Update in Clinical Medicine.** The Sheraton Scottsdale Resort. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

Nov 5–7. **Gynecologic Surgery: Principles, Techniques, and Management of Complications.** 16 Cat 1 Credits. Contact: Registrar, The American College of Obstetricians and Gynecologists, 600 Maryland Ave SW, Suite 300 East, Washington, DC 20024-2588. Tel: 1-800-633-8811.

CALIFORNIA

Nov 5–7. **San Francisco Otology Update—1987.** 17 Cat 1 Credits. Holiday Inn Union Square Hotel. Contact: University of California, Extended Programs in Medical Education, Room U-569, San Francisco, CA 94143-0742. Tel: (415) 476-5808.

Nov 6–8. **The 33rd Annual Group Therapy Symposium.** Cathedral Hill Hotel, San Francisco. Contact: Extended Programs in Medical Education, University of California, Room U-569, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

Nov 11–15. **16th Annual Meeting, International Congress of Gynecologic Endoscopy.** Sheraton Palace, San Francisco. Contact: Jordan M. Phillips, MD, American Association of Gynecologic Laparoscopists, 13021 East Florence Ave, Santa Fe Springs, CA 90670. Tel: (213) 946-8774.

(*continued on p 16A*)

Mutual trust...

As a physician, you know the importance of trust between you and your patients. What's more, you're well aware that the foundation of that trust lies in the knowledge and experience you have demonstrated as a professional.

Expertise is also the cornerstone of AMA Advisers, Inc., a subsidiary of the American Medical Association. We naturally have your investment interests in mind. That's why we dedicate our efforts to physicians like you...and why we provide a variety of alternative investment strategies that can be tailored to your specific needs.

You can build your investment program around The AMA Group, the mutual funds managed by AMA Advisers, Inc., designed to help you meet your financial goals with a broad choice of investment strategies. Plus, we offer a full range of investor services, including our newest service, Asset Allocation, and, of course, professional management of the highest quality.

Whether you're new to investing or an "old pro", AMA Advisers' money management expertise may help you reach your financial goals. Simply complete and mail the coupon for your FREE Information Kit on the funds in The AMA Group. We'll send you a prospectus with more complete information detailing fees and expenses. Please read the prospectus carefully before you invest or send money.

Services and products as described herein are not offered for sale in any state where they are not lawfully registered.

Building Mutual Trust

AMA ADVISERS, INC.

AMA Advisers, Inc. is a subsidiary of the American Medical Association and manages the mutual funds in The AMA Group.

FOR FASTER ACTION, CALL TOLL FREE 1-800-AMA-FUND EXTENSION 2043, ANYTIME!

MAIL TO: The AMA Group NY2043
Mutual funds managed by AMA Advisers, Inc.
P.O. Box 1923, West Chester, PA 19380-1923

☑ YES. I want my FREE Information Kit on the funds in The AMA Group, as well as a prospectus with more complete information detailing fees and expenses.

Name ________________________

Address ________________________

City ________________________

State ______________ Zip ______________

Daytime Telephone (______) ______________

Classified Advertising

"SELLING PRACTICES IS OUR BUSINESS"

Want to maximize the return on your investment? Before you buy or sell, call for an appraisal to assure the best financial and transfer terms. We are a full service practice broker, not simply a listing service. Since 1981, Countrywide has been instrumental in helping hundreds of doctors like yourself buy and sell their practices. We guide you through the entire sales process from initial meeting to closing. Our offices serve New York, New Jersey and New England.

Countrywide Business Brokerage, Inc.
319 East 24th Street, Suite 23-G, New York, NY 10010
(212) 686-7902 (203) 868-3666 (617) 232-1075

PHYSICIANS WANTED

EMERGENCY MEDICINE POSITIONS—Full/part time emergency medicine physicians sought by multi state professional association for openings in metropolitan NY, PA, MD, DC, FL, New England and throughout U.S. Contact or send CV to Liberty Healthcare Corporation, 399 Market Street, Suite 400, Philadelphia, PA 19106, (215) 592-7400 or outside Pa (800) 331-7122.

ARIZONA-BASED PHYSICIAN RECRUITMENT firm has quality opportunities coast to coast. Available positions in most primary care and surgical specialties to include OB/GYN, Orthopedics, ER, and ENT. "Quality Physicians for Quality Clients since 1972." Call 602-990-8080; or send CV to: Mitchell & Associates, Inc., PO Box 1804, Scottsdale, AZ 85252.

GENERAL INTERNIST—Immediate need due to recent death of respected physician. Take over established practice, fully equipped office; coverage with 4 other internists who enthusiastically support this effort. Unlimited potential with first year compensation guaranteed. Colorful community in western New York; close to Finger Lakes with excellent family environment. Reply Dept. 449 c/o NYSJM.

GREAT OPPORTUNITY for Directorship and staff positions in newly designed emergency room located near the mountains in south central New York; just three hours from New York City. 28,000 annual patient visits. Lucrative compensation package for director including benefits. Professional liability insurance procured on your behalf. Flexible scheduling gives you the free time to enjoy the recreational aspects of the area. Contact: Christine Fairchild, Coastal Emergency Services, Inc., 550 Latona Rd., Building C, Rochester, NY 14626; (716) 225-4090 (collect).

INTERNIST WANTED—Busy three man General Internal Medicine practice in community setting seeks board eligible/board certified internist for primary care responsibilities. Excellent benefits and opportunties offered for association. Contact (201) 837-3200 or send C.V. to Dr. Peter W. Coppola, 751 Teaneck Road, Teaneck, NJ 07666.

DISABILITY EVALUATING PHYSICIANS: Full or part-time positions available performing independent disability determinations, no-fault examinations and various other physical examinations in rapidly expanding Albany office. Send C.V. to AIP, P.O. Box 13964, Albany, New York 12212.

POSITIONS WANTED

SURGEON FOR HIRE. Experienced general surgeon available 3 days a week, for first assisting, Westchester area. O.R. work only. Must include monetary offer in your reply. Dept. 450 c/o NYSJM.

POSITIONS WANTED—CONT'D

ENDOCRINE PRACTICE DESIRED: M.D., 1983, ABIM 1986; B/E Endocrinology/Metabolism, 1988; University trained; clinical experience in general endocrinology, diabetes, reproductive endocrinology, and lipoprotein disorders. Available July, 1988. Desire practice in NYC/metropolitan area. Reply to Alex Stagnaro-Green, M.D., 228 Voorhis Ave., River Edge, New Jersey 07661. (201) 265-4859.

PRACTICES AVAILABLE

FOR SALE: FAMILY PRACTICE. Retiring, immediate occupancy. Professional Building, long standing active practice. In excellent community on main thoroughfare in Nassau County, 200 feet across Queens border. Ideal for new ambitious M.D. or relocation. 1200 square feet modern rooms, fully equipped with x-ray. Low overhead. 10 minutes from 4 major hospitals. Reply Dept. 447 c/o NYSJM.

DERMATOLOGY PRACTICE FOR SALE. Terms negotiable, 70's upper east side. Call (212) 744-6809.

EQUIPMENT

50% OFF PREVIOUSLY OWNED MEDICAL—Laboratory, x-ray, ultrasound equipment. Buy, Sell, Broker, Repair. Appraisals by certified surgical consultants. Medical Equipment Resale & Repair, 24026 Haggerty Road, Farmington Hills, MI 48018. 1-800-247-5826. (313) 477-6880.

DISCOUNT HOLTZER SCANNING SERVICES starting at $35.00. Spacelabs holter recorders available from $1,275.00. Smallest and lightest holters update. Fast service (24–48 hours) turnover. Hook-up kits starting at $4.95. Special introductory offer of three free tests at the purchase or lease of the holter. Stress test electrodes available at 34 cents. Scanning paper available at $18.95. Cardiologist overread available at $15.00. If interested call 1-800-248-0153.

REAL ESTATE FOR SALE OR RENT

FOR SALE—Combination professional office and home, Oceanside, NY; opposite South Nassau Community Hospital on 1/3rd acre lot. Never pay rent again. Telephone (516) 536-6279.

REAL ESTATE FOR SALE OR RENT—CONT'D

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

MANHATTAN, 220 EAST 69TH STREET. Share expenses. Luxury building. Own 12 X 15 consultation room, garden window; or share two days. Share 3 equipped examination rooms. Laboratory, x-ray services on premises. Suitable for Internist, Surgeon or Psychiatrist. (212) 737-4020.

FOR RENT completely equipped office in upstate New York. Office has large waiting, consultation, treatment, x-ray, lab, physiotherapy, and 2 bathrooms. Utilities included. Attached garage. Only physician here for 54 years. Contact: Francis D. Brown, M.D., Maple-Bank, Hobart, NY 13788.

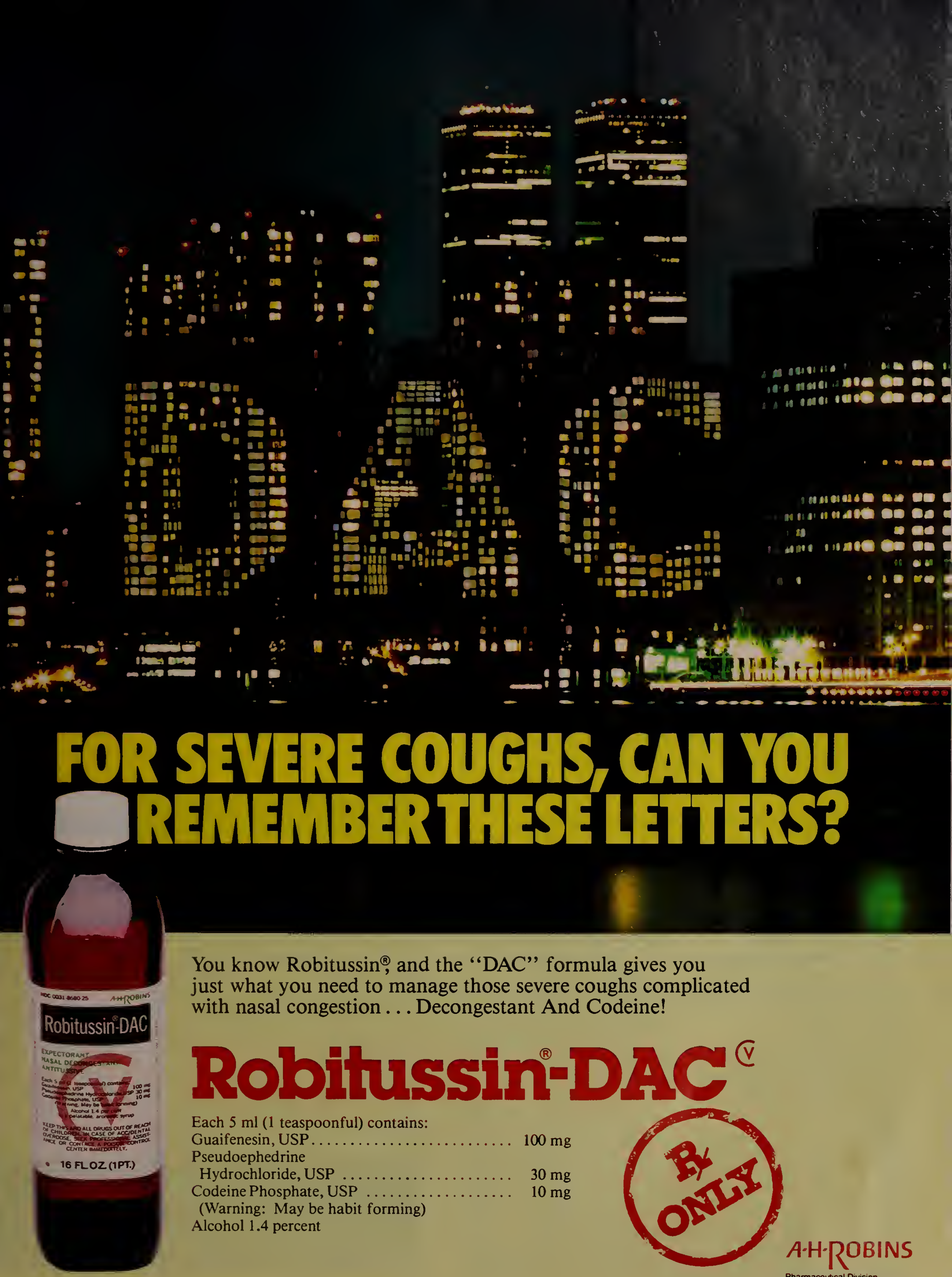
THE CONTINENTAL—Professional Co-op office space for sale. 70-20 108 St. Premier, prestigious building in the heart of Forest Hills, Queens, NY. Superb location, subway and buses at your door. 24 hour attended garage, central A/C, separate professional wing. 370–1600 sq. ft. possible to combine. Call: Mr. Marvin Weingart, (718) 792-1081.

OFFICE SPACE NEW ROCHELLE, NY. 3 Internists in office, all lab and x-ray. Call (914) 632-1952.

FOR SALE—OFFICE BUILDING, L.I. Suffolk County. Family practitioner retiring after 33 years. Building has 3 office suites, C/A, W/W carpeting, paneling, separate entrances, private parking and 3 zone heat. Excellent location, near hospitals. Suitable all specialties. For information please call (516) 667-7872.

OFFICE FOR RENT: Furnished, excellent location. Center of Village, Schuyler Hospital two blocks away. Paul F. Willwerth, M.D., 105 Genesee Street, Montour Falls, N.Y. 14865 (607) 535-9671 or (607) 546-2450.

CUSTOM BUILD superbly designed professional home located in the East Northport area on highly visible 1/2 acre, just minutes from local hospitals, parkways and schools. Reception room plus 900 sq. ft. useable space on lower level. Upper level features wonderful contemporary living area with highhat lighting, skylights and cathedral ceilings. Asking $425,000. The Daniel Gale Agency, Camille Sonesen (516) 754-3400 or Joan Gannon (516) 692-6770.



You know Robitussin®, and the "DAC" formula gives you just what you need to manage those severe coughs complicated with nasal congestion . . . Decongestant And Codeine!

Robitussin®-DAC Ⓥ

Each 5 ml (1 teaspoonful) contains:

Guaifenesin, USP	100 mg
Pseudoephedrine Hydrochloride, USP	30 mg
Codeine Phosphate, USP	10 mg

(Warning: May be habit forming)
Alcohol 1.4 percent

℞ ONLY

A-H-ROBINS
Pharmaceutical Division
Richmond, Virginia 23261-6609

SARANTOS & COMPANY

S. Randy Sarantos, D.D.S., President

THE DOCTORS' MONEY DOCTOR

1 *Investment Management Services*
Pension, Personal and Childrens' Educational Asset Management Programs

2 *Total Financial Planning Services*
Wide selection of 10 different financial plans for every budget level

3 *Insurance Advisory Services*
Custom-designed Insurance and Risk Management programs

4 *Practice Management Services*
Buy/Sell, Associate Advisory Services, Practice Evaluations, Cash-Flow Analysis

5 *Seminar Education Services*
Special Financial Seminar programs for professional societies, groups & institutions

Exclusive Marketing Agents For:

Pro-Tech Practice Evaluations	*National Association of Doctors (NAD)*
Low-cost Expanded Option Analysis, including 180 values for your practice.	Exclusive agents in Eastern PA, NJ and CT, providing low-cost high quality financial services.

YES: I would like to know more about Professional Financial Services. Please send me a free brochure.

Name ____________________

Address ____________________

ty ____________ State ________ Zip ________

Sarantos & Company, Inc. 240 Cedar Knolls, Rd., Suite 310
Cedar Knolls, NJ 07927 (201) 539-4000

— *OR CALL 1-800-223-0164* —

The principals of Sarantos & Company, Inc., a Registered Investment Advisor, are also representatives of Integrated Resources Equity Corp. Member NASD & SIPC, and a registered broker dealer

ORTHOPEDISTS

Tired of the hassles of practice?
Tired of the malpractice burden?

I need two additional full-time orthopedists to join our 15 man group. One for our facility in Syracuse, and one for Rochester.

We do evaluations only, no treatment.

Banker's hours, pleasant work, good remuneration. No nights or weekends.

If you are ready to give up surgery for a more placid life, send your C.V. to me:

Harvey Lewis, M.D.
3155 G. Eggert Road
Tonawanda, NY 14150

In New York City, for the Newest and Finest Medical Space, Remember:

THE 4 ESSENTIAL REQUIREMENTS

1. LOCATION!
Liberty Residences at Battery Park City

2. LOCATION!
Windsor Pavilion
(3rd Avenue & 32nd Street)

3. LOCATION!
205 East 64th Street (3rd Avenue)

AND

4. LOCATION!
Normandie Pavilion
(3rd Avenue & 95th Street)

For Further Information Contact Lester Schwalb
(212) 708-0846
Milstein Properties Corp.

Essential reading for every physician

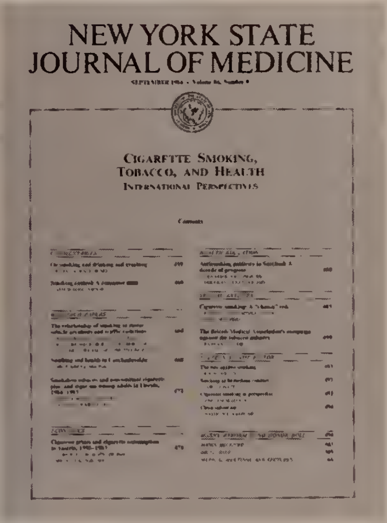
NEW YORK STATE
JOURNAL OF MEDICINE

THE WORLD CIGARETTE PANDEMIC

After a man's heart...
...when smokers find out the good things Chesterfields give them Nothing else will do

NEW YORK STATE
JOURNAL OF MEDICINE

THE WORLD CIGARETTE PANDEMIC
Part II


NEW YORK STATE
JOURNAL OF MEDICINE

CIGARETTE SMOKING,
TOBACCO, AND HEALTH
INTERNATIONAL PERSPECTIVES

Contents

The December 1983, July 1985 and September 1986 issues of the **New York State Journal of Medicine** constitute the most comprehensive coverage on the subject of smoking ever published by a medical journal.

SEND A COPY TO COLLEAGUES AND MEDICAL STUDENTS in the US and abroad who might not normally receive the Journal. Or order an extra copy for your patients and yourself.

Extra press runs have enabled the New York State Journal of Medicine to offer single or bulk copies of its issues on smoking. As the official publication of the Medical Society of the State of New York since 1900, the Journal is one of the foremost independent peer-reviewed general medical journals in the country. Providing timely and stimulating commentary, challenging research papers, analysis of clinical trends, debate on topical issues, and in-depth coverage of medical news, the Journal makes important reading—and a welcome gift—to physicians and students in the United States or abroad. A free copy of the July 1985 issue will be included with each subcription.

The Following, Reduced Rates Are Now Being Offered On The Smoking Issues

Quantity	**Per Copy***
1–24	$4.00
25–49	$3.50
50–99	$3.00
100–149	$2.50
150–500	$2.00

ORDER FORM

Name____

Address______________________________________

City/State/Zip_________________________________

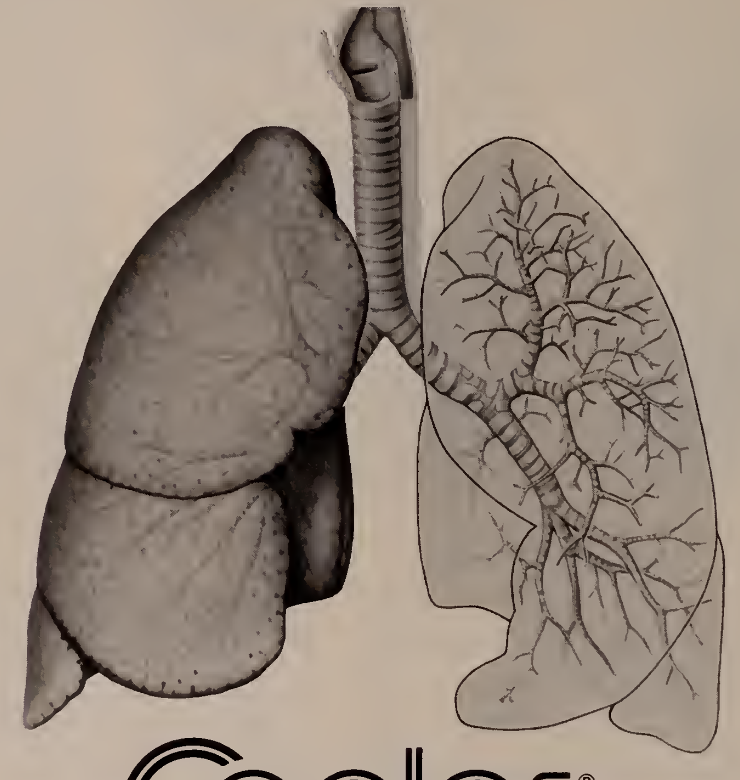
Please send _______ copies of December 1983, _______ copies of July 1985, _______ copies of September 1986 issue of the Journal.

Enter my subscription to the Journal. ☐ $25.00 U.S. & Canada; ☐ $33.00 Foreign

Total amount enclosed $ _________ ***Note:** Domestic postage included. Please add New York state & local sales tax where applicable, or sales tax exemption certificate. Foreign postage additional.

Mail to: Circulation Department, **New York State Journal of Medicine**
420 Lakeville, Rd., P.O. Box 5404, Lake Success, N.Y. 11042.

Consider the causative organisms...



Ceclor®
cefaclor

250-mg Pulvules® t.i.d.
offers effectiveness against the major causes of bacterial bronchitis

Haemophilus influenzae, Streptococcus pneumoniae
(ampicillin-susceptible and ampicillin-resistant)

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

Ceclor® (cefaclor)

Summary. Consult the package literature for prescribing information.

Indications: Lower respiratory infections, including pneumonia, caused by susceptible strains of *Streptococcus pneumoniae, Haemophilus influenzae*, and *Streptococcus pyogenes* (group A β-hemolytic streptococci).

Contraindication:
Known allergy to cephalosporins.

Warnings:
CECLOR SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS.

Administer cautiously to allergic patients.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea. Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.

Precautions:
- Discontinue Ceclor in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of nonsusceptible organisms.
- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- Ceclor should be administered with caution in the presence of markedly impaired renal function. Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk. Exercise caution in prescribing for these patients.

Adverse Reactions: (percentage of patients)

Therapy-related adverse reactions are uncommon. Those reported include:
- Gastrointestinal (mostly diarrhea): 2.5%.
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
- Hypersensitivity reactions (including morbilliform eruptions, pruritus, urticaria, and serum-sickness-like reactions that have included erythema multiforme [rarely, Stevens-Johnson syndrome] or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever): 1.5%; usually subside within a few days after cessation of therapy. Serum-sickness-like reactions have been reported more frequently in children than in adults and have usually occurred during or following a second course of therapy with Ceclor. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.
- Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.
- As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely.
- Rarely, reversible hyperactivity, nervousness, insomnia, confusion, hypertonia, dizziness, and somnolence have been reported.
- Other: eosinophilia, 2%; genital pruritus or vaginitis, less than 1%; and, rarely, thrombocytopenia.

Abnormalities in laboratory results of uncertain etiology
- Slight elevations in hepatic enzymes.
- Transient fluctuations in leukocyte count (especially in infants and children).
- Abnormal urinalysis; elevations in BUN or serum creatinine.
- Positive direct Coombs' test.
- False-positive tests for urinary glucose with Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (glucose enzymatic test strip, Lilly).

[072886R]

PA 8794 AMP

©1987, ELI LILLY AND COMPANY

Lilly

Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285.

Eli Lilly Industries, Inc
Carolina, Puerto Rico 00630

700241

Residents / Fellows

A $500 AWARD will be presented by the Medical Society of the State of New York to the intern, resident or fellow who submits the best paper on a clinical subject for the Society's 1987 Merit H. Cash Competition.

Entrants need not be members of the MSSNY but must be in training at a hospital within the State of New York.

Entries should be "sponsored" by a faculty member, and all papers submitted should be of sufficient quality to be considered for original publication in the *New York State Journal of Medicine.*

The deadline for receipt of entries has been extended to December 1, 1987, and the prize for the winning paper will be awarded at the MSSNY's annual meeting in April of 1988.

Address all entries to: Division of Scientific Activities
Medical Society of the State of New York
420 Lakeville Road
P.O. Box 5404
Lake Success, NY 11042

Questions? Contact either the Division of Scientific Activities or MSSNY's Resident Physicians Section at (516) 488-6100.

1988 ANNUAL CME ASSEMBLY

Medical Society of the State of New York
The New York Hilton, New York City

Newer Approaches to Habit Abuse and Dependency

FRIDAY, APRIL 22

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Plenary Session: Human Abuses Through the Ages: "From the Womb to the Tomb," *Sutton South/Regent Parlor*
2:00 p.m. - 5:00 p.m.	Women's Medical Society/Occupational Health, *Gramercy B*
	Cardiology, *Sutton South*
	Physical Medicine & Rehabilitation/Family Practice/Orthepedics, *Gramercy A*
	Emergency Medicine/Critical Care, *Murray Hill B*
	Pathology/Pediatrics, *Bryant Suite*
	General Surgery/Radiology, *Regent Parlor*
	Neurosurgery, *Murray Hill A*
	Medical Liability Mutual Insurance Co., *Rendezvous Trianon*

SATURDAY, APRIL 23

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Internal Medicine (All Day), *Regent Parlor*
	Ophthalmology (All Day), *Sutton South*
	Hand Surgery/Neurology (All Day), *Sutton Parlor North*
	Psychiatry (All Day), *Gramercy B*
	Urology/Radiology, *Nassau A*
	Dermatology, *Murray Hill A*
2:00 p.m. - 5:00 p.m.	Internal Medicine, *Regent Parlor*
	Ophthalmology, *Sutton South*
	Hand Surgery/Neurology, *Sutton Parlor North*
	Psychiatry, *Gramercy B*
	Chest Diseases/Otolaryngology, *Murray Hill B*
	Computers in Medicine, *Nassau A*

SUNDAY, APRIL 24

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Allergy & Immunology, *Gramercy B*
	Obstetrics and Gynecology, *Murray Hill A*
	Gastroenterology/Colon and Rectal Surgery, *Nassau A*
	Orthopedics/Family Practice/Physical Medicine & Rehabilitation/Anesthesiology, *Regent Parlor*
	Alcoholism, *Sutton Parlor Center*
	Sleep Disorders—Apnoea, *Nassau B*

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis. The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians.

◆

Technical Exhibits

NEW YORK STATE
JOURNAL OF MEDICINE

October 1987 • Volume 87, Number 10

COMMENTARIES

The myth called Medicare

In 1965 Lyndon Johnson created Medicare. At that time he said, "No longer will older Americans be denied the healing miracle of modern medicine. No longer will illness crush and destroy the savings they have so carefully put away over a lifetime so that they might enjoy dignity in their later years."[1] Since 1965 a half a trillion dollars have been spent on Medicare. Medicare spending has grown from $1 billion in 1966 to $70 billion in 1985. This sum, however, pays less than half the total health care costs of its beneficiaries.

The elderly population has been lulled into believing that the Medicare program will provide quality care compatible with their ability to pay at a time in life when their resources are limited. Few understand that under the rules as currently constituted the Medicare beneficiary must pay $492 deductible for each period of hospital confinement; pay $123 for each day of hospitalization from the 61st through the 90th day; pay $246 for each day of hospitalization from the 91st through the 150th day; pay all costs after 150 days; pay an additional $75 deductible each year for certain other covered services; pay at least 20% of all surgeons' and physicians' fees as well as fees for certain supplies and services; and pay any out-of-hospital nursing charges, as well as charges for private duty nursing.

Older people with an income of less than $10,000 a year can expect to spend one half of this on health care, most of it on long-term care. Seventy percent of the elderly believe that Medicare covers long-term nursing home care. The fact is that only Medicaid provides extended nursing home coverage, and only the impoverished qualify. The cost of nursing home care averages $20,000 a year. Obviously there is a need for long-term care insurance, but insurance companies are only now testing the water. Remember, too, that the well-to-do are not immune from catastrophe, since one third of all couples and two thirds of all single people who move into nursing homes will become impoverished within 13 weeks. The federal bureaucrats responsible for operating this program are forcing the elderly with major illnesses and disabilities to be further harassed by painful hospital bills and the debt collection process as the bureaucrats attempt to remove what is left of their meager income. The most outrageous proposal to surface recently is a plan advocated by the Inspector General of the US Department of Health and Human Services. Under this plan, sums would be deducted from the monthly Social Security checks of Medicare beneficiaries who are unable to pay their share of Medicare hospital debts.

The Great Society programs are today in serious financial trouble, and most economists predict bankruptcy by early 1990. In an attempt to hold down costs, in 1983 the Congress put together a legislative program for changing the way hospitals are paid for the care of Medicare patients.[2] The program is called a prospective payment system (PPS), and its key ingredient is diagnosis related groups (DRGs). New York State was a late entry into the program, which has been in effect here only since January 1, 1986. Before PPS, hospitals were reimbursed on a fee-for-service basis. Payment depended upon length of stay and any ancillary services the patient received. With PPS the reimbursement rates have been predetermined for 468 different diagnoses ranging from hip fractures to cataract surgery. Factors that establish a patient's DRG include the diagnosis and surgical procedures performed, any complications or comorbidities, and the patient's age and sex. If the hospital uses more resources than are allocated for a specific DRG, it will lose money. If the hospital uses fewer resources, it will realize a savings. It is because of this that there is anxiety on the part of the Medicare population that they will be catapulted out of the hospital doors prematurely. A Senate Special Committee on Aging has investigated a large number of alleged premature patient discharges and the problem was found to be more severe and widespread than the Health Care Financing Administration estimates had indicated.[3]

The prospective payment system, by reason of both the DRGs and the current peer review quality assurance program, is causing fear and misunderstanding among the elderly population. As physicians become aware of the inequities of Medicare, they will be better able to work in concert with those groups as they try to develop a more responsive and higher quality health care system.

It appears that the DRGs can cut costs, but at the expense of quality of care for our older Americans. It seems that senior citizens are being abandoned to fend for themselves in a health care system that is inadequately prepared to care for them. Financially efficient hospitals are rewarded and those that exceed the target DRG payment are penalized, hence the emphasis on early discharge. The elderly must "get well or get lost."

To guard against unnecessary expense and to guarantee quality health care, the government created monitoring and review agencies called peer review organizations (PROs). They have been in place since 1972 and employ hundreds of physicians and nurses, but until recently they have been more concerned with reducing lengths of stay than with increasing quality of care. There is general agreement that PROs have effected a decline in hospital admissions—4.0% since the PROs began their reviews.[4] In 1982 there was no change in the Medicare admission rate, and there was only a 0.5% decline in 1983. There has also been a continued decline in the average length of stay for patients over age 65, which measured 7.6% between 1983 and 1984. Critics emphasize, however, that this concentration on shortening stays and avoiding unnecessary admissions has been at the expense of quality concerns. The PROs have until recently addressed quality by a contractual agreement to concentrate on a selected number of surgical and medical quality objectives. These have proven to be restrictive and unimaginative, and lack any baseline data to validate the existence of quality problems. Health Care Financing Administration officials have agreed to a new concept using "generic screens" to identify quality and to develop standards for measuring it. This calls for screening charts for a variety of quality indicators, such as hospital-acquired infection, unscheduled return to the operating room, abnormal temperature or blood pressure on discharge, and so on. With the emphasis on reduction of admissions and a shift towards ambulatory care, many feel the scope of PRO review is too narrow. PROs ought to examine what happened before the patient entered and after he left the hospital. A test of this approach will come through PRO review of the care Medicare patients have received in health maintenance organizations (HMOs). The PRO should investigate deaths occurring at home associated with a hurried hospital discharge as well as medical complications, including death, brought about by denial of hospital admission. Other areas which should rightfully be subjects for PRO review are instances of failure to prepare patients for home care as well as the reliability of home health services. Frequently patients require more extensive care and supervision than many of these agencies can provide. Quality of care problems resulting from inadequate outpatient care could be identified if all admissions for ruptured appendix, gangrene, cellulitis, digitalis toxicity, diabetic acidosis, perforated ulcers, etc, were retrospectively analyzed. Finally, the PRO should be funded to undertake studies to explain the extreme variations in admission, length of stay, and cost of hospitalization that occur in this country today for identical disease processes. We must explain variations in rates of hernia repair, hysterectomy, hemorrhoidectomy, pneumonia, and prostatectomy—the diseases are identical but the disparity in incidence rates and patient fee statistics is shocking! We must narrow this range of variation in practice and to do this we need research aimed at establishing a consensus within the medical profession with respect to appropriate care. We must be certain that these variations are the result of sound medical judgments formed by the most thorough analysis of the outcomes of treatment alternatives.

Vita Ostrander,[5] past president of the American Association of Retired People (AARP), has stressed that the Gramm-Rudman spending cuts, $36 billion annually until the budget is balanced, will have profound effects on the elderly people who are trying to live on a moderate income. The elderly wonder how the Gramm-Rudman amendment will change what they can expect to pay for health care when they are ill, or what will happen when their housing subsidies are cut, their telephone bills go up, or their community support services are reduced. Will hospitals discharge them prematurely under PPS? Can skilled nursing facilities and home health agencies provide the needed services under existing guidelines? Will HMOs and competitive medical plans (CMPs) assure access to and provide for quality care? Concerns that the elderly will be deinstitutionalized are receiving increasing publicity, since deinstitutionalization carries the risks of increased death rates, more postoperative infections, and an overall deterioration in the quality of care provided. The AARP has been supportive of the PROs, and has called for comprehensive uniform data about providers, particularly hospitals and physicians. They, too, are demanding a reevaluation of PRO quality objectives along the lines outlined above. The AARP is also requesting a smoothing out of variations in physicians' practice patterns and a mandate to review outpatient services. It should be emphasized that the population of Medicare recipients has a very limited appeals process, namely, the PRO intervention. There is no access to the courts, and at present the providers do not have the right to represent their patients. It is also obvious that the elderly often do not have the stamina and will to see an appeals process all the way through.

Does the future provide any good news for change in the health care system as it serves the geriatric population? Every year we witness changes in the way Medicare and Medicaid are financed and managed. Leaders in the health economy field tell us that Medicare's health insurance fund, which pays hospital costs for the elderly and disabled, will be bankrupt by 1990 and possibly earlier.

Reforms are needed not only to control costs but to correct serious drawbacks in both Medicare and Medicaid. Much of Medicare's resources are spent on expensive hospital care and sophisticated techniques to treat patients in the last phase of a terminal illness. Some 9% of the elderly consume 70% of Medicare's funds. Medicare's most serious flaw is its lack of coverage for nursing home care and rehabilitative treatment for chronic disease, even though 80% of problems affecting the elderly on Medicare are long-term ailments such as diabetes, stroke, or arthritis.

In 1984, former Colorado governor Richard Lamm, speaking at a meeting of the Colorado Health Lawyers

Association, said the terminally ill elderly "have a duty to die." He said, "We are approaching the day when medical technology can keep people alive in hospitals hooked up to tubes and things far beyond when any quality of life is left at all." On the question of how many heart transplants the nation can afford, Lamm said that the elderly should "get out of the way . . . and let the other society—our kids—build a reasonable life."[6]

Riddick[7] has called attention to the role of rationing in some countries, where scarce resources are doled out in limited quantities. Either there is a long queue and prolonged waiting time for elective procedures, or there is simply failure to provide the service at a certain point. For example, renal dialysis is not available in Great Britain for those beyond age 55. When asked for the justification of this cutoff, a British physician said, "We all get a bit crumbly around that age, and patients of that age are not suitable candidates for dialysis."[7] He added that as more and more medical care is delivered on a price-sensitive basis, as research-based institutions are increasingly squeezed, there may not be much extension of the "gee-whiz" technology to the general public.

A *Wall Street Journal* editorial[8] suggests that since the Medicare regulatory framework is not working, we should try deregulated competition in health care. The article agrees that up until now most Medicare reforms have taken the form of price controls, which is window dressing. It suggests dismantling the entire Medicare bureaucracy and dividing the budgeted dollars among the beneficiaries as a yearly health stipend. This program would allow the Medicare population to do their own shopping and would throw the entire health care field open to the marketplace.

Payment reforms as currently practiced are certainly not the answer. Anderson and Raposa[9] point out that these result in decreased funding for new technology and graduate medical education, and in uncompensated care. The result will be a decline in the development and marketing of new technology, the size of graduate medical education programs, the amount of hospital-based research, and the number of hospitals financially willing and able to care for the elderly and indigent population. At the same time there will be more nonhospital services as they unbundle their services to maximize reimbursement. Competition will grow as providers lower their pricing with an eye to the competing market. There will follow a redistribution of wealth in the health care industry—among the hospitals, across regions of the country, between urban and rural settings, and among physician specialties. The formula would seem to be: more elderly people and more technology equals more expensive health care. It seems apparent that no one knows what the future health care sector should look like or even how to gain control over the health care system.

Physicians should be in the forefront in demanding that quality not be compromised for cost containment. They should lend their support to those organizations such as the AARP and the National Council of Senior Citizens that are struggling so hard to correct the flaws in the Medicare legislation. Finally, as Butler[10] recently pointed out, physicians must be ever vigilant against "ageism," which is defined as institutionalized and individual prejudice against the elderly, stereotyping, myth making, distaste, and/or avoidance.

JOHN T. PRIOR, MD
Medical Director
Empire State Peer Review Organization

Pathologist, Director of
Laboratories
Community-General Hospital
Broad Rd
Syracuse, NY 13215

1. Medicare in need of reform. *American Medical News* 1985; 28(33):4.
2. Tax Equity and Fiscal Responsibility Act of 1982, Pub L No. 97-248, 96 Stat 324 (codified as amended in scattered sections of 42 USC); Omnibus Legislation, 1983.
3. Staff of Senate Special Committee on Aging: *Impact of Medicare's Prospective Payment System on the Quality of Care Received by Medicare Beneficiaries*, 99th Cong, 1st sess (1985) (John Heinz, Chairman).
4. Owen J: Policy outlook PRO's: A hospital perspective. *AMPRA Review* 1985; 2:3.
5. Ostrander V: Medicare DRGs pose problems for elderly [interview by Jeffrey Finn]. *Hospitals* 1985; 59(24):75–76.
6. Lamm's "duty to die" remarks draw fire. *Hospitals* 1984; 58(9):24.
7. Riddick FA Jr: Future directions in graduate medical education. *Arch Pathol Lab Med* 1986; 110:1006–1011.
8. Wohl S: Deregulate to cure health care paralysis. *Wall Street Journal*, October 9, 1985, p 32.
9. Anderson GF, Rapoza CR: Health care payment reform: Projections for the next 15 years. *NY State J Med* 1985; 85:644–646.
10. Butler RN: Thoughts on aging. *Am J Psychiatry* 1978; 135(suppl):14–16.

A view of the proposed New York State regulations pertaining to the activities of medical students

On June 2, 1987, the Ad Hoc Committee on Emergency Services, appointed by New York State Health Commissioner David Axelrod, announced several recommendations that were subsequently inserted into draft revisions of the New York State Health Code, Section 405. In a separate position statement, the Associated Medical Schools (AMS) of New York has responded to those sections of the Ad Hoc Committee recommendations and the code revisions dealing with the work of residents and attending physicians.[1] AMS, however, is also gravely concerned with other proposed regulations in Section 405 pertaining to medical students. This position statement emphasizes these concerns.

Section 405, subsection (h), dealing with medical students, states that students may only

> observe the provision of patient care services by an appropriately licensed practitioner granted privileges pursuant to that license to practice in the hospital, but shall not be assigned or directed to provide any patient care services for which a license is required pursuant to the requirements of New York State Education Law with the exception of taking patient histories and conducting physical examinations. These can be performed only under the supervision and countersignature within twenty-four hours by at least a second year resident.

This requirement, if enacted, would result in medical students who have completed more than an entire year of clinical training assuming no more responsibility than that which had existed during their second year of medical education. Such a restriction violates nationally accepted standards for the clinical education of medical students.

Students clearly are not licensed to practice medicine and should not be allowed to function independently in any procedure for which a license is required. It is equally unacceptable, however, that students under the appropriate supervision of licensed, credentialed physicians should be unable to participate in graded clinical experiences which will, in turn, legitimize their receipt of the MD degree and prepare them for their residency. Indeed, it is a fundamental principle of the medical education experience across the continuum from medical school through residency training that students and residents have gradually increasing responsibility in order to increase their knowledge, skills, and experience under appropriate supervision.

One of the most valuable aspects of medical school is the senior year clinical experience during which time students, under close supervision, take night call and assist residents and the attending staff in the clinical care of patients. There has rarely, if ever, been an adverse outcome resulting from these arrangements. When isolated events have occurred, they have been the result of inadequate supervision. To prevent students from assuming clinical responsibilities until they start their residencies will ill serve the public as well as our students. A more intense and prolonged training period will be needed in order to allow residents to progress to levels of achievement and responsibility which are now typical of medical school graduates under our current system of medical education. The result will be increased costs and less adequate care of patients by less well prepared graduates. These problems will be compounded if the medical schools of New York—as a result of these new restrictions—become less attractive to top quality candidates from New York and elsewhere.

The Associated Medical Schools strongly urges that this subsection be revised to allow medical students to engage in such activities and perform those procedures deemed appropriate by national standards and by the schools of medicine under careful supervision and, when appropriate, under direct observation of licensed and appropriately credentialed physicians.

AMS recognizes the valid concerns about proper supervision. Its members will continue to review current procedures and work with appropriate public and private agencies to assure the best possible supervision of students.

ASSOCIATED MEDICAL SCHOOLS OF NEW YORK
Bellevue Hospital Center
First Ave and 27th St
New York, NY 10016

1. *Position Statement on Proposed Regulations Governing the Activities of Residents.* New York, Associated Medical Schools of New York, August 3, 1987.

The "exaggerated death" of peritoneal lavage in the management of trauma patients

In this issue of the *Journal*, Barker and Fromm[1] present a patient with an unusual sequela—bile peritonitis—from a blunt trauma injury to the liver. The patient survived and is doing well.

A measure of success in the treatment of any patient is the restoration of wellness, which in this instance was achieved. Given even the best of descriptions, it is generally not possible to give a reader or listener an exact feel for a clinical scenario. While adhering to basic principles in the resuscitation, evaluation, and treatment of trauma patients, clinicians vary their management styles according to available personnel, equipment, and clinical experiences.

The management of the patient presented here illustrates an evolutionary change in the diagnosis and treatment of patients with blunt torso trauma. Just over two decades ago, the diagnosis of this patient's liver injury would have been made by four-quadrant abdominal paracentesis or by performing an exploratory laparotomy. The first of these diagnostic modalities is of historic interest, having been replaced and incorporated in part in the performance of a formal diagnostic peritoneal lavage. Now, with the growing acceptance of computed tomography (CT) scanning as a diagnostic method, the role of peritoneal lavage is undergoing scrutiny.

At the Shock Trauma Center of the Maryland Institute for Emergency Medical Services Systems in Baltimore, diagnostic peritoneal lavage is used liberally to diagnose intra-abdominal injury. Collectively, the center's trauma surgeons have employed the test several thousands of times over the past two decades to assess intra-abdominal injury. The once-favored, classic open method described by Root and colleagues in 1965[2] has given way to a semi-open technique developed by Cox and Dunham[3] at our center.

As in many other centers, lavage is performed in our center in patients with altered sensoriums, including patients with trauma due to head injury and/or drug use (usually alcohol); patients with multiple trauma (particularly above and below the diaphragm); patients with significant abdominal pain and/or tenderness without distention, who have stable vital signs; and patients who must undergo prolonged surgical procedures to treat nonabdominal injuries.

Using quantitative criteria to interpret lavage results, primarily a red blood cell (RBC) count of ≥100,000/mm³, Fischer and colleagues[4] noted false-negative and false-positive results of 1.2% and 0.2%, respectively, among 2,586 patients who underwent lavage for suspected intra-abdominal injury. In a report from our center, Cox[5] noted slightly higher false-negative and false-positive results of 0.3% and 3.3%, respectively, among 640 patients with intra-abdominal injuries. The criterion for exploration in those patients was a red blood count of ≥50,000/mm³.

After his motorcycle crashed, the patient described by Barker and Fromm[1] was admitted with vague right upper quadrant pain and mild tenderness. His hematocrit was low (37%). Given this scenario, some would have been more aggressive in diagnosing intra-abdominal injury, often within the first hours after admission. In the past, surgeons would have performed peritoneal lavage shortly after admission. Now, some would evaluate the abdomen using CT scanning. If lavage had been performed, a positive result would have been obtained in one of three ways: spontaneous drainage of blood through the lavage catheter on its insertion, aspiration of gross blood prior to performing the lavage, or lavage effluent containing gross blood. This would have led to the performance of an exploratory laparotomy and discovery of the liver injury. Most likely, the bleeding would have stopped or been minimal and easily controlled by compression of the organ (manually or by placing gauze packs about the right lobe for 10–15 minutes). The abdomen would have been closed without drains.[6,7] The biliary problem would have become apparent days later, leading to a second laparotomy.

Barker and Fromm adopted a less aggressive style of management in their young patient with minimal abdominal signs and symptoms, normal vital signs, and a low hematocrit. The patient was admitted and carefully monitored with the performance of repeated abdominal examinations and the tracking of serial vital signs and hematocrit determinations. Twenty-four hours after admission, a CT scan of the abdomen was obtained, which demonstrated the liver defect. If the patient had been transferred to this center at that time, with stable vital signs and having required only two units of blood, most of the clinicians here would have continued to manage the patient in a cautiously expectant fashion, as was done. If this rather unusual complication had not developed, he would have had no abdominal surgery. As it turned out, he had one surgical procedure compared to the two he would have had if he had undergone an early peritoneal lavage.

The report of Barker and Fromm[1] contributes to the growing body of literature that is helping to define the role of abdominal CT scans in the diagnosis and treatment of patients who sustain blunt torso trauma. Reports extolling the virtues of CT scanning in diagnosing intra-abdominal injuries continue to increase,[8-10] noting the test's ability to detect bleeding from liver injuries that do not require surgery.[11] The reports show that, in contrast to peritoneal lavage, enhanced CT studies can usually delineate specific organ injuries with great clarity. Trunkey and Federle[10] commented that the achievement of acceptable results with CT requires high-quality studies and the immediate review of equivocal studies by a staff radiologist with expertise in reading such scans.

The enthusiasm of some clinicians concerning the role of CT scanning in the diagnosis of abdominal injuries may be summarized by Trunkey and Federle's[10] editorial statement: "CT scanning is a different modality [compared to peritoneal lavage] and represents the next revolutionary step in [the] diagnosis of trauma." Is it time to shelve peritoneal lavage? To answer this question one should consider a previous statement by Blaisdell and Trunkey.[12] They noted, "We remain surprised at the reluctance of surgeons to explore the abdomen of a traumatized patient and at the same time readily accept a negative exploration rate of 20–30% for appendicitis as representing good practice." [12] This statement alludes to a cardinal principle of trauma care: one must be aggressive in identifying potentially life-threatening injuries. The introduction of peritoneal lavage has been a major advance in the management of trauma victims. As indicated, using quantitative criteria for interpretation, results greatly surpassing the standard for appendicitis have been achieved.

Currently, there is no single test without associated morbidity that is 100% sensitive and specific in detecting all intra-abdominal injuries. The introduction of peritoneal lavage allowed for a major leap forward to be made in the rapid diagnosis of life-threatening injuries. The use of CT allows for another great advance in trauma care: a reduction in the incidence of nontherapeutic laparotomies. One should remember, however, that it is neither practical nor safe to interrupt the resuscitation/evaluation of some patients by moving them to the CT scanning suite. Among critically ill patients, a quickly performed peritoneal lavage will indicate whether emergency surgery is necessary. Not uncommonly, surgery is avoided in patients who appear to need a laparotomy. An obsession with avoiding nontherapeutic laparotomies can lead to the sin of omission in the treatment of a life-threatening injury. While one does not have to apologize for an occasional nontherapeutic laparotomy, one must seek forgiveness for missing an easily identifiable life-threatening injury.

When a considerable number of clinicians have gained much needed experience with CT scanning of the abdomen in the management of trauma patients, one will then appropriately see a significant reduction in the performance of peritoneal lavage. Clinical expertise with CT scanning is not sufficiently advanced among clinicians who treat trauma victims.

Mark Twain once cabled from Europe, "The report of my death was an exaggeration." Indeed, the demise of peritoneal lavage should be considered the same.

CARL A. SODERSTROM, MD
PHILIP R. MILITELLO, MD
University of Maryland
School of Medicine
Department of Surgery/Traumatology
Maryland Institute for Emergency
Medical Services Systems
22 S Greene St
Baltimore, MD 21201

1. Barker SL, Fromm D: Bile peritonitis following expectant management of liver fracture. *NY State J Med* 1987; 87:565-567.
2. Root HD, Hauser CW, McKinley CR, et al: Diagnostic peritoneal lavage. *Surgery* 1965; 57:633-637.
3. Cox EF, Dunham CM: A safe technique for diagnostic peritoneal lavage. *J Trauma* 1983; 23:152-154.
4. Fischer RP, Beverlin BC, Engrav LH, et al: Diagnostic peritoneal lavage: Fourteen years and 2,586 patients later. *Am J Surg* 1978; 136:701-704.
5. Cox EF: Blunt abdominal trauma: A five-year analysis of 870 patients requiring celiotomy. *Ann Surg* 1984; 199:467-474.
6. Dunham CM, Militello P: Surgical management of liver trauma. *Am Surgeon* 1982; 48:435-439.
7. Fischer RP, O'Farrell KA, Perry JF Jr: The value of peritoneal drains in the treatment of liver injuries. *J Trauma* 1978; 18:393-398.
8. Federle MP, Crass RA, Jeffrey RB, et al: Computed tomography in blunt abdominal trauma. *Arch Surg* 1982; 117:645-650.
9. Goldstein AS, Sclafani SJ, Kupferstein NH, et al: The diagnostic superiority of computerized tomography. *J Trauma* 1985; 25:938-946.
10. Trunkey D, Federle MP: Computed tomography in perspective [editorial]. *J Trauma* 1986; 26:660-661.
11. Federle MP: Computed tomography of blunt abdominal trauma. *Radiol Clin North Am* 1983; 21:461-475.
12. Blaisdell FW, Trunkey DD: Preface, in Blaisdell FW, Trunkey DD (eds): *Trauma Management. Vol I: Abdominal Trauma.* New York, Thieme-Stratton, 1982.

Nonoperative management of blunt abdominal trauma: The cost/benefit margin

The appropriate utilization of computed tomography (CT) in the management of blunt abdominal trauma is currently at issue. This radiologic instrument allows visualization of the retroperitoneal as well as intraperitoneal spaces and may help identify specific organ injury, particularly to solid viscera. Formerly, diagnostic peritoneal lavage (DPL) was almost exclusively relied on to determine the need for urgent celiotomy following both blunt and penetrating injuries. Numerous reports attest to the exceptional accuracy of DPL in detecting or excluding intraperitoneal hemorrhage.[1-4] However, DPL's sensitivity for even small amounts of blood can lead to unnecessary surgery in as many as 12% of patients in whom DPL is the sole diagnostic criterion.[1,4] At laparotomy, these patients usually have self-limited injury to the liver or spleen. CT enthusiasts argue that in selected patients this consequence can be avoided with serial CT examination and clinical observation. But careful application of CT is required to avoid more serious complications. This is true for the acute as well as convalescent phases of trauma management; the report by Barker and Fromm[5] in this issue of the *Journal* underscores this fact.

CT accuracy is highest for trauma to solid viscera and is reported to exceed 95% for splenic injury.[6,7] But these exemplary results have only been achieved in uncontrolled, retrospective series. In three prospective comparisons of DPL and CT following blunt trauma, this success was not duplicated.[8-10] In fact, CT was only 40-60% sensitive in the detection of injury mandating operative intervention, compared with a 90-100% accuracy rate for DPL. In these and other reports, CT has failed to demonstrate a substantial number of liver injuries that required hepatotomy for direct suture control of bleeding, parenchymal debridement, and perihepatic drainage.[11-13] Moreover, hemoperitoneum in excess of 500 cc has escaped detection by CT. This may have particular significance in hepatic trauma, in which the quantity of hemoperitoneum seen on CT may be a decisive factor for laparotomy. False-positive CT studies of hepatic injury have also been reported.[11] Scintigraphy provides an alternative to CT in the assessment of solid organ injury. It has achieved excellent results in direct comparison to CT and has the advantage of being better able to assess the agitated patient.[13] However, its scope is limited to visualization of the liver, spleen, and kidneys.

Apparent discrepancies in the accuracy of CT can be attributed to differences in patient selection, scanning equipment, and institutional experience. Of greater significance, patient safety concerns and logistical difficulties can arise with untimely CT usage. Experienced trauma personnel must be available to monitor the patient throughout the obligatory 45 to 60 minutes required to execute the basic study. The location of the CT suite and the need to provide ongoing skilled patient care can be restrictive. The inconsistent availability of technical personnel, radiologists expert with interpretation, and the scanner itself can be limiting. The uncooperative patient will further compromise accuracy and may require sedation, with its attendant risks. Finally, the necessary administration of a relatively large volume of oral as well as intravenous contrast-enhancing agents has its own risks. Even under optimal conditions, reliance on CT as the primary triage tool for blunt abdominal trauma has incurred an unnecessary laparotomy rate of 19%.[6] It is imperative that institutions research their own resources and capabilities.

Expectant management of hepatic trauma mandates careful consideration. The liver is the intraperitoneal organ most frequently injured following blunt trauma; coincident damage to remaining intra-abdominal viscera is frequent. Pancreatic ductal disruption or hollow visceral perforation can be life-threatening, yet poorly appreciated by CT. Moreover, liver complications of recurrent bleeding, hematobilia, bile leakage, and abscess formation can ensue, further obfuscating clinical evaluation. Whether nonoperative treatment of liver injuries is cost effective is not known. Considerable clinical and laboratory monitoring together with multiple radiographic studies are required to optimize patient safety. Clearly, a single patient death due to delay in operation will nullify the cost savings of innumerable, unrewarding abdominal explorations. The paradox is that the patient in whom CT-directed expectant management is most appropriate is the individual in whom the morbidity associated with delayed laparotomy is negligible.

Clinical criteria for nonoperative management of liver injuries have been defined at the San Francisco General Hospital.[14] Patients who sustain blunt abdominal trauma, are hemodynamically stable, and have no other indications for laparotomy are candidates for CT evaluation. Observation is permissible if CT reveals a simple parenchymal laceration or intrahepatic hematoma without continued hemorrhage, and if the amount of intraperitoneal blood is less than 250 cc as estimated by restriction to the Morison pouch. In a series of 24 patients who fulfilled these guidelines, none required laparotomy or transfusions for hepatic injuries, and no complications developed. Notably, these 24 patients represent only 6% of the liver injuries evaluated during the 30-month period covered by this study.

In summary, DPL rapidly and accurately discerns the presence of intraperitoneal hemorrhage in patients who suffer blunt injury. A small number of such patients with minor liver or spleen lacerations may be subjected to unnecessary operation based on DPL results alone. However, this occurs in an acceptably low percentage. DPL also measures leukocytic and enzymatic response, reflecting damage to other intra-abdominal organs that are less well defined by CT. Conversely, the qualities of CT and scintigraphy make these diagnostic aids suitable for managing the stable patient with suspected, isolated hepatosplenic injury. In the level I patient, CT can be a valuable adjunct to DPL, but its inconsistent record and logistic problems speak against its being the sole instrument used for decision making.

JOHN A. MARX, MD
ERNEST E. MOORE, MD
Denver General Hospital
University of Colorado
Health Sciences Center
Denver, CO 80204

1. Fischer RP, Beverlin BC, Engrav LH, et al: Diagnostic peritoneal lavage: Fourteen years and 2,586 patients later. *Am J Surg* 1978; 136:701-704.
2. Parvin S, Smith DE, Asher WM, et al: Effectiveness of peritoneal lavage in blunt abdominal trauma. *Ann Surg* 1975; 181:255-261.
3. Powell DC, Bivins BA, Bell RM: Diagnostic peritoneal lavage. *Surg Gynecol Obstet* 1982; 155:257-264.
4. Thal ER, Shires GT: Peritoneal lavage in blunt abdominal trauma. *Am J Surg* 1973; 125:64-69.
5. Barker SL, Fromm D: Bile peritonitis following expectant management of liver fracture. *NY State J Med* 1987; 87:565-567.
6. Federle MP, Crass RA, Jeffrey RB, et al: Computed tomography in blunt abdominal trauma. *Arch Surg* 1982; 117:645-650.
7. Jeffrey RB, Laing FC, Federle MP, et al: Computed tomography of splenic trauma. *Radiol* 1981; 141:729-732.
8. Davis RA, Shayne JP, Max MH, et al: The use of computerized axial tomography versus peritoneal lavage in the evaluation of blunt abdominal trauma: A prospective study. *Surgery* 1985; 98:845-850.
9. Fabian TC, Mangiante EC, White TJ, et al: A prospective study of 91 patients undergoing both computed tomography and peritoneal lavage following blunt abdominal trauma. *J Trauma* 1986; 26:602-608.
10. Marx JA, Moore EE, Jorden RC: Limitations of computed tomography in the evaluation of acute abdominal trauma: A prospective comparison with diagnostic peritoneal lavage. *J Trauma* 1985; 25:933-937.
11. Moon K Jr, Federle MP: Computed tomography in hepatic trauma. *AJR* 1983; 141:309-314.
12. Toombs BD, Sandler CM, Rauschkolb EN, et al: Assessment of hepatic injuries with computed tomography. *J Comput Assist Tomogr* 1982; 6:72-75.
13. Uthoff LB, Wyffels PL, Adams CS, et al: A prospective study comparing nuclear scintigraphy and computerized axial tomography in the initial evaluation of the trauma patient. *Ann Surg* 1983; 198:611-616.
14. Meyer AA, Crass RA, Lim RC, et al: Selective nonoperative management of blunt liver injury using computed tomography. *Arch Surg* 1985; 120:550-554.

Joseph F. Shanaphy, MD

Joseph F. Shanaphy, MD, Vice-President of the Medical Society of the State of New York (MSSNY), died on Friday, July 10, 1987, after a brief illness. He had been elected Vice-President of the Society at the 1987 House of Delegates meeting in New York City in March, the same month he retired as director of the Department of Obstetrics and Gynecology at St Vincent's Medical Center of Richmond County. Dr Shanaphy had served in the latter post for ten years and had practiced obstetrics and gynecology on Staten Island since 1952.

Dr Shanaphy, who was born in 1921 in West Hoboken, New Jersey, received a BS degree from the College of the Holy Cross and went on to study medicine at the Long Island College of Medicine, from which he received an MD degree in 1945. He did his internship and residency in obstetrics and gynecology at the US Public Health Service Hospital on Staten Island, and later attended the New York University Basic Science Post-Graduate School, from which he received an MSc degree in 1951. He served as a Senior Surgeon in the USPHS from 1945 to 1953, spending part of this time as Chief of Obstetrics and Gynecology at the US Public Health Service Hospital in New Orleans.

Dr Shanaphy was active in organized medicine, serving as the President of the Richmond County Medical Society in 1964. In 1975 he was elected Vice-Speaker of the MSSNY House of Delegates, and was speaker from 1976 to 1981. Subsequently he was a member of the MSSNY Board of Trustees from 1981 until his election as Vice-President. He was an alternate delegate to the American Medical Association House of Delegates in 1976 and 1977, and from 1979 to 1981, and a full delegate from 1982 until his death. Dr Shanaphy was widely known and respected in his specialty. He served in the departments of obstetrics and gynecology at several hospitals, including Richmond Memorial Hospital and St Vincent's Medical Center in Staten Island and New York University Hospital in Manhattan. He was also a clinical instructor in obstetrics and gynecology at New York University-Bellevue Hospital Center. Dr Shanaphy is survived by his wife, Ellen, and four children, Jean, Nancy, Patricia, and Joseph, Jr.

Pascal James Imperato, MD
Editor

"The Board of Trustees of the Medical Society of the State of New York wishes to express its deep sorrow at the untimely passing of the Society's Vice-President, Joseph F. Shanaphy, MD. Dr Shanaphy served the Society and the people of New York State with compassion and skill for many years. He was greatly admired for his warm and giving personality, his intelligence, and his leadership. Dr Shanaphy was a friend to his patients and colleagues alike. Our profound sympathy goes to his widow, Ellen, and their four children."

Richard D. Eberle, MD
Chairman
Board of Trustees
Medical Society of the State of New York

"The Officers and Councilors of the Medical Society of the State of New York extend their deepest sympathy to the family of Joseph F. Shanaphy, MD, our esteemed colleague, who served the Society and the medical care interests of the people of New York State with distinction for many years. He was greatly gifted and admired by his patients, the citizens of his community of Staten Island, and by his fellow physicians. This admiration is his lasting tribute."

Samuel M. Gelfand, MD
President
Medical Society of the State of New York

Physicians' attitudes toward and knowledge about generic drug substitution

DANIEL FRIEDMAN, MD; ARNOLD JAFFE, PHD; SUSAN STEINHARDT, MPA

ABSTRACT. Recent federal and state legislation has caused considerable confusion for those who prescribe, dispense, or purchase prescription drugs. This study investigates physician understanding of and attitudes toward prescribing of generic drugs. Respondents to a survey included 245 physicians from several disciplines who were members of the Suffolk County Medical Society. Only 12.9% of respondents reported having knowledge of Food and Drug Administration procedures for testing generic drug products. Physicians are mixed in their beliefs about the efficacy of generic drugs, and the level of prescribing common generic drugs is low. A majority of respondents reported a willingness to prescribe generic drug products on a patient's request, but few such requests are made. Generic drug prescribing habits differed among the specialties included in the sample. It is recommended that information on testing and efficacy of generic drugs be provided to physicians. Perhaps such information could facilitate the decision-making process when physicians prescribe drugs.

(NY State J Med 1987; 87:539–542)

In 1938 Congress passed legislation to ensure the safety of drugs marketed in the United States.[1] In 1962, federal legislation mandated that efficacy (as well as safety) must be certified.[2] The Drug Price Competition and Patent Term Restoration Act of 1984 was intended to make prescription drugs more affordable to the public.[3] One of its primary goals was to simplify the application process by which generic drug products can gain the approval of the Food and Drug Administration (FDA). Prior to this law a New Drug Application (NDA) often required 150,000 pages of detailed documentation. The new law provides for an Abbreviated New Drug Application (ANDA), which is appropriate only for generic drug products. An ANDA does not require manufacturers to test for safety or effectiveness. They are only required to demonstrate that the generic drug product has bioequivalence of +20% or +30%, depending on the innovator product. The percentage varies by class of drug. (See the "75-75" rule below.)

The essence of the new FDA regulations includes the following:[3]

- Deriving efficacy data from 18 to 24 healthy men, ages 21 to 35, who are within 10% of their ideal body weights. Sampling of a pediatric or a geriatric population is not considered.
- Accepting a generic drug product as "pharmaceutically equivalent" if it contains the same active ingredient in the identical concentration and form as the innovator product. Excipients, defined as inert ingredients, are expected to differ.
- Testing and comparing blood concentrations of subjects in the sample. Clinical trials on patients may be required for some drugs.
- Approving the generic drug product contingent on its meeting the "75–75" bioavailability rule. The generic drug product is required, in only 75% of the test subjects, to achieve blood concentrations not less than 75% or greater than 125% of those obtained by the brand name product. (For some psychotropics, the rule is relaxed to "70–70.") Bioavailability is distinct from bioequivalence. It is defined by the Federal Registry as "the rate and extent to which the active ingredient or therapeutic moiety is absorbed from a drug product and becomes available at the site of drug action."

Passage of this new legislation coincided with the termination of patent rights on a large number of brand name drugs. There has since been a flood of ANDA applications. During the past two decades, all 50 states have passed legislation that either permits or requires pharmacists to substitute a generic drug for its brand name congener. The goal of such legislation is to enhance attempts to reduce consumer cost without sacrificing quality, reliability, or efficacy.

Legislation has led to increased confusion relating to issues of substitution policy, bioavailability, bioequivalency, and economic and fiscal impact. Twenty percent of the $17.6 billion spent for drugs during 1984 was for generic products—a figure that is expected to grow to more than $10 billion (a 25% increase) by 1995.[4,5] Though it is difficult to achieve a relative exactness of dosage of gener-

---

From the Department of Family Medicine, State University of New York at Stony Brook, Stony Brook, NY.

Address correspondence to Dr Friedman, Department of Family Medicine, Health Science Center, State University of New York at Stony Brook, Stony Brook, NY 11794-8461.

ics and innovators, it is not clear that the efficacy of generic drugs depends on such strict equivalence.[6,7] Mazullo[8] points out, in a cautionary note, that the most important determinant of drug pricing is neither the pharmaceutical manufacturer nor the physician; it is the pharmacist.

As prescriptions for generic drugs are refilled, manufacturers' products are often interchanged without the knowledge of either the physician or the patient. Both need to know the details of the laws within their state to benefit from the laws' intent and to avoid unanticipated consequences. Unfortunately, the lack of uniformity in state laws provides mechanisms by which both advocates and opponents of generic prescribing attempt to thwart the intended legislative purpose.[9]

Francetic et al[10] found wide variations in retail pharmacy prices. In nearly 30% of the cases, savings were not passed on to the customer. The additional administrative costs to state and federal governments for generic drug prescribing programs are rarely calculated into the cost of generic prescribing. Brater and Pettinger[11] question who is liable when a pharmacist "selects an equivalent" drug product as authorized by state laws. Effective July 1, 1987, New York State legislation relieves the physician of such liability.

In a study that covered 39 states and almost 900,000 prescriptions, Bloom et al[12] concluded that it is foolish to assume that generic prescribing will result in significant cost savings. Lasagna[13] believes that pharmacists favor generic drugs because their markup tends to be higher. In a concise review of these issues, Schwartz[14] delineates the dilemmas that face physicians as they try to maintain knowledge and awareness of the potential differences and dangers when substituting one drug product for another.

Do physicians understand the FDA generic drug approval procedures, and are they aware that the guidelines include a variable standard for bioavailability of the generic drug as compared to the innovator product? How have legislative and administrative issues influenced the daily prescribing habits and attitudes of the practicing physician? This study explored physicians' opinions about prescribing generic drug products.

METHODS

A questionnaire was mailed to a sample of 500 members of the Suffolk County Medical Society. Names were chosen with the use of a random numbers table from a membership list of approximately 1,600. After two mailings, a total of 245 responses were received. The questionnaire was designed to ascertain respondents' knowledge of several requirements related to prescription writing; their attitudes toward several specific drugs that have been the center of controversy concerning the bioequivalency issue; and the influence of patient opinions and other factors on the physician's prescribing behavior. Age, specialty, practice setting, terminal year of residency training, board certification status, and the average number of prescriptions written each day were ascertained. The data were analyzed to discern differences that relate to these characteristics.

RESULTS

Table I provides an analysis of respondents by specialty, practice type, and age. Specialties were distributed rather evenly between primary care and non-primary care subspecialties. Psychiatrists represented the smallest percentage of respondents (5.5%) and surgeons the largest (17.4%). Practice types were about evenly divided between solo practices and groups of two or more physicians.

TABLE I. Characteristics of Respondents

	N	%
Practice specialty		
General Internal Medicine	36	14.9
Family/General Practice	34	14.1
Obstetrics-Gynecology	28	11.6
Pediatrics	28	11.6
Psychiatry	13	5.5
Surgery	42	17.4
Subspecialty Internal Medicine	34	14.1
Other	26	10.8
No response	4	—
Total	245	100.0
Practice type		
Solo/private	112	46.5
Large private group (five or more)	26	10.8
Small private group (two to four)	88	36.5
Other	15	6.2
No response	4	—
Total	245	100.0
Age (yr)		
25–40	44	18.6
41–55	112	47.2
56 and over	81	34.2
No response	8	—
Total	245	100.0
Average number of prescriptions written daily		
5 or less	53	21.8
6–15	94	38.7
16–25	54	22.2
26 or more	42	17.3
No response	2	—
Total	245	100.0

Table II analyzes the respondents' beliefs regarding the efficacy of generic drug products. Respondents were evenly divided in their beliefs concerning the reliability of generic as compared to brand name drugs. A small majority (56.8%) agreed with the Food and Drug Administration's assertion that equivalent bioavailability indicates equal therapeutic efficacy. Only 12.9% of the respondents stated that they were familiar with the FDA procedure for determining a generic drug's bioequivalence.

TABLE II. Respondents' Beliefs and Knowledge About Generic Drugs

	N	%
Generic drugs as reliable as trade name drugs		
Agree	104	44.0
Disagree	105	44.5
Not sure	27	11.5
No response	9	—
Total	245	100.0
Equal bioavailability indicates equal efficacy		
Agree	129	56.8
Disagree	98	43.2
No response	18	—
Total	245	100.0
Respondent is knowledgeable about the FDA procedure for equivalence testing		
Yes	30	12.9
No	151	65.1
Not sure	51	22.0
No response	13	—
Total	245	100.0

TABLE III. Frequency of Prescribing Common Drugs Generically

	Penicillin		Diazepam		Digoxin		Theophylline	
	N	%	N	%	N	%	N	%
Never	30	13.7	59	29.1	83	52.5	64	37.9
Rarely	61	27.9	93	45.8	33	20.9	49	29.0
Often/very infrequently	128	58.4	51	25.1	42	26.6	56	33.1
No response	26	—	42	—	87	—	76	—
Total	245	100.0	245	100.0	245	100.0	245	100.0

The frequency of generic prescribing for several common medications (Table III) was relatively low. The majority of prescriptions for diazepam, digoxin, and theophylline were written for the trade name drug, while a majority of penicillin prescriptions were written for generic products. There were significant differences based on the number of prescriptions a physician wrote per day. Physicians who wrote few prescriptions (Table IV) reported the least knowledge of FDA procedures for equivalence testing ($\chi^2 = 13.41$, $p < 0.01$). Knowledge increased as the number of prescriptions written increased. Physicians' opinions did not differ according to their ages or practice type (solo vs group). Few respondents indicated that their willingness to prescribe generic drugs was influenced by state laws, FDA regulations, or the statements of pharmaceutical company representatives.

Differences in belief about the reliability of generic drug products by physicians' specialties are described in Table V. Pediatricians were most likely to believe that generic drug products are reliable while obstetricians-gynecologists and family/general practioners were least likely ($\chi^2 = 11.01$, $p < 0.05$).

The majority of physicians were influenced by a patient's request for a generic prescription (Table VI). More than 60% of respondents stated that they acceded to a patient's wish to receive generic drug products "often" or "very frequently." Patients do not, however, make such requests very frequently. Our sample reported that 72.8% of patients "rarely" or "never" ask for generic drug products (Table VII). Internists were most willing to prescribe generic drugs and were most frequently asked by patients for these drug products ($\chi^2 = 25.73$, $p < 0.01$). Though obstetricians-gynecologists are willing to prescribe generic products, they are least likely to report patient requests for generic drugs ($\chi^2 = 39.30$, $p < 0.01$).

DISCUSSION

The issue of deciding whether to prescribe a generic drug is complex. Some preparations are essentially equal in efficacy to the innovator drug, as in the case of oral penicillin. Some generic drugs are not bioequivalent, such as digoxin, ferrous sulfate, or thyroid hormone preparations. Other generic drugs, such as hydrochlorthiazide and phenytoin, are bioequivalent. Complexity is also the rule regarding cost savings. The use of generic drug products may result in significant cost savings. Pharmacy practices, however, may eliminate any savings to the patient.[12]

TABLE IV. Number of Prescriptions Written Daily Related to Respondents' Knowledge of FDA Procedures for Equivalence Testing

No. Prescriptions Written per Day	N	Knowledge of FDA Procedures Yes	No
5 or less	40	7.5%	92.5%
6–15	73	12.3%	87.7%
16–25	32	37.5%	62.5%
25	35	17.1%	82.9%
Total	180	16.7%	83.3%

$\chi^2 = 13.41$, $p < 0.01$.

In this survey, physicians reported considerable uncertainty about the procedures for testing and establishing efficacy of generic drug products. Only 12.9% of the sample reported knowledge of FDA procedures for testing these drugs, but the physicians were evenly divided in their beliefs about the effectiveness and reliability of substitute products.

The data from this study indicate that when physicians are asked by their patients to prescribe generic products, 60% often comply with their patients' requests. Although many physicians are willing to substitute generic drugs on the request of a patient, relatively few patients make the request. Physician willingness to prescribe generic products also varies with the type of drug in question—for example, generic prescriptions for penicillin are much more common than those for diazepam, digoxin, or theophylline.

Physician subspecialty and frequency of prescribing were related to the participants' attitudes and prescribing habits. In general, the more frequent prescriber and the primary care physician prescribed generic drugs most frequently. The less frequent prescriber and the subspecialist employed fewer generic drug products.

It is likely that a combination of both physician resistance and patient disinterest contributes to the low frequency of generic drug prescribing. A program of instruction may be necessary to improve both physician and patient awareness of the testing, efficacy, pricing, and other aspects of the generic drug dilemma. If generic drugs are to be prescribed optimally, both patients and

TABLE V. Differences in Opinion About Reliability of Generic Drugs By Specialty

Specialty	N	Generic Drugs as Reliable as Trade Name Drugs Yes	No
Family/General Practice	29	34.5%	65.5%
Obstetrics-Gynecology	24	33.3%	66.7%
Pediatrics	24	70.8%	29.2%
Internal Medicine	28	53.5%	46.4%
Surgery	39	59.0%	41.0%
Internal Medicine subspecialties	29	51.7%	48.3%
All specialties combined	173	50.9%	49.1%

$\chi^2 = 11.01$, $p < 0.05$.

TABLE VI. Frequency of Generic Prescribing Upon Patient Request By Specialty

Specialty	N	Never	Rarely	Often/ Very Frequently
Family/General Practice	32	12.5%	18.8%	68.7%
Obstetrics-Gynecology	25	4.0%	24.0%	72.0%
Pediatrics	27	11.1%	33.3%	55.6%
Internal Medicine	32	3.1%	12.5%	84.4%
Surgery	37	18.9%	48.6%	32.5%
Internal Medicine subspecialties	32	0.0%	34.4%	65.6%
All specialties combined	185	8.6%	29.2%	62.2%

$\chi^2 = 25.73$, $p < 0.01$.

TABLE VII. Frequency of Patient Requests for Generic Drug Products by Specialty

Specialty	N	Never	Rarely	Often/ Very Frequently
Family/General Practice	34	2.9%	61.8%	35.3%
Obstetrics-Gynecology	28	0.0%	96.4%	3.6%
Pediatrics	28	7.1%	71.5%	21.4%
Internal Medicine	36	2.8%	52.8%	44.4%
Surgery	42	16.7%	76.2%	7.1%
Internal Medicine subspecialties	34	0.0%	82.4%	17.6%
All specialties combined	202	5.4%	72.8%	21.8%

$\chi^2 = 39.30$, $p < 0.01$.

physicians will require instruction concerning the merits and disadvantages of generic prescribing. Opinions and practices need to be based on the full knowledge of the issues.

REFERENCES

1. Federal Food, Drug and Cosmetic Act of 1938, Public Act No. 717.
2. Harris-Kefauver Amendment of 1962, Public Law No. 87-781.
3. The Drug Price Competition and Patent Term Restoration Act of 1984, Public Law No. 98-417, 98 Stat 1585 (1984).
4. Collins PJ: Practical briefings. *Patient Care*, 1986; 20(1):21-22.
5. Sun M: Generics, Roche joust for valium market [news]. *Science* 1985; 228:472-473.
6. Chodos DJ: Generic substitution: A form of pharmaceutical Russian roulette? in Lasagna L (ed): *Controversies in Therapeutics*. Philadelphia, WB Saunders Co, 1980, pp 72-82.
7. Horvitz R: Generic substitution: A safe way to cut medical costs, in Lasagna L (ed): *Controversies in Therapeutics*. Philadelphia, WB Saunders Co, 1980, pp 63-71.
8. Mazullo JM: Generic substitution and Grandma's secret recipe, in Lasagna L (ed): *Controversies in Therapeutics*. Philadelphia, WB Saunders Co, 1980, pp 83-84.
9. Morgan JP, Schwartz LL, Sherman FT: A survey of generic drug legislation and geriatric pharmacotherapy: Opinions of those who generate the literature. *J Am Geriatric Soc* 1983; 31:535-539.
10. Francetic J, Lasagna L, Weintraub M, et al: Prescription prices under the New York generic substitution law. *Ann Intern Med* 1980; 92:419-423.
11. Brater DC, Pettinger WA: The generic prescribing issue. *Ann Intern Med* 1980; 92:426-427.
12. Bloom BS, Wierz DJ, Pauly MV: Cost and price of comparable branded and generic pharmaceuticals. *JAMA* 1986; 256:2523-2530.
13. Lasagna L: The economics of generic prescribing: Winners and losers [editorial]. *JAMA* 1986; 256:2566.
14. Schwartz LL: The debate over substitution policy. Its evolution and scientific basis. *Am J Med* 1985; 79:38-44.

The diagnostic significance of free erythrocyte protoporphyrin and the FEP/hemoglobin ratio in plumbism

SOLOMON RAJKUMAR, MD; VALERIE GEIBEL, PHD; BHASKAR DEVANAGONDI, MD;
BALAKRISHNA KAUL, PHD; AUDREY K. BROWN, MD

ABSTRACT. The diagnostic significance of elevations of free erythrocyte protoporphyrin (FEP) and the FEP/hemoglobin (Hb) ratio was evaluated in relation to anemia, iron deficiency, and iron deficiency anemia in 170 children with high blood lead levels (Pb > 30 mg/dL). Anemia was defined as an Hb level of less than 11 g/dL; transferrin saturations of less than 7% and 8-15% were considered as "definite" and "intermediate" iron deficiency, respectively. Anemia occurred in 6% (n = 10) of the children, iron deficiency in 29% (n = 49), and iron deficiency anemia in 9% (n = 16). The mean FEP as well as the FEP/Hb ratio were significantly higher ($p < 0.001$) in children with plumbism associated with anemia, iron deficiency, or iron deficiency anemia, than in those children with plumbism alone.

From these observations, we conclude that in children with high blood lead levels, there is a higher incidence of a biochemical iron deficiency state than iron deficiency anemia. Furthermore, iron deficiency associated with plumbism causes a significantly greater elevation of FEP and FEP/Hb ratio than plumbism or iron deficiency alone. Therefore, biochemical iron deficiency merits consideration in the assessment of FEP and the FEP/Hb ratio in pediatric lead poisoning.

(NY State J Med 1987; 87:542-545)

Elevation of free erythrocyte protoporphyrin (FEP) occurs not only in lead poisoning, but also in iron deficiency

---

From the Department of Pediatrics, State University of New York Health Science Center at Brooklyn, Brooklyn NY.

Address correspondence to Dr Rajkumar, Kings County Hospital Center (code 29), 451 Clarkson Ave, Brooklyn NY 11203.

anemia.[1-5] The incidence of iron deficiency anemia associated with plumbism is estimated by nationwide lead poisoning programs to be 4%.[6] Because iron deficiency anemia and lead poisoning occur in the same age groups, the interpretation of elevated FEP values in children with high blood lead levels is problematic. Elevated FEP values have not been shown to readily distinguish between the relative toxicity of lead and the degree of associated biochemical iron deficiency or iron deficiency anemia. This study was undertaken to evaluate the diagnostic significance of FEP and the FEP/Hb ratio in children with high blood lead levels.

PATIENTS AND METHODS

One hundred seventy children with lead levels greater than 30 mg/dL were studied. Forty-two children with lead levels of less than 29 mg/dL and with iron deficiency or iron deficiency anemia served as controls. Children with inherited hemoglobinopathies (eg, Hb SS, Hb SC, and Hb S-Thal diseases) were excluded from the study.

Venous blood samples were drawn from all subjects for the estimation of hemoglobin, hematocrit, lead level, serum iron, and total iron binding capacity (TIBC). The cyanmethemoglobin and Clay Adams microhematocrit methods[7,8] were used to estimate the hemoglobin level and hematocrit, respectively. Serum iron and TIBC were measured according to the method of Fischer and Price.[9] Lead levels were determined by atomic absorption spectrophotometric analysis.[10] Erythrocyte protoporphyrin was measured using the extraction technique.[11] Transferrin saturation was calculated as the ratio of serum iron to TIBC × 100. The FEP/Hb ratio was calculated as a ratio of FEP mg/dL to Hb g/dL.

Anemia was defined as a hemoglobin level of less than 11 g/dL.[12] Transferrin saturation of greater than 7% was used as an indicator of "definite" iron deficiency.[13] Values between 8% and 15% were considered as indicating "intermediate" iron deficiency.[13]

All data were punched onto cards, and statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS).[14]

RESULTS

The mean age of the children in the study population was five years and nine months (range, 1 yr, 2 mo–13 yr, 9 mo) and two years and five months (range, 6 mo–8 yr, 9 mo) for the controls. While it is generally believed that lead poisoning is not much of a problem after six years of age, 38% of the study population was over six years, one third being in class III of the Risk Classification defined by the Centers for Disease Control (CDC) of the Public Health Service.[15]

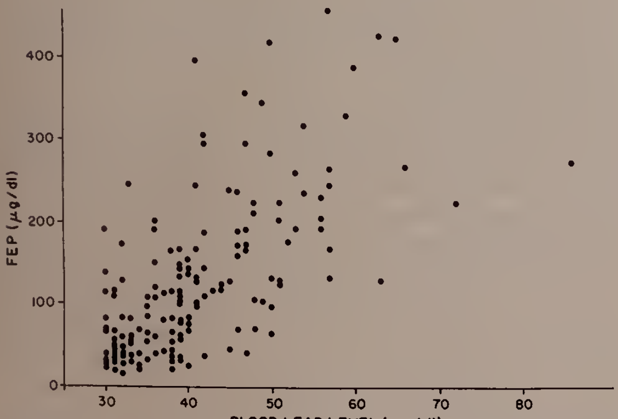


FIGURE 1. The relationship of lead levels to free erythrocyte protoporphyrin (FEP) in plumbism.

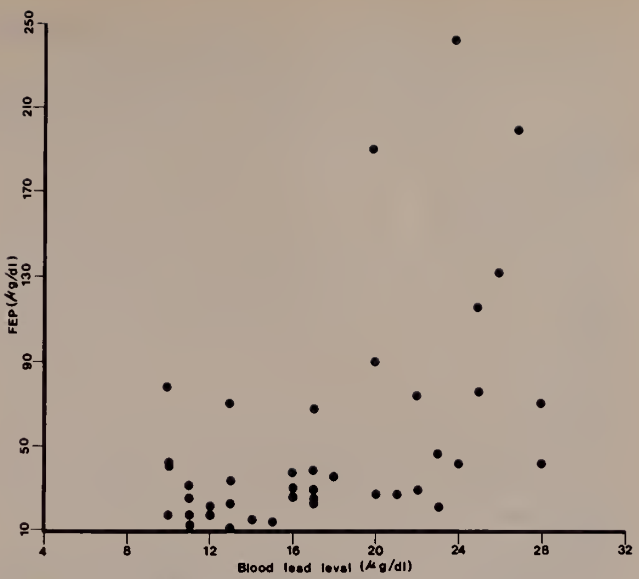


FIGURE 2. The relationship of lead levels to free erythrocyte protoporphyrin (FEP) in iron deficiency.

Free Erythrocyte Protoporphyrin. The relationship of lead level to FEP for both study and control groups is shown in Figures 1 and 2. In both groups, higher values of FEP were associated with elevated lead levels; the relationship was of a curvilinear nature. The positive relationship was more pronounced in the study group than among controls (r = 0.7; controls, r = 0.5). A considerable variation in FEP values was observed for a given range of blood lead levels (Table I). FEP values greater than 50 mg/dL were found in 129/170 (76%) of the children in the study group and in only 12/42 (28%) of control children.

Incidence of Iron Deficiency. Forty-four percent (75/170) of children with high blood lead levels had anemia, iron deficiency, or iron deficiency anemia. Anemia occurred in 6% (n = 10), iron deficiency in 29% (n = 49), and iron deficiency anemia in 9% (n = 16).

FEP and FEP/Hb ratio. FEP and the FEP/Hb ratio for different groups of children are presented in Table II. The mean FEP for children with only lead burden was 100 mg/dL; for those with lead burden and anemia (but no iron deficiency or iron deficiency anemia), the FEP was 156 mg/dL. For children with increased lead burden and iron deficiency (but no anemia) the FEP was even higher: for intermediate iron deficiency it was 161 mg/dL (control, 47 ± 12 mg/dL); and for definite iron deficiency it was 218 mg/dL (control, 40 ± 7 mg/dL). The greatest increase in FEP was noted in children with both increased lead burden and obvious iron deficiency anemia: intermediate iron deficiency = 170 mg/dL (control, 44 ± 13 mg/dL); definite iron deficiency = 237 mg/dL (control, 92 ± 28 mg/dL). The relationship of FEP to biochemical iron deficiency, iron deficiency anemia, lead burden alone, lead burden plus iron deficiency, lead burden plus anemia, and lead burden plus iron deficiency anemia is illustrated by plotting 95% confidence limits (Fig 3).

TABLE I. Distribution of Number of Children According to Risk Classification of Free Erythrocyte Protoporphyrin Versus Blood Lead Level

Blood Lead (mg/dL)	Free Erythrocyte Protoporphyrin (mg/dL)				
	≥ 49	50–109	110–249	≥ 250	Total
30–49	41	47	42	6	136
50–69	0	2	15	15	32
≥70	0	0	1	1	2
Total	41	49	58	22	170

TABLE II. **Free Erythrocyte Protoporphyrin and Free Erythrocyte Protoporphyrin/Hemoglobin Ratio***

	Pb** = <29 mg/dL							Pb = >30 mg/dL						
	No.	Pb	Hb**	Iron	TIBC**	FEP	FEP/Hb Ratio	No.	Pb	Hb	Iron	TIBC	FEP**	FEP/Hb Ratio†
Plumbism														
No anemia														
No iron deficiency														
No iron deficiency anemia														
Hb > 11g														
TS** > 15%								95	40 ± 0.9	12.9 ± 0.1	94 ± 3	358 ± 5	100 ± 8	7.9 ± 0.7
Anemia														
Hb < 11g														
TS > 15%								10	42 ± 2.7	9.8 ± 0.4	89 ± 4	372 ± 21	156 ± 29	16.8 ± 3.8
Iron deficiency														
Hb > 11g														
TS, 8–15% (intermediate)	10	17 ± 1.9	12.1 ± 10.1	42 ± 2	365 ± 12	47 ± 12	3.9 ± 1.0	34	42 ± 1.9	12.4 ± 0.2	50 ± 2	389 ± 13	161 ± 21	13.2 ± 1.7
TS, < 7% (definite)	6	20 ± 1.7	12.4 ± 0.5	23 ± 3	425 ± 27	40 ± 7	3.3 ± 0.6	15	45 ± 3.7	12.7 ± 0.3	24 ± 2	435 ± 16	218 ± 39	17.6 ± 3.2
Iron deficiency anemia														
Hb < 11g														
TS, 8–15% (intermediate)	17	15 ± 1.2	10.2 ± 0.1	38 ± 2	354 ± 14	44 ± 13	4.3 ± 1.2	8	46 ± 3.4	10.5 ± .01	46 ± 4	381 ± 17	170 ± 46	13.9 ± 5.0
TS, < 7% (definite)	9	21 ± 2.1	9.7 ± 0.3	24 ± 2	449 ± 29	92 ± 20	9.9 ± 2.4	8	45 ± 4.7	10.2 ± 0.2	26 ± 2	450 ± 31	237 ± 65	23.6 ± 7.0

* Mean ± standard error of the mean.

† Analysis of variance $p < 0.0001$.

** Pb = lead, Hb = hemoglobin, TIBC = total iron binding capacity, FEP = free erythrocyte protoporphyrin, TS = transferrin saturation.

Discussion

The erythrocyte protoporphyrin level, expressed as free erythrocyte protoporphyrin (FEP), is used as the primary diagnostic screening test for lead poisoning.[15,16] However, an elevated FEP level in lead poisoning may also be due to associated anemia, iron deficiency, or iron deficiency anemia.

The mechanism of anemia associated with lead poisoning, although not completely elucidated, can be attributed to either inhibition of heme synthesis or shortened life span of red blood cells. Anemia unrelated to iron deficiency or inherited hemoglobinopathies occurred in 6% of this study population. Within this group, the possibility of patients with thalassemia traits cannot be ruled out. However, Koenig et al[4,5] demonstrated that FEP levels can be used to differentiate iron deficiency anemia from alpha or beta thalassemia traits. They found the FEP levels in iron deficiency anemia to be greater than 185 mg/100 mL red blood cells, as compared to less than 107 mg/100 mL red blood cells in thalassemia traits (normal, 25–100 mg/100 mL red blood cells). In this regard, the authors suggested that FEP may even be a better parameter than the Mentzer index.[5] In a study by Cohen et al,[17] the incidence of anemia was 12%. However, the combination of anemia and microcytosis occurred only in one out of 58 patients (2%), suggesting that microcytic anemia in lead poisoning is rather uncommon. Hence, the probability of patients in this group having thalassemia traits is low.

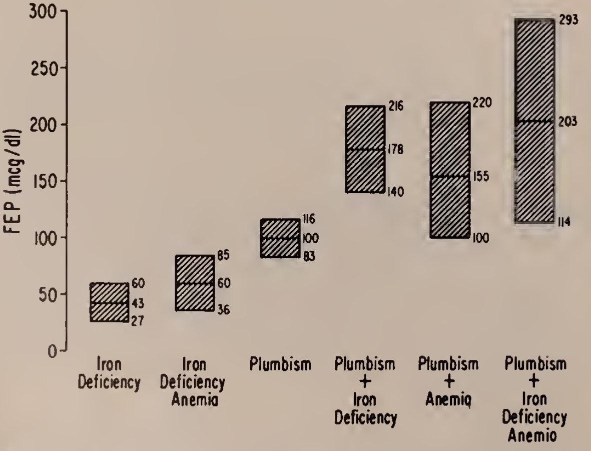


FIGURE 3. Ninety-five percent confidence limits for free erythrocyte protoporphyrin (FEP).

The most specific indicator of lead toxicity is urinary aminolevulinic acid (ALA). However, measurements of urinary ALA in random voidings are unreliable, necessitating 24-hour urine collection. On the other hand, erythrocytic protoporphyrin, another indicator of toxicity, can be estimated easily from blood obtained by venipuncture or fingerstick. Chisolm[18] has demonstrated a statistically significant linear relationship between urinary ALA and FEP. In later studies, although Chisolm was unable to confirm these findings, he attributed the difficulties to modifications in methodology[19] (personal communication, August 1984).

Iron deficiency or iron deficiency anemia, by themselves capable of elevating FEP levels, can cause such levels to become more pronounced in the setting of plumbism; this effect is referred to as background response. In fact, this response was demonstrated by Chisolm,[18] utilizing the hematocrit as an indicator of anemia. The study reported on here, utilizing hemoglobin and transferrin saturation as indicators of anemia and iron deficiency, respectively, clearly demonstrates the magnitude of this background response. This could explain, at least in part, the variation of FEP values observed for a given level of blood lead (Table I). Hannah et al[20] observed a similar phenomenon.

The FEP/Hb ratio, yet another indicator of heme bio-synthesis, has been demonstrated to increase not only with iron deficiency anemia, but also with decreasing transferrin saturation.[21] Data from the control subjects support that conclusion. In addition, these data enable comparisons of the FEP/Hb ratio with anemia, biochemical iron deficiency, and iron deficiency anemia. The pattern of increase in the FEP/Hb ratio closely paralleled that of FEP.

This study has shown that more children with high blood lead levels have biochemical iron deficiency (29%) compared to anemia (15%). Biochemical iron deficiency, even without anemia, raises the FEP and FEP/Hb ratio in children with lead poisoning. This elevation in FEP may erroneously be interpreted as indicative of lead toxicity. Therefore, biochemical iron deficiency in plumbism merits consideration in the proper interpretation of elevated FEP and FEP/Hb ratio in the absence of anemia. Transferrin saturation and/or ferritin level should be determined to help distinguish between the degree of associated biochemical iron deficiency and relative lead toxicity.

Acknowledgment. The authors thank Dr Julian Chisolm Jr for his comments and review of the manuscript.

REFERENCES

1. Watson JR, Decker E, Lichtman HC: Hematologic studies of children with lead poisoning. *Pediatrics* 1958; 21:40–46.
2. Watson CJ: Erythrocyte coproporphyrin; variation in respect to erythrocyte protoporphyrin and reticulocytes in certain anemias. *Arch Int Med* 1950; 86:797–809.
3. Braitman R: Marked elevation of FEP with iron deficiency anemia [letter]. *J Pediatr* 1978; 93:155–156.
4. Koenig HM: Classification of microcytic anemia by flurometric analysis of free erythrocyte porphyrins (FEP). *Ann Clin Res* 1976; 8(suppl 17):151–155.
5. Koenig HM, Lightley A, Schanberger J: The micromeasurement of free erythrocyte protoporphyrin as a means of differentiating alpha thalassemia trait from iron deficiency anemia. *J Pediatr* 1975; 86:539–541.
6. Centers for Disease Control: Surveillance of childhood lead poisoning. *MMWR* 1980; 29:170–171.
7. Cannan RK: Proposal for a certified standard for use in hemoglobinometry. *Amer J Clin Path* 1958; 30:211–215.
8. Mcgovern JJ, Jones AR, Steinberg AG: Hematocrit of capillary blood. *N Engl J Med* 1955; 253:308–312.
9. Fischer DS, Price DC: A simple serum iron method using new sensitive chromogen tripyridyl-s-thiazine. *Clin Chem* 1974; 10:21.
10. Hessel DW: A simple and rapid quantitative determination of lead in blood. *Atoms Abs Newsletter* 1968; 7:55.
11. Piomelli S, Davidow B, Guinee VF, et al: The FEP (free erythrocyte porphyrins) test: A screening micromethod for lead poisoning. *Pediatrics* 1973; 51:254–259.
12. American Academy of Pediatrics Committee on Nutrition: Iron balance and requirement in infancy. *Pediatrics* 1969; 43:134–142.
13. Pearson HA: Iron nutrition revisited—infancy, childhood, adolescence, in Oski FA, Pearson HA (eds): *Report of Eighty-Second Ross Conference on Pediatric Research.* Columbus, Ohio, Ross Laboratories, 1981, pp 8–9.
14. Klecka WR, Nie NH, Hull CH: *Statistical Package for the Social Sciences (SPSS) Primer.* New York, McGraw-Hill Book Co, 1975.
15. Centers for Disease Control: Preventing lead poisoning in young children. *J Pediatr* 1978; 93:709–720.
16. Centers for Disease Control: Increased lead absorption and lead poisoning in young children. *J Pediatr* 1975; 87:824–830.
17. Cohen AR, Trotzky MS, Pincus D: Reassessment of the microcytic anemia of lead poisoning. *Pediatrics* 1981; 67:904–906.
18. Chisolm JJ Jr, Barrett MB, Mellits ED: Dose-effect and dose-response relationships for lead in children. *J Pediatr* 1975; 87:1152–1160.
19. Chisolm JJ Jr, Hastings CW, Cheung DK: Microphoto-fluorometric assay for protoporphyrin in acidified acetone extracts of whole blood. *Biochem Med* 1974; 9:113–135.
20. Hannah TL, Dietzler DN, Smith CH, et al: Erythrocyte porphyrin analysis in the detection of lead poisoning in children: Evaluation of four micromethods. *Clin Chem* 1976; 22:161–168.
21. Piomelli S, Brickman A, Carlos E: Rapid diagnosis of iron deficiency by measurement of free erythrocyte protoporphyrin and hemoglobin: The FEP/ hemoglobin ratio. *Pediatrics* 1976; 57:136–141.

FROM THE LIBRARY

LEAD POISONING ASSOCIATED WITH ASCARIASIS

This incident was reported to the Poison Control Center as a case of lead poisoning. For about a year the mother had observed the two-year-old child eating bits of plaster but was unaware of the harmful effects which it may cause. The child was under supervision in a child health station, and because of a failure to gain weight and a history of pica, the patient was referred to the hospital for further investigation and possible treatment.

On admission to the hospital the only complaint was abdominal pains, and the presenting symptoms were anemia and underweight. The laboratory findings showed the lead blood level to be 0.07 mg. per 100 ml. and the urine lead level to be 0.06 mg. per liter. Spinal fluid and protein determinations were not done. The hemoglobin was 9.4 Gm. per 100 ml., and the red blood cell count was 3,300,000. After two weeks of hospitalization and therapy, the child was discharged.

This case was complicated by the finding in the stools of a parasitic infestation (ascariasis). In addition to ascariasis, a diagnosis of lead intoxication was made. Following her discharge from the hospital, the child was treated in the outpatient department with piperazine hydrochloride for the parasitic infestation and with iron therapy for the anemia.

Lead poisoning and parasitic infestation are not infrequent in Puerto Rican children, and their coexistence is not uncommon. A confirmed diagnosis of lead poisoning, particularly in a Puerto Rican child, should not exclude a stool examination for the determination of another condition which also may contribute to the anemia. . . .

(*NY State J Med* 1962; 62:2551–2553)

The role of physicians in the life insurance industry in the 19th century

JOHN S. HALLER, JR

Although life insurance in its most primitive forms can be traced to the ancient mutual and fraternal orders of the Greeks and Romans, its establishment on a more rational and scientific basis occurred in England during the reign of Queen Anne, where so-called "friendly" or "benevolent" societies paid out small sums for burial expenses and provided annuities to widows of clergymen. Many of these societies failed due to their lack of sound business principles, and the fact that they assessed premiums without a careful analysis of the risk. As long as societies accepted applicants on the basis of charity or fraternity and without a medical examination, their very survival was in jeopardy. Attributes such as good fellowship, mutual aid, and sociability had their place, but not in business.[1]

Later in the 18th century, applicants for life insurance were required to sign affidavits of good health before a policy could be issued. Later, applicants were required to appear before a board of directors who, as laymen, determined by inspection the value and classification of the risk, and set the amount to insure. Eventually associations recognized that their prosperity depended upon a more scientific understanding of life and health and called upon physician examiners to serve as their gatekeepers. However, the earliest medical examinations were superficial at best.[2] In 1770, the health certificate for life insurance simply required the signature of two individuals, one of whom was a physician. The directors of the company then conducted a secret vote on whether the applicant should be accepted or rejected. Although the first physician examiner was appointed in 1779, they did not become a regular part of the insurance industry until 1820. One of England's oldest insurance companies, founded in 1706, did not retain a physician examiner until 1855.[3,4]

THE MEDICAL EXAMINER

Obviously a business as vast as life insurance required substantial administration. "Like the temple of Gaza," noted C.C. Bombaugh, MD, at the 11th annual meeting of the American Academy of Medicine at Washington in 1887, "the structure of life insurance has two chief supporting pillars. The one is the mathematical, the other is the medical. The one we leave to the actuary; be it our care to see that no mischievous or antagonistic forces are applied to the other."[5] In the financial risk-taking of these institutions, the role of the physician examiner became critical to each company's program. Like a bad loan for a bank, bad risks for an insurance company weakened it; too many broke it. As one physician noted,

> A company's natural constitution is represented by its assets, its power of endurance by its surplus, its liability to premature death by the character of its risks; hence a company without assets has no natural constitution, one without surplus no powers of endurance, one loaded down with bad risks is ready to die at any time.[6]

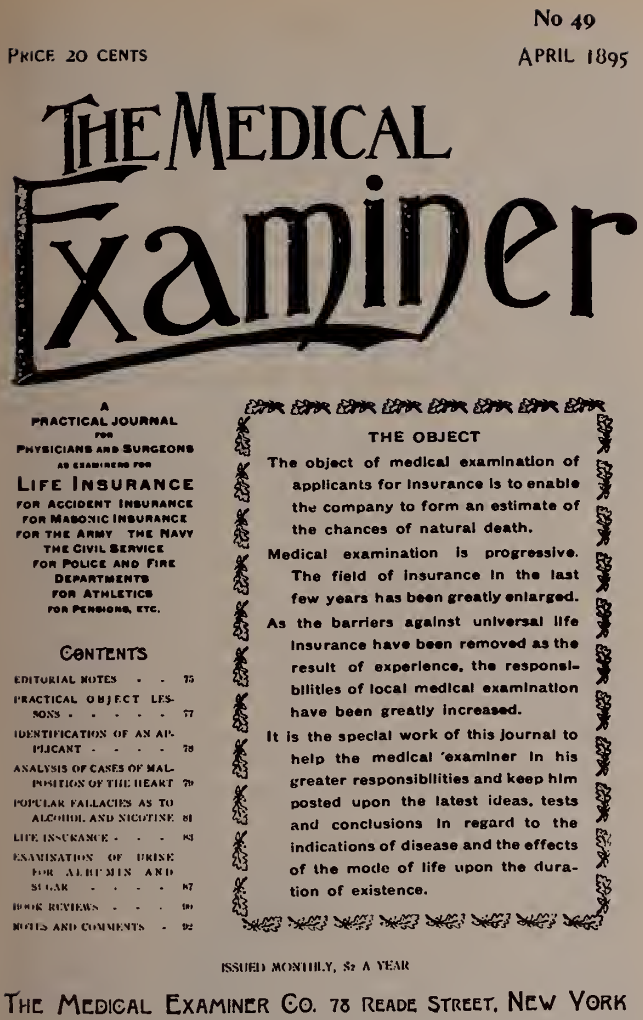
The physician examiner stood to the insurance company as the legal examiner did to a court; decisions by both the company and the court required the skillful assessment of facts. "No medical referee having a due regard for his own or the reputation of his profession," wrote J.B. Temple, president of the Southern Mutual Life Insurance Company of Kentucky, "will fail to remember that confidence of interested parties—not the company only, but the future orphans and widows whose trustee it is—has been reposed in him. He is not the employee of an agent or an applicant who may chance to call on him, but of a society wielding important and delicate trusts."[7]

From the point of view of the medical profession, the physician examiner was the most important officer on the roster of a life insurance company. Without his ability and integrity a company opened itself to financial disaster. For this reason, companies expected certain qualities from the physician examiner, including being a "believer in life insurance, not only theoretically but practically."[8] Unless an examiner accepted the utility and benefits of insurance, he failed in his responsibilities to both the insured and the company.[8,9]

Characteristics of the ideal physician examiner, as explained in numerous 19th century publications, included mental adaptability, good judgment, accurate powers of discrimination, and a keen eye trained to discover the laws of character and heredity. Although the final decision whether to accept or reject an applicant would lie with the company itself, the data obtained from the medical examination gave the only clear basis for the decision. For this reason, remarked William Detmold, MD, in the *Medical Record* of 1877, "strict honesty. . .is much more im-

---

Address correspondence to Mr Haller, Professor of History, University of Colorado at Denver, Denver, CO 80202.



No 49

Price 20 cents

April 1895

THE MEDICAL Examiner

A
PRACTICAL JOURNAL
FOR
PHYSICIANS AND SURGEONS
AS EXAMINERS FOR
LIFE INSURANCE
FOR ACCIDENT INSURANCE
FOR MASONIC INSURANCE
FOR THE ARMY THE NAVY
THE CIVIL SERVICE
FOR POLICE AND FIRE
DEPARTMENTS
FOR ATHLETICS
FOR PENSIONS, ETC.

CONTENTS

EDITORIAL NOTES - - 75
PRACTICAL OBJECT LESSONS - - - - - - 77
IDENTIFICATION OF AN APPLICANT - - - - 78
ANALYSIS OF CASES OF MALPOSITION OF THE HEART 79
POPULAR FALLACIES AS TO ALCOHOL AND NICOTINE 81
LIFE INSURANCE - - - 83
EXAMINATION OF URINE FOR ALBUMIN AND SUGAR - - - - 87
BOOK REVIEWS - - - 90
NOTES AND COMMENTS - 92

THE OBJECT

The object of medical examination of applicants for insurance is to enable the company to form an estimate of the chances of natural death.

Medical examination is progressive. The field of insurance in the last few years has been greatly enlarged.

As the barriers against universal life insurance have been removed as the result of experience, the responsibilities of local medical examination have been greatly increased.

It is the special work of this journal to help the medical 'examiner in his greater responsibilities and keep him posted upon the latest ideas, tests and conclusions in regard to the indications of disease and the effects of the mode of life upon the duration of existence.

ISSUED MONTHLY, $2 A YEAR

THE MEDICAL EXAMINER CO. 78 READE STREET, NEW YORK

FIGURE 1. Cover of an early journal addressing the issue of medical examination of applicants for insurance.

portant than scientific assumptions."[10,11]

Other characteristics as well were expected of the physician examiner. While he may have had a preference for certain companies or insurance plans, he could not discuss these with the applicant; nor could he work for a company he could not endorse as sound and legitimate. "A physician should no more lend the weight of his professional . . . standing as a citizen to the assistance of a quack life insurance company," wrote a physician in the *Indiana Medical Journal* in 1896, "than he would give certificates to quack remedies."[12] If he examined for more than one company he should avoid betraying confidences involving applicants or actions of the company. He could not reveal pending transactions and, acting in his capacity as examiner, should disregard any relationship to the individual as personal physician. Thus personal knowledge of a disease which the applicant preferred to remain secret could not be kept confidential. Company loyalty, avoiding personal relationships with applicants, maintaining precision in information, attending promptly to all appointments, showing discretion and industriousness, and never asking for business were other qualities noted as essential for every examiner.[13,14]

Physician examiners preferred to be responsible solely to the Medical Department of the company and believed that their reports should be private, with neither the agent nor the applicant having access to them. The final determination of acceptance or rejection would lie with the Medical Department, acting on the report of the examiner. However, this was not always the case. Often, physician examiners and agents divided their commissions, which, practically speaking, amounted to offering a bribe to the examiner for passing a risk. Cases where local agents and physician examiners pooled their commissions and examination fees resulted in devastating statistics that usually sent "cold chills over the home office."[8,15]

Agents, who usually appointed their own physician examiners, sometimes abused their power of appointment. By allowing themselves to be motivated by the number of policies written rather than the potential "risk" of a policy holder, they encouraged physician examiners who were less cautious for fear of losing a client. Clearly the unscrupulous agent could write policies only with the support of an unscrupulous doctor—or an incompetent one. Such results, noted the editor of the *Western Journal of Medicine*,[16] in 1868, were the inevitable product of a system which erected a "beggarly standard of professional fees." The outcome was a "system of cheap physicians." When this occurred, the editor went on to say, "the aggregate worthlessness of appointments, begotten of the niggardly principle on which services are judged of, and their price adjusted, must fearfully increase the natural liability of losses."[16] This wasteful situation, when compounded by similar wrong judgments—by both the agent in seeking the client and the physician in approving the policy—brought into question the savings to the company and the price of insurance.[16]

Articles in medical journals cautioned physicians to be on their guard with agents or solicitors whose personal interest was to have the physician examiner "pass" every potential client for life insurance, because some agents knowingly wrote life policies on unsound applicants, thinking only of the fee or commission attached to the policy. To protect themselves from pressure, doctors were urged never to allow the agent to be present at an examination.[17]

In 1874, a number of American insurance companies organized themselves into an association known as the Chamber of Life Insurance. Included in its mission of promoting life insurance interests was the need to maintain a list of accepted physician examiners. This suggested, by implication, that the examiner functioned as an expert witness for the company in the selection of lives and that he could be trusted to treat all individuals impartially. Since considerable patronage followed these appointments, the association expected a higher level of responsibility from its member physicians. Advocates of this change believed that while no physician was expected to make a specialty of examinations, he should be thoroughly qualified and knowledgeable on such matters as longevity, heredity, occupation, and habits—areas directly bearing upon insurance matters.[18]

By the late 1880s, the home offices had begun to select local examiners. Physician examiners thus became independent of agents, generally better paid for their services,

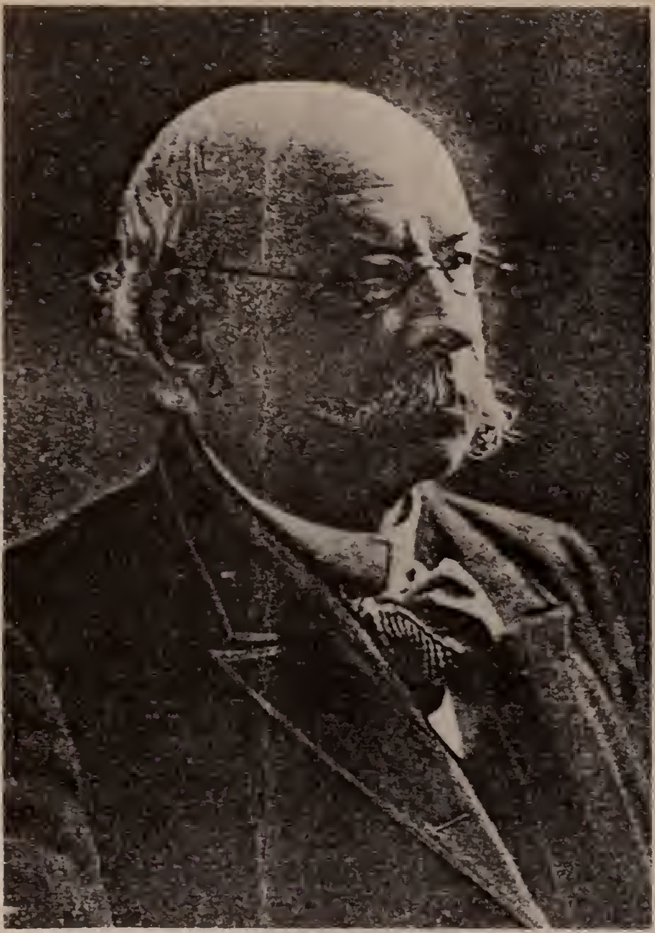


FIGURE 2. Francis Peyre Porcher, MD, President of the Medical Society of South Carolina in 1895, and an examiner and referee for the Mutual Life and the Equitable of New York insurance companies.

and more aware of their responsibilities. The physician had the legal duty to ascertain and report to the company on the health or physical condition of the applicant and whether he suffered any life-shortening disease or defect. Only the physician who acted "without fear or favor" was qualified to fill this role.[6]

Since most insurance companies did not recognize homeopaths as examiners, homeopathic physicians preferred to work for fraternal societies rather than accept what they considered the discriminatory attitude of insurance companies. In 1890, the Aetna Life Insurance Company of Hartford had 56 physician examiners in 20 cities, none of whom were homeopaths. The same held true of New York Life. Northwestern Mutual Life Insurance of Milwaukee had two homeopaths out of a total of 43 examiners, Berkshire Life Insurance Company had nine homeopaths out of 558 examiners, Equitable of New York had one out of 132 examiners, and Metropolitan Life Insurance had one out of 49 examiners in eight cities. Similar statistics existed for Travelers of Hartford and John Hancock Mutual Life Insurance Company of Boston, which had no homeopaths affiliated as examiners.[19]

MEDICAL EXAMINATION

American life insurance companies tended to use application forms that were generally similar throughout the industry. The application form requested the amount, character, and condition of policy desired by the client; the family or hereditary history of the applicant; the individual history of the applicant including past occupations, residence, diseases, and habits; and finally, an assessment of the present condition of the applicant. The key elements of any application included information on the age, habits, and personal and family history. Of these, age was the only provable element; the rest depended upon the honesty of the applicant and the ability of the physician examiner to discern health and family history.[17,20]

By the 1860s and 1870s, insurance companies required applicants to answer many detailed questions on their health and family history, and insisted upon the absolute truthfulness of each statement. The law as interpreted by the courts rendered a policy void if the applicant answered the questions dishonestly. This gave companies discretionary powers that allowed them to take advantage of mistakes made by the applicant, even those that were unintended and immaterial to the risk. Eventually, legislation forbade companies from voiding contracts on the basis of these mistakes unless they were material to the risk, willfully made, and "life terminated because of something of which a truthful answer would have given the company a warning note."[12] By the turn of the century, companies voluntarily adopted policies which made applications indisputable after three years regardless of misstatement. Because of the competitive nature of insurance companies in the United States, many eventually chose to reduce the limitation to one year. In effect, no misstatement in the application voided a policy after it had been in force for one year.[12]

Physician examiners faced a more difficult task in examining for life insurance than dealing with a typical office visit. The reasons were obvious. Patients calling upon physicians to find a cure for a particular ailment were anxious to acknowledge every pain and symptom of disease; on the other hand, those wanting life insurance seldom communicated the cause or history of any malady. Instead, they preferred to withhold all information and respond as indirectly as possible to questions that delved into the risk side of insurance. Many applicants feared that an examination would disclose some poor habit, bad health situation, disease, or hereditary trait that might void the application.[21]

Articles and handbooks written for the physician examiner urged caution in guarding against applicants who attempted to conceal defects of personal health or family history. Seldom did the physician know the patient before the examination; most often, the physician had to become an instant expert on the personal and family history of the applicant.[22–26] Through obtaining the family history, the physician examiner could learn facts concerning the family longevity, including any hereditary tendency to diseases such as consumption, insanity, and epilepsy. If the family history suggested anything that might make the applicant objectionable, the nature of the individual history became all the more important.

Of principal concern among the supposed hereditary diseases was consumption. Doctors were cautioned to watch for histories that included deaths from exposure to cold, shipwreck, journeys across the plains, or child-bed. Too often, these were thinly disguised examples of deaths from hereditary consumption. Although medical science gave "no rules or ratio of the frequency of this inheritance," doctors generally agreed that the disease occurred through a "hereditary taint" as well as by independent acquisition.[17] Increasingly, however, Felix von Niemeyer's (1820–1871) *Clinical Lectures on Pulmonary Consumption* (1870) inclined the medical world toward the idea of independent acquisition, with consumption originating in pneumonia, bronchitis, pulmonary congestion, and pulmonary hemorrhage rather than in a tubercular diathesis. Nevertheless, many physicians recommended rejecting an applicant if both parents had died of consumption or when even one parent had died of the disease "unless it can be clearly shown that the death was from an acquired consumption, dating years after the birth of the applicant, and that the latter shall have passed the age of forty-five in perfect health."[27] The same held true if two or more brothers or sisters had died of consumption.

Insanity was another supposed hereditary disease which physician examiners considered important in applicants; others included cancer and gout, and "hereditary intemperance." Physicians cautioned against approving applicants whose family was exposed to drunkenness and even teetotaling ("for in a large proportion of cases the teetotaler is a reformed drunkard"). The same held true for tobacco addiction which included symptoms of heart palpitation, irregularity of pulse, dyspepsia, emaciation, and general debility.[10]

FIGURE 3. Thomas Chappell Cook, AM, MD, medical examiner for the Manhattan Life Insurance Company and the Mutual Life Insurance Company, both of New York.

Insurance companies also relied to a certain degree on evidence of classical constitutional pathology.

> If the swarthy are bilious, the lymphatic indolent and intemperate; if short necks, florid faces and heavy frames are correlated to gouty change with fragile blood vessels disposing to apoplexy; if blue eyes, golden hair and velvety skins are indexes to a strenuous diathesis favoring consumption, glandular swellings, spinal caries and lumbar abscesses; if slender bones, taut muscles, compact abdomens and large fore brains mark the nervous temperament—logical subjects for neurotic maladies and drug habits; if these physical distinctions are imparted to offspring, who subsequently develop the pathological states foreshadowed by definite physical qualities, then is hereditary transmission of disease not to be gainsaid; then are tendency and heredity convertible terms.[28]

In the individual history of each patient, physician examiners sought to verify those significant diseases which had afflicted the patient, as well as his physical development and ability to resist disease. The applicant's actual condition proved equally important. This included proportion of height to weight, excessive obesity or emaciation, figure or form of the applicant (round shouldered, flat chested, etc), countenance or general expression, and appearance. Other factors included the physical examination of the applicant, in which the physician distinguished between normal and abnormal sounds elicited by auscultation and percussion, and inspected the thoracic and abdominal viscera, noting the existence or absence of hernias, varicose veins, tumors, ulcers, cicatrices, and glandular enlargements.

Obviously, occupation influenced both the health and longevity of the client. Applicants whose work environment contained dust were susceptible to lung problems. Thus, bakers, upholsterers, ax-grinders, and stone and marble cutters were always considered poor risks. The same was true of those who were exposed to intense heat or sudden cold. Members of the learned professions, on the other hand, had the greatest longevity (Table I).[10]

No longer was it sufficient for examiners simply to thump the chest, poke the belly, or guess at the pulse rate. Hasty examinations that made no note of heredity, occupation, habits, tendencies, and family history, and that made no effort to test the urine or blood, were useless. By the mid-1880s, even beneficiary associations were beginning to require examinations as well as tests of the urine, lungs, and heart as part of the application for membership.[29,30]

Although American physician examiners differed among themselves in the scope of their inquiries and the forms used in their examinations, the differences among countries were even greater. At the Second International Congress of Insurance Physicians in Amsterdam in 1902, a universal medical form was introduced. The commission had spent two years preparing the form but achieved no consensus among the member physicians. American physicians objected to the fact that the form lacked questions on the cause of death or state of health of grandparents. Moreover, the form contained 63 questions, exclusive of family history, and an appendix with 100 additional questions. American physicians preferred a form with a maxi-

TABLE I. Professions Ranked from Most Dangerous to Most Favorable*

Class I	Class II	Class III	Class IV
Brakeman on freight trains	Bridge builder, boatman	Barkeeper	Apothecary, architect
Buzz sawyer, circular sawyer	Barber on steamboat	Blacksmith (working)	Artist, painter, author, writer
Powder maker, seaman	Brakeman on mail train	Bricklayer	Attorney, lawyer
	Clerk on river steamboat	Captain on river steamer	Bank officer or clerk
	Car coupler	Car driver	Baker, barber
	Conductor on freight trains	Carman (drayman)	Boot and shoe maker
	Cooper	Carpenter and joiner	Builder (not laborer)
	Engineer on river steamer	Chief engineer	Civil engineer
	Fireman (locomotive)	Conductor on passenger trains	Clergyman (minister)
	Horse shoer	Distiller	Clerk (generally)
	Laborer (wharf, warehouse, elevator)	Foundry & furnace employee	Chemist, manufacturing
	Miner (underground)	Hod carrier	Dentist, dressmaker
	Sailor	Laborer, common	Editor, farmer
		Mason	Marble cutter
		Miner (surface)	Photographer
		Puddler	Physician, postmaster
		Stone cutter and dresser	Painter (house, ornamental)
		Teamster	Plumber, printer
		Wood chopper	Tanner, tailor
			Teacher, tool maker
			Vitriol manufacturer
			Wheelwright
			Watchman

* Abstracted from work cited in reference 17.

mum of 40 to 50 questions and which placed more emphasis on the actual physical examination. They also objected to the lack of questions on body temperature, condition of knee jerk reflexes, pupillary reflexes, and a determination of the applicant's environment.[31]

Dr Price, who was known as the "Father of correct life insurance," warned insurance companies of the dangers of inaccurate or uninformed medical examinations. "Those persons will be most for flying to these establishments," he remarked, "who have feeble constitutions or are subject to distempers which they know render their lives particularly precarious." The disposition to pass "bad lives" onto the lists of an insurance company offered short term profit to the agent but a substantial and long term loss for the company. Admittedly, agents lacked the skill to detect latent disease which the educated physician was presumed to possess; only when both agent and physician exercised good moral judgment was it reasonable to conclude that the decision to insure was both reasonable and fair.[7]

Information sharing among companies was common, especially on undesirable risks. Companies found it mutually convenient to share among themselves "interchange rejections." Thus, when a company declined a particular risk, it sent a notice of the decline to other companies in the interchange. Eventually, companies found it easier to send these notices to a distribution center. This plan continued in use into the 1890s.[32]

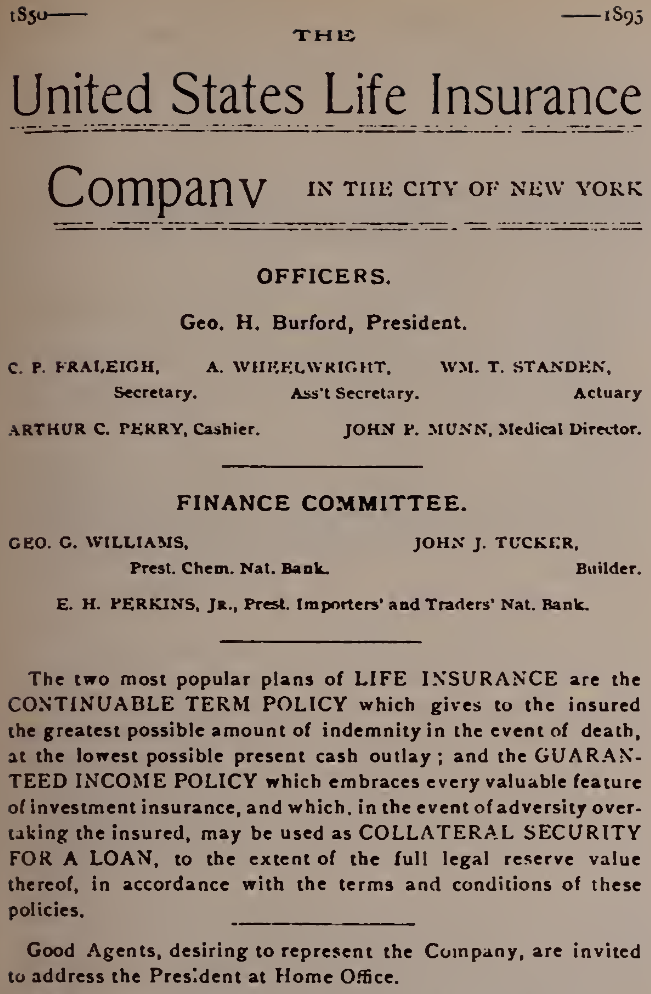
FEES

"Of physician examiners," commented the editor of the *Western Journal of Medicine* in 1868, "none will deny that many are ignorant, some are ductile, and that a few are probably corrupt."[16] Life insurance companies were to blame for this situation, argued the editor, because physicians were paid so poorly in spite of the importance placed on their examination. No lawyer, for the same pitiful sum of money, would hold a consultation to weigh circumstances, examine facts, and furnish a sound legal opinion. While the lawyer received $20 for such a function, the physician was grudgingly paid only $2. Were companies to take more seriously the services performed by the examiner, they would avoid the "broken-down, diseased, and dying incumbrances" which drew upon the resources of the companies.[16]

Because of low fees, some physicians refused outright to serve as examiners. S.H. Stout, MD, of Dallas, Texas, was so opposed to examiners' low salaries that he urged his fellow physicians to refuse to work for these "great moneyed corporations."[33] For too long, physicians had willingly accepted poor compensation on the mistaken belief that the companies were "purely benevolent institutions." If physicians would "study the subject of life insurance,. . . refuse to be no longer sponged upon by them or their agents, and [refuse] to lend medical skill for an inadequate compensation," then there would be reason to cooperate with them.[33]

RETROSPECT

Through most of the 19th century, medical schools made little mention in their curricula of the life insurance industry and the role of the medical profession in it. Medical journals across the nation, however, discussed it at length, and journals such as *The Medical Examiner*, New York (1891–1909) and *The Medical Examiner; A Semi-Monthly Journal of Medical Sciences*, Chicago (1860–1875), were founded expressly for the purpose of communicating to physicians the needs and purposes of the insurance industry as it applied to the profession. One of the earliest physicians to actually teach the subject of the physician's role in life insurance was J.T. Craig, MD, who lec-



1850—— ——1895

THE

United States Life Insurance Company IN THE CITY OF NEW YORK

OFFICERS.

Geo. H. Burford, President.

C. P. FRALEIGH, Secretary. A. WHEELWRIGHT, Ass't Secretary. WM. T. STANDEN, Actuary

ARTHUR C. PERRY, Cashier. JOHN P. MUNN, Medical Director.

FINANCE COMMITTEE.

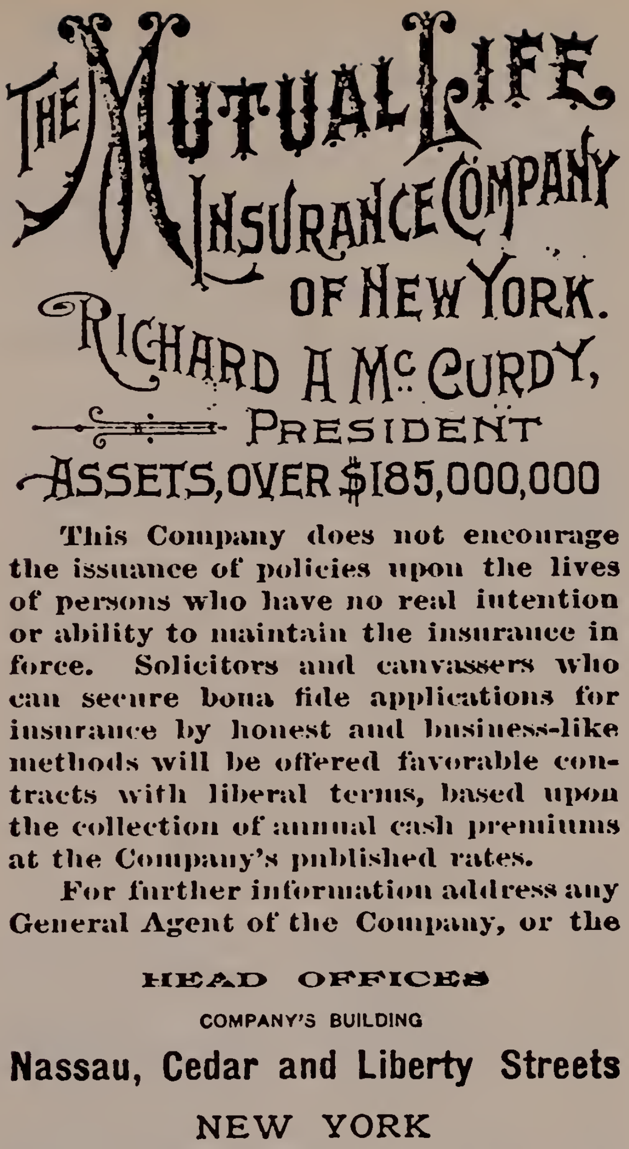
GEO. G. WILLIAMS, Prest. Chem. Nat. Bank. JOHN J. TUCKER, Builder.

E. H. PERKINS, Jr., Prest. Importers' and Traders' Nat. Bank.

The two most popular plans of LIFE INSURANCE are the CONTINUABLE TERM POLICY which gives to the insured the greatest possible amount of indemnity in the event of death, at the lowest possible present cash outlay; and the GUARANTEED INCOME POLICY which embraces every valuable feature of investment insurance, and which, in the event of adversity overtaking the insured, may be used as COLLATERAL SECURITY FOR A LOAN, to the extent of the full legal reserve value thereof, in accordance with the terms and conditions of these policies.

Good Agents, desiring to represent the Company, are invited to address the President at Home Office.

FIGURE 4. Advertisement in the April 1895 issue of *The Medical Examiner* soliciting life insurance agents for the United States Life Insurance Company.



THE MUTUAL LIFE INSURANCE COMPANY OF NEW YORK.

RICHARD A. McCURDY, PRESIDENT

ASSETS, OVER $185,000,000

This Company does not encourage the issuance of policies upon the lives of persons who have no real intention or ability to maintain the insurance in force. Solicitors and canvassers who can secure bona fide applications for insurance by honest and business-like methods will be offered favorable contracts with liberal terms, based upon the collection of annual cash premiums at the Company's published rates.

For further information address any General Agent of the Company, or the

HEAD OFFICES

COMPANY'S BUILDING

Nassau, Cedar and Liberty Streets

NEW YORK

FIGURE 5. Mutual Life Insurance Company advertisement for "solicitors and canvassers" (published in the April 1895 issue of *The Medical Examiner*).

tured to the class of 1898 of the College of Physicians and Surgeons in the Medical Department of Kansas City University. Craig believed it of sufficient importance to be part of the didactic education of every medical student. "I have the proud distinction," he noted, "of being the first lecturer to deliver a regular course on this all-important subject in this city or elsewhere in the West."[3]

Ultimately, medical examiners hoped that life insurance would carry the same level of esteem with the public as fire insurance. To achieve this end, they encouraged companies to investigate the hygiene of cities, make further inquiries into the hereditary tendencies and personal habits of the insured, and look more closely at appropriate rules of health.[29] Their recommendations carried with them the desire that medical examiners would receive respectability and remuneration equivalent to the seriousness of their decisions.

Although physician examiners oftentimes lamented their depressed socioeconomic status,[34] ironically, the meager payment given to them for performing insurance examinations was both a symptom of their economic plight as well as a safeguard against the competition caused by the fact that there were too many physicians and too few patients. The examination fee provided a modicum of financial security, though seldom did it reach a level of compensation sufficient to provide the physician examiner with an independent income base. But for the young practitioner seeking to establish a practice, or the retired or semiretired physician attempting to supplement an income, the fee from medical examinations offered a margin of financial security and served, in some measure, to reduce the need for physicians to cut their rates in an effort to compete successfully with more established physicians.

REFERENCES

1. Brown GL: Duties of physicians as examiners for life insurance. *Buf Med Surg J* 1891–92; 31:69–74.
2. Brower DR: Some observations on medical examinations. *Med Exam* 1903; 13:83–84.
3. Craig JT: Lecture on medico-insurance. *Med Exam* 1899; 9:15–18.
4. VanMeter LM: Medical examination for life assurance. *Memphis Med Mon* 1902; 12:355–361.
5. Bombaugh CC: The later phases of medical examination for life insurance. *Chicago Med J Exam* 1887; 55:189–194.
6. Foster TA: The relation of life insurance to the medical profession, with

hints to medical examiners. *Trans Maine Med Assoc* 1886-88; 35/36:303-314.
7. Temple JB: Relations of the medical profession to life insurance. *Louisville Med News* 1876; 1:111-113, 269-271.
8. Danforth IN: Life insurance and the medical profession. *Chicago Med J Exam* 1884; 49:349-359.
9. On the relation of the medical examiner to the business of life insurance. *Med Rec* 1873; 8:245-248.
10. Detmold W: Examination for life insurance. *Med Rec* 1877; 12:81-84, 97-98, 113-115.
11. Morris M: Medical supervision in distinction from medical selection in life insurance. *Med Rec* 1875; 10:51-55.
12. Finch JA: Medical examinations for life insurance. *Indiana Med J* 1896; 15:447-450.
13. Nicholson WA: Life insurance examinations. *Med Exam* 1901; 11:12-15.
14. Grant WE: Lecture on medical life insurance. *Med Exam* 1898; 8:275-277.
15. Sholl EH: The practical relation of the physician to life insurance. *Trans Med Assoc State of Alabama* 1890, 276-285.
16. Editorial. *West J Med* 1868; 3:189-193.
17. Steedman JGW: The duties of physicians to life insurance companies and their policy-holders. *St Louis Med Surg J* 1871; 8:204-218.
18. Medical experts in the selection of lives. *Med Rec* 1874; 9:222-223.
19. Burlingame FW: The relation of homeopathy to life insurance. *Trans Homeop Med Soc State of Penn* 1900; 188-194.
20. Sieveking EH: The medico-legal aspects of life insurance. *Med Rec* 1874; 9:218-222.
21. Dodge CL: Some points in the examination of persons for life insurance. *New York Med J* 1890; 52:206-207.
22. Skinner CE: Medical examination for life insurance in the field. *New York Med J* 1898; 58:234-267.
23. Sieveking EH: *Medical Advisor in Life Insurance.* Hartford, Conn, NP Fletcher, 1886.
24. Keating JM: *How to Examine for Life Insurance.* Philadelphia, WB Saunders Co, 1890.
25. Allen JA: *Medical Examinations for Life Insurance.* New York, Spectator Co, 1880.
26. Brinton W: *On the Medical Selection of Lives for Assurance.* New York, The Johns Hopkins Press, 1863.
27. VonNiemeyer F: *Clinical Lectures on Pulmonary Consumption.* London, New Sydenham Society, 1870.
28. Smith FA: Heredity and its value as a factor in life insurance. *Med Exam* 1891; 11:463-466.
29. Himes IN: Remarks and cases connected with medical examinations for life insurance. *Trans Ohio State Med Soc* 1885; 93-101.
30. Cluness WR: The medical examiner's duties in connection with life insurance. *Pacific Med Surg J* 1876; 18:412-420.
31. Green CL: Modern life insurance—Does its medical selection reflect the scientific advance of the past century. *Med Exam* 1902; 12:87-93.
32. Albright CE: The medical man in life insurance. *Milwaukee Med J* 1893; 1:352-357.
33. Stout SH: Ethical relationships of the medical profession and life insurance companies. *Texas Med J* 1894; 9:493-497.
34. Haller JS: Business and ethics of medicine, in *American Medicine in Transition, 1840-1910.* Urbana, University of Illinois Press, 1981, pp 234-279.

FROM THE LIBRARY

THE MEDICAL EXAMINER FOR LIFE INSURANCE AND HIS RESPONSIBILITIES

. . . The medical examiner should have constantly in mind two important differences in mental attitude between the patient and the applicant for insurance. In the first place, many, especially at their first examination or among the younger applicants, are extremely nervous; the idea that the examination may reveal some hidden ailment so takes possession of their minds as to disturb considerably the nervous equilibrium—to induce, as it were, a mild degree of shock. In such cases the pulse may be found extremely rapid or intermittent; there is apt to be pallor or muscular tremor. The picture is one of nervous debility or of a want of normal bodily vigor. Whenever an examiner meets with this condition he should be able by tactful handling to reassure the applicant and to re-establish the normal nervous balance; at any rate, he should make due allowance in his report for the disturbed mental state of his subject.

As to the other differences to which we have referred, when he consults his physician the patient endeavors to describe every detail of the disease of which he complains. He conceals nothing. His mental attitude is one of unreserved co-operation. When he is a candidate for life insurance, the case is very different; his memory for details is less acute; his state of mind is one of antagonism. He believes himself to be a good risk, and his bias of mind in that direction is so strong as—no doubt, unconsciously—to color his entire history. On this account a medical history for life insurance is a very different matter from that which is obtained from a patient. The patient assists his physician—the applicant for insurance does not assist the examiner. It requires time for any physician to adjust himself to this difference in mental attitude. The skilled medical examiner has learned this lesson.

It is on this account particularly that the examiner for life insurance must bring to his task complete independence of character, absolute integrity, and the ability to detect attempts at fraud. He must constantly bear in mind that he is a guardian of the interests of the company, and that upon his vigilance, judgment, and integrity depends, largely, its success or failure. . . .

S. OAKLEY VANDER POEL, MD
(*Trans Med Soc St New York* 1900; 288-294)

The physician-patient relationship in interesting times

Joseph A. Stankaitis, MD, MPH

There is an old Chinese curse that states, "May you live in interesting times." One could surmise that "interesting times" represents an era of great turbulence and flux, when events occur at a rapid pace and when familiar institutions that customarily grant stability and comfort seem to be disappearing. It would seem that the physician-patient relationship is the recipient of such a curse as it now finds itself in the midst of tremendous change and instability, creating great distress for both the physician and the patient. Indeed, things are no longer what they used to be.

Economic factors and the availability of resources influence this relationship in a major way. In years past, this was felt to be a land of opportunity, multitudinous possibilities, and unlimited resources. Now, however, there is a realization that economic resources are limited, and that some potentially painful choices will need to be made. Medical care costs are now in excess of 10% of the gross national product. The United States faces budget deficits of trillions of dollars. The federal government has understandably entered a cost-cutting phase, and health care expenditures, unlike those for defense, have and will continue to face reductions. Large corporations, which for years have allowed their health care costs to rise with apparent indifference, have suddenly awakened to this situation and realized that if they are to compete successfully with foreign corporations whose employee costs are lower, they too must pare down their health care expenses. Thus, previously "generous" or indifferent payers for health care in the United States now insist on stringent cost containment measures, and because they pay a significant portion of these expenses, they are in many ways influencing the physician-patient interaction.

Other factors contribute to this turbulence. The technology of medicine today far surpasses even the dreams of practitioners 50 years ago. The development of antibiotics, intensive care units (ICU) with respirators and vasopressors, computed tomography (CT), magnetic resonance imaging (MRI), cardiac surgery, transplant surgery, and dialysis, to list a few, has created multitudinous diagnostic and treatment options unavailable to physicians of days gone by. Many of these modalities we take for granted today; however, they raise many ethical and cost/benefit issues that previously did not have to be addressed.

Consumerism has also entered the physician-patient relationship. Whereas in the past the physician was given the upper hand in the relationship by virtue of his professional authority, this has generally been supplanted by more of a contractual relationship between equals. Most people feel that this change is highly appropriate. What this change has also produced, however, is the increased probability of conflict between these parties, especially when there is a greater realization of how inexact the practice of medicine can be.

An outgrowth of some of these events has been the ongoing malpractice crisis. Previously trusting partners in the physician-patient relationship have become adversaries. The expectations of successful treatment that modern-day medicine holds out to its patients are mitigated by the hard realities of human imperfection and inexactitude, leading the disappointed patient to seek solace in financial rewards offered by an ever-willing tort system.

Physicians also have not been immune to the influences of financial inducements. Daily, via journal advertisements, mailings, and salespersons, physicians are bombarded with information and enticements on how to increase their income by the purchase and use of various gadgets and procedures. Given the increasing expenses of medical practice, coupled with ever-decreasing revenues, such temptations are difficult to ignore.

THE PHYSICIAN-PATIENT RELATIONSHIP

In ancient Egyptian and Mesopotamian societies there was a division between the supernatural and natural treatment of illness. One sought help from a priest for the former, and from a medical "craftsman" for the latter. At least initially, the craftsmen had a much lower social status than their priest counterparts. However, with time and increased success, they acquired more respect from their patients. It was during this time that sanctions and rules to control the activities of physicians and surgeons were first issued.[1]

The Greeks were able to absorb much from these cultures, primarily via trade, and began to develop their own medical system. They developed the medical craft into a science. With the study of philosophy, Greek physicians added theoretical and philosophical knowledge to their

---

From the Department of Medicine, State University of New York at Buffalo School of Medicine, and Health Care Plan, Buffalo, NY.

Address correspondence to Dr Stankaitis, Health Care Plan, 2075 Sheridan Dr, Buffalo, NY 14223.

technical skills. The Greek study of order and philosophy naturally led to the development of codes of ethics and behavior with regard to the practice of medicine. These were traditionally attributed to Hippocrates (ca 460-377 BC), though it is unclear whether he actually authored such codes and an oath. Nevertheless, the various codes that were compiled during that time implied that the physician's responsibility to the individual patient was of paramount importance.[2]

The importance of this relationship has continued through the ages. Jewish law views the physician-patient relationship as a sacred one involving the patient, the physician, and God. It is the physician's responsibility to treat the patient, and the patient's to obtain the best medical care possible, all in order to "collaborate with God in the process of continuing creation and to mend the imperfections in the world and in men."[3] With the advent of Christianity, the treatment of ill individuals was motivated by compassion for the needy individual and by love of God.[1] A person afflicted by a malady sought out the advice and care of another person who was "empowered" with the knowledge and skill to cure or ameliorate the affliction. Thus, historically, the physician-patient relationship became the center of medicine, where the primary concern of the physician is the health and care of the patient.

Exactly what are the responsibilities of the physician? It might be said that the physician's central responsibility is to use his medical expertise to assist the patient in the preservation of health. This can be accomplished by diagnosing the patient's condition and then informing and educating the patient about it (including prognosis and treatment options); the physician can then recommend a course of action that in his opinion constitutes the best approach for the problem; and finally, the physician can carry out the alternative chosen by the patient.[4]

The most crucial aspect of this sequence of events is the physician-patient relationship, which should and must be more than just a business relationship. The physician is a member of a profession, and this distinguishes the physician's activities from those of the automobile mechanic, plumber, or other craftsman. The word *profession* originates from the Latin word *profiteri*, meaning to declare aloud, or to make a public avowal. Thus entry into the profession was a public declaration of belief and intent to practice certain ideals. Through the ages it also came to mean a declaration of the possession of special skills to be used in the service of others.[5] Thus, the profession of medicine signifies the commitment of the special skills of medical knowledge and expertise to the service of others.

What held true for Peabody 60 years ago still holds true for clinicians today: "The good physician knows his patients through and through, and his knowledge is bought dearly. Time, sympathy and understanding must be lavishly dispensed, but the reward is to be found in that personal bond which forms the greatest satisfaction of the practice of medicine. One of the essential qualities of the clinician is interest in humanity, for the secret of the care of the patient is in caring for the patient."[6] In order to properly care for any patient, a physician needs to make a tremendous investment of self in getting to know the patient. Furthermore, the patient-physician relationship demands that patients put themselves in a very vulnerable position. They must undergo intense scrutiny, of both their bodies and the most intimate details of their lives. There needs to be a feeling of mutual respect between these parties for this relationship to survive and be successful.

This intimacy forms the heart of the physician-patient relationship. Each relationship is unique. Each patient has his own particular needs, fears, standards, and moral background. Thus no matter how each physician-patient relationship is structured, as Pellegrino[5] states, "the right decision is the one that is good for this patient—not patients in general, not what is good for physicians, for science, or even for society as a whole." Thus, this is indeed a very special professional and human relationship, between a potentially vulnerable individual seeking assistance in healing, and an individual professing to help and heal. One can be a physician only if one satisfies the essence of the medical act: that is, from within the physician-patient relationship, to come to the conclusion that is best for this particular patient.

ROLES WITHIN THE PHYSICIAN-PATIENT RELATIONSHIP

The roles of the physician and the patient have evolved through the ages. At least until the past few decades, paternalism on the part of the physician traditionally was a key element of this relationship. The attitude on the part of both physician and patient was that "doctor knows best." This was based on trust in the physician's technical skills and moral stature, and on the attribution of "magical" powers to the healer, and was characterized by patient dependency and physician control. For those times, it probably was appropriate. Few complex technical modes of diagnosis and treatment were available, and any major impact on morbidity and mortality essentially took place not as a result of medical advances, but because of improvements in public health.[7] Patients found great comfort in the presence of their physician during trying times, and this presence was greatly appreciated for its therapeutic benefits. This all seemed most appropriate when viewed in the context of the ancient precept, "above all, do no harm."

As medicine realized more scientific advances, the physician was equipped with a wider variety of options that could help the patient. Trust in the physician became a more crucial part of the relationship; the physician had to serve as the patient's advocate and represent the patient's interests in medical matters. The patient was extremely vulnerable in the situation in which diagnostic and therapeutic decisions needed to be made. Since most patients had little knowledge or understanding of the medical situation, they had to put their trust (and lives) in the judgment of the one person who knew them intimately and had mastery of the field, the physician. Thus paternalism, in the form of blind trust, ruled this intimate relationship.

As technology became more complicated, lay people also became more educated and sophisticated. They no longer blindly followed the treatment regimens prescribed by physicians without a clear understanding of why such regimens were appropriate. A clear example of this is the history of the treatment of breast carcinoma. For years, women afflicted with this cancer were subjected to the disfigurement of the radical mastectomy. It was through the insistence of lay women and the courage of researchers such as George Crile that less disfiguring procedures were found to be equally effective. People realized that they could and should provide input into medical decisions that affected their lives. Thus paternalism began to give way to a contractual model for the physician-patient relationship.

In this type of relationship, the physician enters into a contract with the patient, each party being viewed as equal. The physician pledges to commit his medical knowledge and skills to the service of the patient. The physician is compensated for his role under this contract; the patient is seeking help and is willing to pay the physician for it.[8] Thus the patient has sought out the physician to serve as his adviser when crucial decisions have to be made, and as the patient's advocate in the development and implementation of these decisions in the medical world. In the role of advocate, the physician speaks for the patient. However, unlike in the previous paternalistic model, the physician does so with the knowledge and informed consent of the patient. This is a special role. The physician is not merely a mouthpiece or a robotic medical technician employed by the patient. The physician works as an aggressive partisan for the betterment of the patient's health, and since it is a contractual relationship, the physician brings his moral ethics into the relationship. He tries to honestly recommend what he thinks is medically in the patient's best interest, and not just what the patient wants. Although there has been a shift in decision-making power to a much more equal basis, the physician also has the added responsibility of educating the patient and of obtaining informed consent. This new autonomy has also brought into question issues of cost, convenience, quality, and choice in health care provided to the patient.

These changes naturally led to a consumer awareness on the part of patients regarding choices in health care plans, physicians, and procedures that might be performed on their bodies.[9] The previously close and trusting allegiances to individual physicians and institutions were fading. In an upstate New York community, for example, an employer offered its employees a choice between continuing the traditional health insurance, which had a sizable deductible, or entering a health maintenance system that required almost no out of pocket expenses for the subscriber. Overnight approximately 30,000 people switched to the new program, many giving up long-standing relationships with physicians who did not participate in the health maintenance organization. This age of consumerism in medicine highlights a greater distancing between the physician and the patient. Previously clear distinctions between a professional relationship and that of a craftsman and client seem at times to be less clear. This distancing certainly complicates the malpractice situation.

According to Siegler, the patient-physician relationship has now entered the Age of Bureaucracy.[10] This is a time of cost containment and cost efficiency; pressures have been placed upon the physician by employers, hospitals, insurance companies, the government, and even investor-owned hospital chains. The distancing between the physician and patient is becoming such that the physician-patient relationship is now regarded by many as the provider-consumer relationship. It seems that something of great value is in jeopardy of being lost.

MEDICAL ECONOMICS

Who is to blame for the rise in health care costs that has led to the implementation of various schemes to control them, schemes that also threaten the physician-patient relationship? We can blame many parties—physicians, patients, employers, insurance companies, lawyers, and government.[11]

The Medicare and Medicaid programs were implemented in good faith to assist those who needed quality health care: the elderly and the impoverished. Until recently, reimbursement for most of these services was cost based. Consequently, there was an incentive for hospitals and other institutions to expand and to acquire expensive, sophisticated equipment, since these costs would be readily reimbursed by governmental funds. There seemed to be a bottomless well from which to obtain funds for such expansion. This fostered a style of medical practice that encouraged the use (sometimes inappropriately) of these resources. During this time, the technology of medicine made great, though expensive, advances, with the development of CT scanners, magnetic resonance scanning, new pharmaceutical agents, and so on, all of which drove up the costs of medical care.

Commercial insurers found it to be to their benefit to encourage practice methods that would increase premiums paid to them. The greater the premiums paid, the more capital would be available to these companies for investment purposes. American corporations and unions negotiated contracts that offered extensive health care benefits for employees since these benefits were not viewed by the government as taxable income. Unlike most insurance products, most health insurance plans had low or nonexistent deductibles, which encouraged beneficiaries to increase their utilization of health services. Physicians certainly complied, since it satisfied the patient and provided them with revenues while someone else was paying the bill. With such seemingly never-ending resources, the application of many costly medical innovations was not fully examined for their costs/benefits to both patients and society.

A situation such as this could not continue forever, and indeed we presently are witnessing a rude awakening to this reality. Government is faced with huge deficits and is now scrambling to implement containment mechanisms for health care expenditures. Employers cannot effectively compete with foreign companies and are unwilling to fund expensive health benefits, and insurance companies are devising newer and different health care plans that will meet the needs of employers yet remain profitable. People who have been used to carte blanche medical programs now must choose between programs that restrict subscriber choices and those that require higher out-of-pocket expenditures. The physician is caught in the midst of this turbulence.

Many health economists believe that it is the physician, not the patient, who controls most of the health care costs. Patients do have control when it comes to the selection of the physician and the extent of their demand for health care. However, most medical decisions are prescribed and implemented by the physician. It is the physician who advises the patient which diagnostic or treatment options should be exercised; it is the physician who generally possesses the professional expertise and knowledge regarding these options; and it is the physician who ultimately orders the implementation of these options. Thus, physicians tend to be the primary decision makers, either directly or indirectly, in the health care system.[12] It therefore is not surprising that a substantial amount of pressure is being applied by insurers, government, employers, and society in general to contain the costs that are generated in the care of patients. Unfortunately the physician cannot escape these pressures.

Besides these external influences, the physician is also experiencing increased personal financial pressures that certainly affect his relationship with patients. The cost of a medical education has risen dramatically over the past 20 years. The tuition fees of many private medical schools are approaching $20,000 a year. If one adds on to this the costs of living while in school, it is not surprising that a substantial number of medical school graduates have educational debts of $80,000–$100,000. This, in the face of rising malpractice premiums and ever-increasing start-up costs, makes entry into the private practice of medicine difficult economically.

Reimbursement from sources such as Medicare and Medicaid has been essentially frozen for many physicians already in practice; some states (such as Massachusetts) do not allow "balance billing." With an increasing number of new physicians being produced and the exodus of many patients into managed care systems, many physicians are experiencing a gradual loss of income in comparison to prior years. Many (especially younger physicians) have sought employment with managed care systems as a means of obtaining a secure form of income. Given this state of affairs, it is not surprising that many physicians in private practice are looking for other ways to supplement their incomes. It is not uncommon to find increased use of office procedural and laboratory services, investment in larger capital items such as CT scanners, ultrasound, etc, and investment in for-profit medical systems.

One wonders what effect such actions might have on the physician in his relationship with the patient. If Dr Jones has invested in a CT scanner, might he not be influenced to recommend a CT scan of the head for his patient who complains of headaches? In pre-ownership days, he would have performed a thorough neurological examination, and probably would have declined to perform a CT scan, satisfied with the diagnosis of migraine, which indeed the patient had. What about the physician who insists on performing flexible sigmoidoscopy yearly on asymptomatic patients for screening purposes, and then bills for this procedure as a limited colonoscopy? Are these and other similar actions and procedures truly in the patient's best interest? From the patient's perspective, it might appear that these actions are occurring for the physician's benefit under the guise of providing the patient with "top quality care," when in reality there is little or no benefit to the patient. One must agree that the benefit of many procedures is unclear. However, one might question the integrity of those physicians who are proponents of various controversial—and expensive—procedures who also happen to be the owners/shareholders of the equipment required for these procedures. There is an incredible temptation for well-meaning physicians to place their own interests above those of their patients.

Employment of physicians by various health care systems poses another different influence. An organization whose goal is to minimize expenses in providing health care can pressure physician employees to cut corners in providing care. These pressures can take the form of performance bonuses, capitation agreements, bounties, and salary. These dangers do exist. However, unlike their counterparts in fee-for-service private practice settings, many managed care systems have voluntarily elected or been mandated by employer groups or governmental agencies to have quality assurance programs in place to monitor the care which they provide to their members. This of course would not eliminate all abuses of such a system. However, it is a safeguard that encourages the physician to provide the top quality care that the patient requires.

THE PHYSICIAN'S RESPONSIBILITY TO SOCIETY

The physician also has a relationship with society in general. Physicians are indebted to the community for the monies spent to support their education, for the patients who were kind enough to share themselves with them in mind, spirit, and body during their training for the profession, and for the generally high level of prestige and income they enjoy as a result of their work.[13] Physicians as individuals and as a group are privileged to hold an elevated station in American society. Much of this is derived from the services they perform for individuals and the aggregate public good. One might speculate that some of this regard could also be a vestige of the belief in the "magical," at times unexplainable, power over life and death that physicians may appear to have. In any event, it seems that physicians should play a special role in ensuring the welfare of society, especially within the realm of health. Society has provided them with many privileges, some of which are rarely granted to other members of society, and as one's stature within society grows, so does one's responsibilities. Under the ancient principle of *noblesse oblige*, physicians have a special duty to promote the welfare and good of others.[14] The American Medical Association has recognized such an obligation and suggests that "the honored ideals of the medical profession imply that the responsibilities of the physician extend not only to the individual, but to society."[15] Thus, in the words of Veatch, the physician has "the well-being of both the individual and the community as an essential responsibility."[2]

This point of view may substantiate and validate the pressure now being placed on physicians to respond to and participate directly in cost containment efforts. One result of this pressure is the development of the "gatekeeper" role for primary physicians. Physicians have actually played such a role previously, since by tradition and law many therapies have had to be prescribed and implemented by a physician. This gatekeeper role has now acquired a component of financial control. In addition to ensuring the quality and coordination of care for the patient, the primary care physician has now become the fulcrum for the curtailment of costs.[16] Gatekeepers determine the necessity for specialized care, be it hospital, subspecialty, or supportive care referral. Without such screening, reimbursement for services may be denied. Thus the primary physician has become not only the controller of entry to medical care, but also the controller of the use of health care resources and the guardian of society's health care coffers.

There have been dramatic changes in the attitudes of government and many other players in the health care drama. Government, which was previously generous, has cut back on health expenditures through such methods as diagnosis related groups and managed care systems. For-profit hospital chains, which previously encouraged their affiliated physicians to maximize hospital charges by increasing the use of ancillary services and procedures, now encourage physicians to decrease hospital length of stay and minimize the use of expensive procedures. In all likelihood there has been much inefficiency and "fat" within the health care system. However, it is unclear how far cost cutting can go before patients experience deleterious effects from these efforts. There is a tremendous risk that physicians might be persuaded that their primary responsibility is the implied obligation for societal good in terms of cost containment.

CONFLICT OF OBLIGATION TO THE PATIENT VS SOCIETY

Given the current state of affairs, it is easy to see that in terms of health care, the needs of the individual and the desires of society are headed on a collision course, with the physician being in the unenviable middle position. On the one hand, the individual patient has placed his confidence in the physician as his advocate, who is doing all that is reasonably possible to promote and maintain the patient's well-being. On the other hand, society, as government, employers, and insurers of health care, is exerting pressure on the physician to restrict health resource usage in order to reduce rising costs. This highlights an incongruence between the value of an identifiable life and that of a statistical life.

In the physician-patient relationship, physicians deal with the value of an identifiable life. As Peabody has stated,". . . the secret of the care of the patient is in caring for the patient."[6] If he is to provide excellent health care, the physician must develop a personal, caring, mutually respectful relationship with his patients, and must have a clear understanding of the patients' needs, fears, and concerns. He must know each patient as a unique individual, with his own individual history. Can physicians as individuals give a monetary value to a spouse's life, a child's life, a close friend's life? It would be almost impossible to do so. If physicians were faced with a life and death situation involving someone dear to them, money would be no object. Yet a similar emotional concern faces the physician who has a caring relationship with his patients. In each case the physician is dealing with an identifiable life.

Society, via government, insurers, employers, and politicians, in making global decisions, usually deals with statistical values for life. For example, is the installation of a traffic signal for a particular intersection worth its cost of $40,000 if perhaps one life would be saved? In all probability, this decision would be made based on budgetary criteria. If indeed a life would be saved by the presence of this particular signal, no one knows whose life would be saved. We cannot give that life a name or a face. If we knew ahead of time whose life it would be, in all likelihood increased efforts, probably spearheaded by the identified individual, would be made to guarantee the installation of that $40,000 traffic signal.

This is the situation facing physicians today. Budgetary decisions for resource allocation are being made by various parties dealing with faceless, statistical lives, while physicians are faced with applying the impact of some of these decisions to the identifiable lives of their patients. Society is hoping that aggregately physicians will accordingly respond to its decisions based upon statistical data. Physicians, in order to remain true to their patients, place a much higher value on the identifiable life, and must continue to make the welfare of their individual patients the foremost consideration in these decisions.

There must be a middle ground between these at times conflicting views of the value of life. On the one hand one can advocate arbitrary criteria in order to conserve resources, such as: "no person over the age of 75 will be cared for in the intensive care unit." At the other extreme, one could encourage cardiac transplantation for all patients with heart disease, no matter what age. Both of these proposals are not likely to be viewed as responsible, yet they do highlight several points. Because we are in an era of financial constraint in health care, we cannot afford the reckless application of health care technology simply because it exists. Heroic procedures, especially high-priced ones, should be implemented only after careful consideration of whether the patient will truly benefit, with an improved quality of life, and whether such an improvement is worth the cost to the patient and society in general. Heart transplants simply cannot be provided on demand; they are too costly. Furthermore, arbitrary exclusion from care without careful analysis and consideration is irresponsible. If the success rate of cardiac transplantation is markedly diminished after a certain age, this would support setting an age limit for transplantation. Use of health resources needs to be evaluated on an individual basis in the context of what society can afford to underwrite. If a patient would meaningfully benefit from the care provided and society can afford to provide this care, the patient should not be excluded from care. The physician's role is to determine with and for the individual patient what would constitute responsible use of available resources; what would constitute meaningful care for the patient; and how the patient's quality of life can be improved by the proposed therapy. There are no simple answers to these questions, but they are questions that need to be explored by the physician in the milieu of the physician-patient relationship.

All of these issues arise under the condition that health care resources are to be expended in the present economic situation. No one has yet determined what portion of our nation's resources ought to be used for the provision of health care. One can enter into the "guns versus butter" discussion, and such a debate needs to occur at a national level. Furthermore, equity issues need to be addressed. Since there apparently are no correct answers to these issues, they must be debated by all responsible sectors of society, with the realization that difficult choices will need to be made. However, to ignore the effects of current constraints on resources while awaiting the outcome of the debate will lead to disastrous consequences for physicians, their patients, and the health care system.

CHOICES FOR THE FUTURE

These are indeed interesting times for the physician-patient relationship. This should not be viewed as a curse, however, but as a challenge to physicians to take an active role in making the practice of medicine better than ever. As a unique profession, different from any other, medicine needs to retain the caring component of the physician-patient relationship if it is to survive. Despite the pressures placed on patients—by insurers, lawyers, and other groups—to view themselves as consumers, it is and will continue to be important for the physician to relate to his patients as patients, not as consumers. The primary concern of the physician should be the maintenance of a close relationship with the patient so that together they can choose the best diagnostic and treatment options.

In so doing, the physician can still execute his duties on behalf of the patient in harmony with the demands of society. One would hope that in response to the needs of society, and without compromise to the patient, the physician will educate himself as to what is efficient, appropriate, and effective medical practice. Such knowledge would not only help eliminate waste of medical resources, but also optimize the care given to the patient. This can only improve the relationship with the patient, and minimize some of the dangers of malpractice by enhancing the standard of practice.

We cannot assume that physicians entered medicine primarily for monetary gain. If this were the case, many would have entered other, more lucrative fields. Nevertheless, it is imperative that physicians recognize the problems that financial self-interest can bring to the physician-patient relationship. We need to examine carefully the reasons we entered this profession. Physicians, especially those in primary care, should honestly question whether their direct ownership of various expensive technologies and equipment is really in the patient's best interest rather than their own. A careful examination of one's motives and responsibilities should hopefully lead to an appropriate decision. Again, this should make the patient in the relationship more of a patient rather than a consumer.

A unionization movement for physicians has developed in New York State and other states. Although this may be an effective means of representing physicians' concerns regarding the economic issues they face, such an effort widens the gap between the physician and patient and further distorts the distinction of medicine as a profession rather than a craft or trade group. If such a movement were to grow significantly, patients would view physicians as only providers of care, further encouraging the view of a provider-consumer relationship. If quality of medical care is the major concern, then the county and state medical societies are legitimate and appropriate vehicles for representing physicians' concerns on these issues. They must strive to support efforts for technology assessment, for quality assurance of medical care rendered, for effective physician peer review, for the innovation and development of new and better ways of providing care, and for representation of the profession in the debate concerning the equity of the allocation of health care resources.

Economic issues have become a reality in health care. It is the responsibility of physicians to acquaint themselves with these issues in order that, either as individuals or through their medical societies, they may represent both the physician's and the patient's viewpoints in society's debate regarding the direction that health care is to take. To avoid this challenge might be construed as an abdication of the responsibilities bestowed on the profession by society for its benefit and that of the physician's patients. Unfortunately, the response to this challenge has tended to be reactive in nature, rather than proactive. It is understandable that many would like to see the return of the "good old days," however, the reality is that conditions will never be the same. Such resistance to change has allowed businesspeople, bureaucrats, and other nonphysicians to take the initiative in the development of various health care models and schemes, some of which are highly unpalatable for physicians and patients. It is critical that physicians take a proactive role in the design and development of health care delivery systems that will allow the preservation of the physician-patient relationship and the delivery of quality health care. Since no one group can succeed by itself, physicians will need to project a posture of cooperation and willingness to constructively approach problems that arise from the interests of the many diverse groups in the health care drama. Furthermore, physicians will need to spearhead a drive to educate patients as to what constitutes quality health care and thus to modify patient expectations and demands from the health care system.

Many models of health care delivery are presently under consideration or are already in operation. For example, one sector of the health care field, the health maintenance organizations (HMOs), has raised the possibility of providing cost-effective care comparable in quality to that of traditional fee-for-service care.[17] Preferred provider organizations (PPOs) are being considered as a possible means of allowing physicians in private practice the capability of continuing to provide high quality care for their patients in conjunction with cost containment. There are many other models worthy of consideration. If the development and implementation of a system of care that is cost effective yet does not compromise the patient's interests can be achieved, it behooves physicians to seriously consider its adoption in the practice of medicine.

In 1984 the American College of Physicians produced a position paper which stated:

> Society has conferred great authority on medicine in the belief that physicians will use such power for the benefit of patients . . . Like any other good citizen, the physician should strive for the well-being of the community and of society. He should support community health endeavors. In particular, he should seek to use all health related resources in a technically appropriate and effective manner and to husband limited resources. He should conduct himself so as to merit the respect of his community, both as a professional and as an individual citizen. . . Under the covenant of personal medical care the physician is ordinarily the advocate and the champion of his patient, upholding the patient's interests above all others.[18]

Thus the challenge to the survival of the physician-patient relationship—and for that matter, of medicine as a profession of science, ethics, and caring—has been made. Action or inaction on the part of physicians will result in repercussions that will last for many years to come. Only then will we know for certain whether these interesting times were truly a curse, or were the opportunity offered to raise medicine to a better and higher state.

Acknowledgments. The author thanks Howard H. Hiatt, MD, and Vickie Myers Stankaitis, RPA-C, for their encouragement and help in conceptualizing the issues addressed in this paper. The Kellogg Foundation provided generous support during the study of these issues.

REFERENCES

1. Amundsen DW, Ferngren GB: Evolution of the patient-physician relationship: Antiquity through the Renaissance, in Shelp EE (ed): *The Clinical Encounter.* Boston, D. Reidel Publishing Co, 1983, pp 3-46.
2. Veatch RM: *A Theory of Medical Ethics.* New York, Basic Books Inc, 1981.
3. Franck I: Jewish religious law as a model of the patient-physician relationship: A comment on Professor Brody's essay, in Shelp EE (ed): *The Clinical Encounter.* Boston, D. Reidel Publishing Co, 1983, pp 141-148.
4. Jonsen AR, Siegler M, Winslade WJ: *Clinical Ethics.* New York, Macmillan Publishing Co Inc, 1982.
5. Pellegrino ED: *Humanism and the Physician.* Knoxville, Tenn, University of Tennessee Press, 1979.
6. Peabody FW: The care of the patient. *JAMA* 1927; 88:877-882.
7. Powles J: On the limitations of modern medicine, in *Science, Medicine and Man.* Elmsford, NY, Pergamon Press, 1973, vol 1, pp 1-30.
8. Quill TE: Partnerships in patient care: A contractual approach. *Ann Intern Med* 1983; 98:228-234.
9. Haug M, Lavin B: *Consumerism in Medicine: Challenging Physician Authority.* Beverly Hills, Calif, Sage Publications Inc, 1983.
10. Siegler M: The progression of medicine. From physician paternalism to patient autonomy to bureaucratic parsimony. *Arch Intern Med* 1985; 145:713-715.
11. Feldstein M, Taylor A: *The Rapid Rise of Hospital Costs.* Council on Wage and Price Stability, 1977.
12. Enthoven AC: Consumer-choice health plan (first of two parts). Inflation and inequity in health care today. Alternatives for cost control and an analysis of proposals for national health insurance. *N Engl J Med* 1978; 298:650-658.
13. May WF: Code, covenant, contract, or philanthropy. *Hastings Cent Rep* 1975; 5:29-38.
14. Ballantine HT: Annual discourse—the crisis in ethics, anno Domini 1979. *N Engl J Med* 1979; 301:634-638.
15. American Medical Association: Principles of medical ethics, 1957.
16. Eisenberg JM: The internist as gatekeeper. Preparing the general internist for a new role. *Ann Intern Med* 1985; 102:537-543.
17. Sloss EM, Keeler EB, Brook RH, et al: Effect of a health maintenance organization on physiologic health. Results from a randomized trial. *Ann Intern Med* 1987; 106:130-138.
18. Ad Hoc Committee on Medical Ethics, American College of Physicians: American College of Physicians Ethics Manual. Part I: History of medical ethics, the physician and the patient, the physician's relationship to other physicians, the physician and society. *Ann Intern Med* 1984; 101:129-137.

FROM THE LIBRARY

THE PHYSICIAN AND THE PATIENT OF THE FUTURE

. . . We build on the experience of the past. The rapid strides in medical discoveries of the last two generations, together with the more practical employment of sanitation and preventive medicine, have already effected great changes in the actual duties of the practicing physician. The daily round of our immediate predecessors was made up largely of the treatment of infectious and contagious diseases. The day book of the family physician usually recorded visits to families for the treatment of measles, scarlet fever, diphtheria, typhoid fever, or malaria only. He was educated to treat disease. He knew little of preventive medicine, sanitation, or hygiene, nor did his medical education provide such knowledge. Older living practitioners know of the changes which mark the new era only by keeping abreast of the times.

National, state, and municipal departments of health have gradually become powerful and controlling factors in preventive medicine, sanitation, and hygiene. Health and welfare organizations, both national and local, have also invaded the field of preventive medicine. Great industrial organizations have introduced various health measures, believing it to be for their own best interests from an economical standpoint, as well as for the benefit of the hundreds of thousands of employees. Pay clinics and group practice have made some slight imprint on the modes of practice of to-day. Furthermore, the publicity work of all these agencies, together with that of our own Association, has gradually created a demand from the public for more information regarding personal health. The outcome of these measures has led to confusion regarding not only the present but also the future of medical practice. But the confusion of the present need never become chaos in the future, provided the medical profession prepares to meet the demands of the patients of the future. . . .

WENDELL C. PHILLIPS, MD
(*NY State J Med* 1926; 26:375-378)

Mandatory Medicare assignment

MARIO V. MENGHINI

Physicians throughout the country are currently expressing grave concern over varying state proposals mandating mandatory acceptance of Medicare assignment. It is ironic to note that the Congress, which enacted Medicare in 1965, was so concerned about the possibility of inadequate physician participation that it sought to give every assurance the new program would not be used as a vehicle to interfere with physician/patient relationships. This assurance finds expression in the first section of the Medicare Act, which remains unchanged to this day: "Nothing [in Medicare] shall be construed to authorize any federal officer or employee to exercise any supervision or control over...the manner in which medical services are provided. . . ."[1]

The congressional concern over avoiding government interference in traditional physician practices through the Medicare Act is especially apparent in the Act's provision governing the billing of program beneficiaries. Congress included a provision under which a physician could elect to accept assignment and limit fees to an amount determined by the Medicare carrier. At the same time it provided physicians with the option to decline assignment acceptance.

Congress specifically gave physicians the option to deal directly with their Medicare patients and to charge accordingly. This set of alternatives finds expression in 42 USC § 1395u(b)(3)(B), which specifies that payment under Medicare will be made either "(i) on the basis of an itemized bill, or (ii) on the basis of an assignment under the terms of which...the reasonable charge is the full charge for the service. . . ."

An assignment is an agreement wherein the Medicare beneficiary transfers to the physician the right to payment of covered services identified on a submitted Medicare claim form. The physician, in return, agrees to accept the reasonable charge determined by the carrier as the full fee (encompassing deductible and coinsurance amounts) for the services rendered. In essence, the physician who accepts assignment is precluded from billing the beneficiary more than the deductible and coinsurance amounts predicated upon the carrier's determination of a reasonable charge. Under this arrangement, it is the physician who elects to accept assignment, whereupon he or she will be paid directly by the carrier; or not to accept assignment, in which case the carrier will pay the beneficiary directly.

On April 12, 1984, the House of Representatives rejected a proposal calling for mandated assignment tied to a one-year freeze in the prevailing charge levels for services provided to hospital inpatients. Since that action, the Congress created the physician "participation" program under the Deficit Reduction Act of 1984, whereby physicians are provided the option to participate in the Medicare program and accept all Medicare claims on assignment. Furthermore, on April 8, 1987, a mandatory assignment amendment to a proposal to incorporate acute care catastrophic coverage into Medicare was offered and withdrawn in a meeting of the Health Subcommittee of the House Ways and Means Committee.

Despite the foregoing, a startling and ominous development occurred in 1985 when the Massachusetts legislature unanimously enacted a law requiring physicians, as a condition of licensure or license renewal, to agree not to charge or collect from Medicare beneficiaries any amount in excess of the reasonable charge for that service, as determined by the Medicare carrier in compliance with guidelines emanating from the Office of the Secretary of the Department of Health and Human Services.[2] That law directs the Massachusetts Board of Registration in Medicine to:

> ...require as a condition of granting or renewing a physician's certificate of registration, that the physician, who if he agrees to treat [a Medicare beneficiary] shall also agree not to charge to or collect from such beneficiary any amount in excess of the reasonable charge for that service as determined by the United States Secretary of Health and Human Services.

Physicians would still be required to bill for deductible and coinsurance amounts, except in cases of financial hardship.

The Massachusetts Medical Society (MMS) and the American Medical Association (AMA) sued to challenge the constitutionality of the state statute on the grounds that the law:

- was inconsistent with the federal Medicare Act, and conflicted with federal Medicare statutes, ie, the US Constitution requires that a federal law always take precedence over a state law (the "Supremacy Clause");

Address correspondence to Mr Menghini, Director, Division of Socio-Medical Economics, Medical Society of the State of New York, 420 Lakeville Rd, Lake Success, NY 11042.

- was violative of due process;
- conflicted with the congressional intent to allow physicians an option in the acceptance of assignment;
- would diminish the access of Medicare beneficiaries in the state to medical care as physicians withdraw from involvement in the Medicare program;
- would have an adverse impact on medical innovation, with the likelihood that the best qualified physicians would refuse to treat Medicare patients; and
- would have a disparate impact on rural areas.[3]

Although the mandatory assignment concept has been rejected legislatively on the federal level, on June 5, 1986, the Federal District Court for the District of Massachusetts upheld the state's statute, ruling that it does not violate the Supremacy Clause of the Constitution (ie, Massachusetts state law was not found to be in conflict with any portion of the federal Medicare Act); encroach upon any field occupied by Congress; or violate due process.[4]

The case was appealed to the First Circuit Court of Appeals, which affirmed the lower court's decision in an opinion issued on March 30, 1987.[5] The opinion of the US Court of Appeals in Boston had a significant impact on the effort to protect a physician's right to decline acceptance of Medicare assignment when mandated to do so as a condition of licensure. Even though Congress had on various occasions voted to give physicians the option of taking or rejecting assignment, the court asserted that it is permissible for states to force physicians to accept assignment. It was the court's assessment that Congress, in enacting the Medicare law, was not clear enough in delineating physician freedom to accept or decline assignment. In *Massachusetts Medical Society v Dukakis*, the appeals court held that states traditionally have regulated physician fees, and when Congress gave physicians the option of taking Medicare assignment, it was not explicit enough in asserting that this option is actually a "right" preventing states from fee interference.[5]

AMA lawyers contested that the distinction the appeals court made between an "option" and a "right" to reject assignment seemed meaningless, in view of Congress' explicitly giving physicians the option of balance billing. Consequently, the question arose as to whether a state can nullify an option Congress established in a federal program. That is what the AMA will argue in applying for a review of the case by the US Supreme Court. Whether the court will decide to hear the case, however, is questionable since it generally hears only about 5% of the cases that come before it, and then usually because there is a difference of opinion among appeals courts in other similar cases.

As the law in Massachusetts stands now, physicians have no right to decline assignment, no matter what the market dictates or the individual patient can afford to pay. The state assistant attorney general has asserted that the appeals court decision will allow other states to pass similar laws. However, if the Supreme Court does hear the case, or if Congress further clarifies its intent that Medicare operates more effectively when physicians are not subject to restrictive government regulations, that option could be precluded.

Given the state's unique environment, it is not surprising that legislation of the foregoing nature was enacted in Massachusetts. Under state law regulating Massachusetts Blue Shield, physicians must sign a participation agreement in order to receive reimbursement from the plan, thereby precluding balance billing of patients; and Blue Shield payments are not permitted to be made to either the doctor or the patient for services performed by a nonparticipating physician.

THE NATIONAL PERSPECTIVE

Although Massachusetts is the only state to date to have enacted a law conditioning physician licensure on acceptance of Medicare assignment, similar legislation was introduced in a number of other states in 1986 and new proposals have surfaced in 1987. While the predominant approach of these proposals has thus far been similar to that of Massachusetts, bills have also appeared which tie refusal to accept the Medicare reasonable amount to violations of a state's Consumer Protection Act, allegations of unprofessional conduct and subjection to medical discipline, the grant or denial of hospital staff privileges, and a misdemeanor violation of criminal law.

The introduction of such legislation in these states is of great concern to physicians, especially with respect to their potential impact on access to quality medical care for the nation's elderly population.

Following are specific examples of the differing types of bills introduced in other state legislatures since enactment of the Massachusetts law.[6]

Acceptance of Medicare Assignment Tied to Licensure. In 1986, bills similar to the Massachusetts law were introduced in other states which, as a condition of licensure to practice medicine, require physicians not to charge or collect from beneficiaries any amounts in excess of Medicare-determined allowable charges. While none of these bills attained passage last year, similar legislation is, or was, pending in the 1987 legislative sessions in Arkansas, Connecticut, Iowa, New Hampshire, New Jersey, New York, Ohio, Oregon, Rhode Island, West Virginia, and Vermont. In Arkansas, New Hampshire, and New Jersey, the 1987 legislative proposals were defeated. While the tie to licensure was eliminated from the bill passed in Vermont, compliance with its provisions will be monitored by the State Medical Board. Vermont physicians are now required to post a summary of the law's provisions in their offices, and to accept assignment on Medicare beneficiaries with annual incomes of $25,000 or less for a single person, or $32,000 or less for a married couple (home and office medical visits are excluded from the requirement for mandatory assignment acceptance).

In Connecticut, the state legislature passed a compromise mandatory assignment bill for physicians who treat Medicare patients, with the legislation establishing a Medicare assignment contract program in the state Department on Aging. The program currently calls for the Connecticut State Medical Society to issue medical courtesy cards to beneficiaries whose incomes are less than $19,950 for individuals or $24,000 for couples, with physicians participating on a voluntary basis, accepting assignment for care given to cardholders.

After July 1, 1988, the program requires that at least:

- 85% of Medicare claims in the state be taken on assignment;
- 85% of the physicians in each county who treat Medicare patients participate in the courtesy card program;
- 80% of physicians statewide in each specialty who treat Medicare patients participate in the courtesy card program;
- 50,000 of income-eligible beneficiaries be enrolled.

If these levels are not met, the Department on Aging is to adopt regulations requiring that all physicians limit their charges to Medicare's reasonable amount for income-eligible patients. Physicians who violate the regulations would be subject to civil penalties of up to $1,000 per charge.

The Connecticut law also requires physicians with Medicare provider numbers to post their policies on Medicare payments and processing in a prominent office location. Despite the fact that the Connecticut State Medical Society's medical courtesy card program is in place for the needy elderly, and that about 75% of the state's physicians voluntarily participate, the Connecticut state legislature deemed it necessary to pass the foregoing legislation.

Acceptance of Assignment and Consumer Protection. In 1986 the Washington state legislature considered a bill which would have redefined "unprofessional conduct" to include the failure to accept the Medicare-determined reasonable charge as payment in full for patients whose income is at or below 200% of the poverty level. In addition, a coalition of senior citizen groups formulated an initiative to be placed on the 1987 ballot which would have made it a violation of the state's Consumer Protection Act for any physician to "charge, collect or attempt to collect for medical services provided to any Medicare-eligible patient, any amount in excess of the Medicare-determined charge." Ultimately, the bill did not pass, primarily because of effective interaction between the Washington State Medical Association and the state's senior citizen groups.

In Montana, a bill was introduced in a recent legislative session which would have made it a violation of the state's Consumer Protection Act for a health care provider to charge Medicare patients more than approved Medicare rates for treatment. The bill, which would have also required posting of its provisions in the place of business of a health care provider, failed to pass.

Acceptance of Assignment and Medical Discipline. In 1986, a proposal in California would have made it an element of "unprofessional conduct" for a physician to refuse to treat anyone over the age of 65 on the grounds that the person's only health insurance is Medicare or Medi-Cal. Although hearings were held on this proposal, a formal bill was not introduced last year in the California legislature. Legislation has been introduced, however, for the 1987 session.

An Ohio bill formulated at the end of 1986 and reintroduced in 1987 would also make it an element of "unprofessional conduct" to knowingly charge or collect from an individual an amount in excess of the Medicare reasonable amount "for a health service the practitioner agreed to provide. . . under Title XVIII of the Social Security Act."

Legislation recently passed by the Rhode Island state legislature and signed by the governor expands the definition of "unprofessional conduct" to include charging more than the Medicare reasonable amount for individuals and couples with income levels of $12,000 and $15,000, respectively.

An Illinois bill calling for the state's Department of Registration and Education to discipline physicians for failure to accept assignment of all Medicare claims has been introduced in the Illinois General Assembly. The bill would subject physicians who balance bill a Medicare beneficiary to disciplinary proceedings that could result in license revocation, suspension, probation, or other disciplinary action as deemed proper by the Department through the Medical Disciplinary Board. Proponents of the measure include the Coalition for Consumer Rights and the Illinois Public Action Council.

Acceptance of Assignment and Hospital Staff Privileges. A 1985 Nevada bill, which failed to pass, would have directed every licensed hospital to "require as a condition of employment or extension of privileges that a practitioner of a healing art accept as full payment for any services provided in the hospital the assignment of benefits payable pursuant to 42 U.S.C. § 1395d and 1395l."

Failure to Accept the Medicare-Determined Amount as a Criminal Violation. A bill introduced in Maryland in 1987 would have made it a criminal violation for a physician to charge a Medicare beneficiary any more than the Medicare-determined charge for that service. Violation of this provision would have subjected a physician to a fine of up to $5,000 or imprisonment of up to five years, but the Maryland bill was defeated.

It is noted that proponents of mandatory assignment include a group calling itself the National Health Care Campaign, a grassroots effort to improve health care headed by Dr Arthur Fleming, former Secretary of the US Department of Health, Education and Welfare. This group has identified mandatory Medicare assignment as among the various issues it will be pursuing in approximately 20 states, including California, Colorado, Connecticut, Florida, Idaho, Illinois, Indiana, Iowa, Maryland, Minnesota, Montana, New Jersey, New York, North Carolina, Ohio, Oregon, Pennsylvania, Rhode Island, and Washington. In some states, the state chapters of the American Association of Retired Persons (AARP) have either opposed state legislation or have taken a neutral position on state mandatory assignment because of the deleterious effect it could have on access to quality medical care for the elderly. However, it should be noted that senior citizen organizations and activist consumer groups have been promoting mandatory assignment at state levels.

Interestingly, the current rate of physician assignment acceptance on a national level is significant. For the last quarter of 1986, 68.2% of all Part B claims and 70% of charges comprising these claims were accepted on an assigned basis. This shows the continuation of a trend toward increased acceptance of assignment for Medicare Part B claims, from 51.4% in 1980 to 67.7% in 1985.

Although most physicians do not formally participate in the Medicare program, nonparticipating physicians nationwide submitted 61% of charges for covered services during the third quarter of 1986, with 46% of these services being accepted on an assigned basis.

Furthermore, it has been determined that acceptance of assignment on a claim-by-claim basis has not resulted in undue hardship to beneficiaries. Based on 1982 Health Care Financing Administration data (when the assignment rate was only 52.8%), only 1,032,600 of 25,363,000 aged enrollees (or a little over 4%) had Part B financial liability in excess of $1,000. In addition, 13,392,600 of the more than 16 million beneficiaries who used Part B services (or a little over 83%) had out-of-pocket expenses of less than $500. These expenses included the statutorily mandated deductible ($75) and coinsurance amounts.

It seems evident that mandated assignment will result in subsidizing the cost of care for a population apparently able to fulfill its financial commitments. In fact, recent statistics point out that many of the nation's elderly are in a position to meet physicians' regular charges, with elderly Americans as a group reported to have the lowest poverty rates and more discretionary dollars to spend, after taxes and expenses, than any other age group in the nation. It is also important to emphasize that mandated assignment would force a shift in costs to other segments of the population and across generations. Requiring physicians to accept assignment for patients who are not in need of such preferential treatment would turn the Medicare program into a welfare-type program for the many individuals who are not in need of subsidy. Consequently, it is logical to conclude that attaining a specific age, as opposed to actual need, should not be the criterion for preferential treatment and governmental presumption of economic need. Most noteworthy, mandated assignment would result in reduced access to services if physicians should choose not to accede to governmental encroachment and refuse to treat Medicare beneficiaries. In fact, a report prepared pursuant to an HCFA grant and published in the Summer 1983 issue of *Health Care Financing Review*[7] indicates that if physicians were placed in the position of having to decide whether to accept assignment for all of their Medicare patients or for none of them, more than two-thirds of physicians would choose to take no assignments.

The New York State Perspective

Concurrent with the foregoing activities on a national level concerning mandatory assignment, Assembly Bill No. A.6801 was introduced in the New York State legislature in May 1987 (significant action on this bill is not anticipated until late 1987 or early 1988). The bill reads as follows:

> An ACT to amend the education law, in relation to requirements for a professional license for a physician.
> *The People of the State of New York, represented in Senate and Assembly,* do enact as follows:
>
> Section 1. Section sixty-five hundred twenty-four of the education law is amended by adding a new subdivision eleven to read as follows:
> *(11) No license or registration shall be issued to any applicant under this section unless the applicant shall agree in writing that if in the course of the practice of his profession, such applicant agrees to treat a beneficiary of health insurance under Title XVIII of the Social Security Act, he shall also agree not to charge to or collect from such beneficiary any amount in excess of the reasonable charge for that service as determined by the United States Secretary of Health and Human Services.*
> § 2. This act shall take effect immediately.

The following information is included in the New York State Assembly memorandum in support of the proposed legislation:

> TITLE OF BILL: AN ACT to amend the education law, in relation to requirements for a professional license for a physician.
> PURPOSE OR GENERAL IDEA OF BILL: The purpose of this bill is to help contain health care costs for older New Yorkers.
> SUMMARY OF SPECIFIC PROVISIONS: This bill would require all physicians who agree to treat Medicare patients, as a condition of licensure, to charge Medicare patients no more than the Medicare determined reasonable fee.
> EFFECTS OF PRESENT LAW WHICH THIS BILL WOULD ALTER: Adds a new subdivision 11 to section 6524 of the Education Law.
> JUSTIFICATION: Established in 1965, Medicare is a federal health insurance program for people 65 and over and some of the disabled. Medicare premiums are paid by the elderly and out of revenues from social security taxes. Medicare beneficiaries are required to pay a deductible and copayment for the various services provided. Under the Medicare program there are two methods of payment. In the first method, the physician agrees to accept "assignment". This means that he/she agrees to accept the reasonable fee set by the Department of Health and Human Services (HHS) as payment in full for the service provided. The physician then directly receives 80% of this reasonable charge from Medicare and collects the remaining 20% from the patient. Under the second method the physician bills the patient directly for the service. The patient then collects reimbursement from Medicare that equals 80% of the charge for that service. The patient is then responsible for the remaining 20% plus 100% of any overcharge above the reasonable charge levied by the physician. The practice by physicians, of charging more than Medicare's reasonable charge, is commonly called "balance billing." In spite of the protection provided by Medicare, health care costs for the elderly remain unpredictable and continue to pose an ominous threat to their financial security and independence. This legislation will limit what physicians can charge their Medicare patients, insure that the rates they are charged are reasonable and predictable and will help to alleviate the burden of rising health care costs for older persons.
> PRIOR LEGISLATIVE HISTORY: New bill.
> FISCAL IMPLICATION FOR STATE AND LOCAL GOVERNMENTS: None to the State.
> EFFECTIVE DATE: Immediate.

The Position of the Medical Society of the State of New York

In opposition to mandatory assignment, the Medical Society of the State of New York (MSSNY) affirms that:

- Physician licensure should only be related to education, training, and ethical criteria, rather than to acceptance of Medicare assignment.
- Mandatory assignment has the potential to compound the existing two-tiered system of medical care created by the Deficit Reduction Act of 1984, which established the Medicare participating and nonparticipating physician categories.
- Mandatory assignment will have an adverse impact

on accessibility to providers and on quality of care, particularly in already underserved rural areas of the state.

- Incentives for physicians to adopt innovative technologies in a state with mandatory assignment could be discouraged, as their income is significantly reduced and spending is affected accordingly.
- Younger physicians will likely not choose to set up practice in a mandated-assignment state, and older physicians will be discouraged from continuing practice and will retire earlier.
- In view of the present 72% rate of acceptance of assignment on Medicare claims by participating and nonparticipating physicians in New York State, mandatory assignment legislation is unnecessary.
- In accordance with AMA assessments, reform of the entire Medicare program should be undertaken rather than addressing its inequities through such approaches as state mandatory assignment legislation.
- The spirit and intent of the Medicare law as originally enacted, which gave physicians the option to bill beneficiaries directly or accept assignment, should be reaffirmed under all circumstances.

The MSSNY believes that the current reimbursement system in which assignment is accepted by nonparticipating physicians on a claim-by-claim basis, and in which Medicare patients have access to nearly all physicians in the state, has not resulted in undue beneficiary liability and has not been abused by the medical profession. The MSSNY continues to urge physicians to consider each patient's financial needs when setting charges, and to accept Medicare assignment, reduce fees, or charge no fee at all in financial-hardship cases.

On behalf of both the state's elderly population and the physician community, the MSSNY:

- has been conducting ongoing liaison activities with the Statewide Seniors Action Council—a leading senior citizen group in New York State—concerning the overall negative ramifications of mandatory assignment legislation;
- plans to sponsor educational senior citizen workshops conducted by volunteer physicians throughout the state concerning the pitfalls of mandatory assignment vis-a-vis accessibility and quality of medical care;
- is preparing testimony stressing the negative impact that mandatory assignment would have on accessibility and quality of care in anticipation of state legislative hearings;
- is studying the feasibility of patient advocacy activities to assist beneficiaries with claims problems experienced with Medicare carriers;
- is assessing the potential of county medical society sponsored voluntary physician assignment acceptance programs;
- reaffirms its encouragement of fee discussions between physicians and patients, with recommendations for assignment acceptance, and appropriate fee considerations for cases of financial hardship.

REFERENCES

1. 42 USC § 1395.
2. Massachusetts Acts of 1985, ch 475.
3. Brief of the Massachusetts Medical Society and the American Medical Association to the United States Court of Appeals for the First Circuit, Boston, 1987.
4. *Massachusetts Medical Society v Dukakis*, No. 85-4312-K, slip op (D Mass, June 5, 1986).
5. *Massachusetts Medical Society v Dukakis*, No. 86-1575, US Court of Appeals, 1st Cir.
6. Division of Legislative Activities: *Mandatory Assignment: A Legislative Guide*. Chicago, Ill, American Medical Association, 1987.
7. Mitchell JB, Cromwell J: Impact of an all-or-nothing assignment requirement under Medicare. *Health Care Finan Rev* 1983; 4(4):59–78.

Bile peritonitis following expectant management of liver fracture

Stephen L. Barker, MD, David Fromm, MD

The liver is the second most commonly injured abdominal organ in cases of trauma, exceeded only by the small bowel in penetrating injuries and the spleen in blunt trauma. Since World War I, when the mortality rate was 66%, there has been a steady decrease in the mortality rate associated with hepatic injuries. Current civilian series report mortality rates of 10–20%, with exsanguination as the leading cause of death.[1] Hemorrhage from these injuries can be massive, reflecting 1,500 mL/min of splanchnic/hepatic blood flow, as well as a potential 1,200 mL/min of renal/pelvic/leg venous return in cases of injury to the retrohepatic vena cava.[2] Despite this, at least half of all liver injuries are not actively bleeding at the time of laparotomy.[3]

Recent reviews of liver injury have stressed surgical control of hemorrhage by various means, ranging from temporary packing to suture, electrocautery, microfibrillar collagen, selective hepatic artery ligation, and hepatic resection.[1-8] While hemorrhage from hepatic injury is the most dramatic and immediately life-threatening consequence of blunt trauma to this organ, delayed and long-term complications relate instead to infectious and biliary tract problems.[9] These insidious processes may become more apparent as a result of recent trends toward nonsurgical management of some cases, as illustrated in this report.

Case Report

An 18-year-old man was brought to the emergency room after a motorcycle accident. His only complaint was vague abdominal pain in the right upper quadrant. He was not in acute distress. His pulse was 54 beats/min, and his blood pressure was 120/86 mm Hg. Mild right upper quadrant tenderness was present. Pertinent laboratory findings included a hematocrit of 37%, white blood cell count of 10,100/mm^3, and a serum glutamic-oxaloacetic transaminase (SGOT) level of 283 U/L (normal range, 0–41 U/L). Plain radiographs of the chest and abdomen were normal.

Over the next 24 hours, the patient's hematocrit fell to 31%. His abdominal discomfort increased, and he noted pain in the right shoulder. A computed tomographic (CT) scan showed a fracture of the right lobe of the liver with fluid in both paracolic gutters (Fig 1). Although many physicians would have operated at this point, it was elected to continue a period of cautious observation. The patient's hematocrit slowly drifted downward, and he received two units of blood. Thereafter he seemed to improve, his hematocrit stabilized, and he was placed on a solid-foods diet.

The hepatic enzyme levels reflected liver injury. Peak values were as follows: bilirubin, 5.1 mg/dL; alkaline phosphatase, 108 U/L (normal range, 20-90 U/L); lactate dehydrogenase, 1,275 U/L (normal range, 100–225 U/L); SGOT, 665 U/L, and serum glutamic-pyruvic transaminase (SGPT), 286 U/L (normal range, 3–36 U/L). These values all slowly declined to less elevated levels.

The patient's temperature rose to 103°F 14 days after the injury. He began to complain of more generalized abdominal discomfort, and ileus was evident on abdominal radiographs. His white blood cell count was 13,300/mm^3, with 85% neutrophils and no band forms. Parenteral nutrition was begun. Paracentesis delivered dark serous fluid, with a bilirubin level of 40.7 mg/dL and a white blood cell count of 3000/mm^3 with 50% neutrophils and 50% mononuclear cells. Antibiotic therapy was begun, but the patient continued to have more diffuse abdominal pain and spiking temperatures. A repeat CT scan showed a decrease in the size of the liver fracture but an increase in peritoneal fluid (Fig 2). Abdominal distention and peritonitis were clinically evident, and the patient was taken to the operating room with a preoperative diagnosis of probable intra-abdominal abscess.

Bilious fluid, 5,200 cc, was evacuated from the peritoneal cavity. The peritoneal surfaces were grossly inflamed and were covered with shaggy fibrinous exudate. The gallbladder appeared somewhat collapsed. Adhesions to the dome of the liver were taken down, revealing a capsular defect corresponding to the recent fracture. There was no bile staining of the retroperitoneal fat behind the duodenum. Because of the uncertain source of the bile leak, a cholecystocholangiogram was performed and disclosed a transected intrahepatic tributary of the right hepatic duct with extravasation of dye through the right lobe (Fig 3). A cholecystostomy was performed, and drains were placed in the right upper abdomen. Aerobic and anaerobic cultures of the peritoneal fluid showed no growth. Bilious fluid drained from the drain sites, decreasing in volume over a ten-day period. The drains were re-

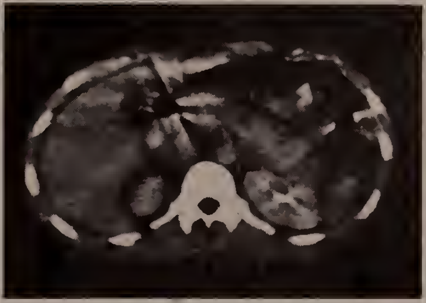


FIGURE 1. CT scan showing a large laceration of the right lobe of the liver with hemoperitoneum.

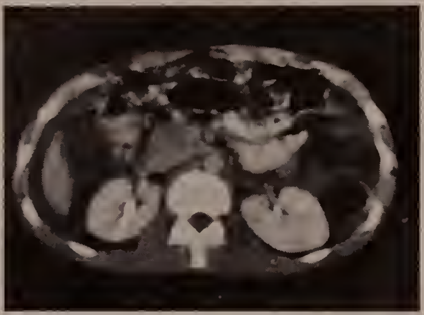


FIGURE 2. CT scan showing increasing intraperitoneal fluid.

From the Department of Surgery, State University of New York Health Science Center at Syracuse, NY.

Address correspondence to Dr Fromm, Department of Surgery, University Hospital, 750 East Adams St, Syracuse, NY 13210.

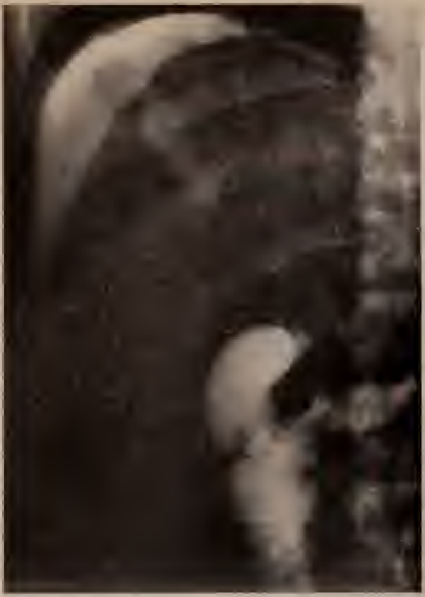


FIGURE 3. Operative cholecystocholangiogram showing the right hepatic duct with contrast extravasating into a laceration of the liver and the subdiaphragmatic space.

moved on the 16th day after surgery. The patient is doing well one year after injury.

Discussion

The true incidence of bilious leak from intrahepatic ductal injury following trauma is not known, although the incidence of biliary peritonitis and fistula has been reported to vary from 2% to 4% in many large series of hepatic trauma.[10] When intraperitoneal bile is found following a blow to the abdomen, the most likely source is a laceration or tear of the liver due to the size and relative fixation of the organ. Rupture of the extrahepatic bile ducts should be considered next, with the gallbladder being the least likely source of choleperitoneum.[11] There are reports of at least 36 cases of avulsion of the common bile duct and 101 cases of gallbladder injuries.[12,13] Conversely, reports of bile leakage from intrahepatic ductal disruption are rare.

Although the risk of postoperative bile drainage from a liver laceration is roughly proportional to the size of the wound, copious flow may occur in small wounds if a major biliary radical is disrupted. There are no reliable criteria by which such cases can be selected prior to or during surgery,[14] a time when there normally appears to be a diminution of bile flow. This has been the rationale for advocating drainage for liver wounds since Madding's[15] World War II series of 829 liver injuries. He expressed the opinion that the establishment of adequate external drainage for both bile and products of tissue injury is the most important feature in the surgical care of liver wounds.

Others also endorse the concept of drainage for all liver wounds, claiming that complications relating to sepsis and the biliary tree are almost universally related to inadequate drainage.[14] According to still others, the sole exception to mandatory drainage for all liver injuries is the laceration which is cleanly incised and superficial, similar to a hepatic biopsy wound, which may be treated by suture only.[16] The scarcity of reports of complications from intraperitoneal accumulation of bile as a result of intrahepatic rupture may be due to the widespread use of drains in cases of parenchymal laceration.

The current trend of nonoperative management in selected instances of hepatic injury leads to the speculation that the incidence of biliary complications may increase. However, complications have been few when strict criteria[17-20] are applied to such management which requires accurate definition of the degree of injury afforded by such modalities as radionuclide scanning, ultrasonography, and, particularly, CT scanning. For example, Meyer et al[19] advocate nonoperative management (in the absence of other clinical indications for laparotomy) in the presence of simple parenchymal laceration or intrahepatic hematoma that does not become enhanced following intravenous administration of contrast material, an estimated amount of free intraperitoneal blood of less than 250 mL (by its restriction to Morison's pouch), and the absence of other intraperitoneal injury. Other authors extend these criteria to include more extensive parenchymal lacerations and regard the amount of intraperitoneal fluid to be the more critical factor in electing a nonoperative course.[20] In this regard, neither hepatic scintigraphy nor diagnostic peritoneal lavage are of much value, because they do not provide data relative to blood volume lost into the peritoneal cavity. Furthermore, lavage has repeatedly been shown to be positive in the presence of small amounts of intraperitoneal blood (as little as 25 mL) and gives little information about the source of bleeding.[20]

Biliary complications have not been reported in four pediatric series (total of 59 patients) and one adult series (24 patients) of blunt hepatic trauma treated without surgery. However, bile drained in one child for 20 days after exploration for delayed rupture of a subcapsular hematoma, six days after injury.[17-19,21,22]

There is still controversy about the effect of bile in the peritoneum. Earlier in this century, much work was done on the mechanism of bile peritonitis, concentrating on bile salts, combination of bacteria and bile, as well as circulatory disturbances caused by inflammatory transudation of fluid into the peritoneal cavity.[11,23,24] There is no unanimity of opinion about the cause of peritonitis when bile leaks into the peritoneal cavity. There also exists the phenomenon of bile ascites, in which patients are without acute symptoms in spite of the presence of up to eight liters of bile in the peritoneal cavity.[25]

A recent review concludes that no factors can be identified that permit prediction of the development of bile ascites as opposed to bile peritonitis.[25] The development of bilious ascites most often follows elective biliary surgery, whereas the most common cause of bile peritonitis is spontaneous perforation. Trauma was the cause of bile ascites in seven of 24 patients and of bile peritonitis in five of 34 patients. Intraperitoneal bile cultures were uniformly sterile in patients with bile ascites, while five of 11 cultures were positive in the peritonitis group. The mortality rate for treatment of bile ascites was 8%, and this increased two and a half fold in the presence of bile peritonitis.

A biliary complication is easily diagnosed in patients with overt peritonitis, either before or during surgery. The patient who manifests vague symptoms of persistent, moderate, right upper abdominal tenderness or minimal generalized tenderness, in the appropriate clinical setting up to 28 days after injury, should be evaluated for the presence of bile ascites. This is an important diagnosis to make, as all 24 patients in a series reported from this institution required surgery for definitive treatment.[25] Paracentesis with the finding of an elevated bilirubin in the aspirate can be diagnostic.

Computed tomography or ultrasound can confirm fluid in the abdomen, but hepatobiliary scanning is the least invasive method necessary to establish the presence of a biliary fistula. Historically, this consisted of measuring the radioactivity of ascitic fluid after the intravenous administration of ^{131}I rose bengal.[26] More recently, Tc-99m IDA scintigraphy has been used successfully with high sensitivity in cases of bile leak into the hepatic parenchyma,[27,28] the peritoneal cavity, and abdominal incisions following biliary surgery and spontaneous perforation.[26,29] Delayed images should be obtained in patients suspected of having a bile leak no matter how normal the study appears within the first hour. It may take time for enough activity to build up in a collection or tract to be detectable. Weissmann et al[26] note that 14 of 23 leaks would have been missed in the absence of delayed views.

There is a subset of patients with blunt trauma to the liver that may be managed without surgery provided there is hemodynamic stability, no other operative indication, and strict CT criteria are applied. This would most often involve a contained subcapsular hematoma, although capsular tears, as in the patient described here, have been observed. Patients with a palpable liver or who are receiving anticoagulant therapy have been shown to have a higher incidence of complications and should therefore be treated more aggressively.[30] Documentation of resolution of the hematoma is necessary with serial radionuclide scanning, ultrasonography, or computed tomography.[31]

CT scanning may be useful in selecting patients who will not have hemorrhagic complications resulting from nonoperative management. Unfortunately, CT scanning cannot be used to predict septic or bilious complications. Increasing abdominal fluid on serial CT or ultrasound as well as heightened clinical suspicion should lead to more liberal use of hepatobiliary scanning and early diagnosis of bile leaks.

In this patient as well as in the majority of the cases reviewed, prompt exploration in the event of a complication, with debridement, lavage, and abundant drainage, has led to a successful outcome. There are considerable time and cost burdens associated with nonoperative management, as well as a small but finite incidence of morbidity. Mortality with this type of approach has been unusual but can be greater than 20% if bile peritonitis supervenes.

References

1. Dickerman RM, Dunn EL: Splenic, pancreatic, and hepatic injuries. *Surg Clin North Amer* 1981; 61:3-16.

2. Witte CL, Zukoski CF: A rational approach to serious blunt hepatic injury. *Am Surg* 1983; 49:446-453.

3. Moore EE: Critical decisions in the management of hepatic trauma. *Am J Surg* 1984; 148:712-716.

4. Brotman S, Oliver G, Oster-Granite ML, et al: The treatment of 179 blunt trauma-induced liver injuries in a statewide trauma center. *Am Surg* 1984; 50:603-608.

5. Carmona RH, Lim RC Jr, Clark GC: Morbidity and mortality in hepatic trauma. A 5-year study. *Am J Surg* 1982; 144:88-94.

6. Dunham CM, Militello P: Surgical management of liver trauma. *Am Surg* 1982; 48:435-439.

7. Hasselgren PO, Almersjo O, Gustavsson B, et al: Trauma to the liver during a ten-year period. With special references to morbidity and mortality after blunt trauma and stab wounds. *Acta Chir Scand* 1981; 147:387-393.

8. Levin A, Grover P, Nance FC: Surgical restraint in the management of hepatic injury: A review of Charity Hospital experience. *J Trauma* 1978; 18:399-404.

9. Madding GF: Wounds of the liver. *Surg Clin N Amer* 1958; 38:1619-1629.

10. Samlouri F, Gruenberg JC, Brown RS: Intrahepatic cutaneous biliary fistulas secondary to trauma. *Henry Ford Hospital Med J* 1980; 28:63-66.

11. Norgore M: Traumatic rupture of the gallbladder. *Ann Surg* 1946; 123:127-134.

12. Carmichael DH: Avulsion of the common bile duct by blunt trauma. *South Med J* 1980; 73:166-168.

13. Soderstrom CA, Maekawa K, DuPriest RW Jr, et al: Gallbladder injuries resulting from blunt abdominal trauma. An experience and review. *Ann Surg* 1981; 193:60-66.

14. Sparkman RS, Fogelman MJ: Wounds of the liver. Review of 100 cases. *Ann Surg* 1954; 139:690-719.

15. Madding GF: Injuries of liver. *Arch Surg* 1955; 70:748-756.

16. Shires GT: Trauma, in Schwartz SI, Shires GT, Spencer FC, et al (eds): *Principles of Surgery*, ed 4. New York, McGraw-Hill, 1984, pp 242-247.

17. Grisoni ER, Gauderer MW, Ferron J, et al: Nonoperative management of liver injuries following blunt abdominal trauma in children. *J Pediatr Surg* 1984; 19:515-518.

18. Karp MP, Cooney DR, Pros GA, et al: The nonoperative management of pediatric hepatic trauma. *J Pediatr Surg* 1983; 18:512-518.

19. Meyer AA, Crass RA, Lim RC Jr, et al: Selective nonoperative management of blunt liver injury using computed tomography. *Arch Surg* 1985; 120:550-554.

20. Moon KL Jr, Federle MP: Computed tomography in hepatic trauma. *AJR* 1983; 141:309-314.

21. Cywes S, Rode H, Millar AJ: Blunt liver trauma in children. Nonoperative management. *J Pediatr Surg* 1985; 20:14-18.

22. Giacomantonio M, Filler RM, Rich RH: Blunt hepatic trauma in children. Experience with operative and nonoperative management. *J Pediatr Surg* 1984; 19:519-522.

23. Cohn I Jr, Cotlar AM, Atik M, et al: Bile peritonitis. *Ann Surg* 1960; 152:827-835.

24. Mentzer SH: Bile peritonitis. *Arch Surg* 1934; 29:227-241.

25. Ackerman NB, Sillin LF, Suresh K: Consequences of intraperitoneal bile: Bile ascites versus bile peritonitis. *Am J Surg* 1985; 149:244-246.

26. Weissmann HS, Byan KJC, Freeman LM: Role of Tc-99m IDA scintigraphy in the evaluation of hepatobiliary trauma. *Seminars Nuc Med* 1983; 13:199-222.

27. Esensten M, Ralls PW, Colletti P, et al: Posttraumatic intrahepatic biloma: Sonographic diagnosis. *AJR* 1983; 140:303-305.

28. Stegman CJ, Levy JM, Serlin S: Case of the month No. 60—laceration of the liver. Bile leakage into the laceration on hepatobiliary scan. *Ariz Med* 1982; 39:640-653.

29. Weissmann HS, Chun KJ, Frank M, et al: Demonstration of traumatic bile leakage with cholescintigraphy and ultrasonography. *AJR* 1979; 133:843-847.

30. Geis WP, Schulz KA, Giacchino JL, et al: The fate of unruptured intrahepatic hematomas. *Surgery* 1981; 90:689-697.

31. Lambeth W, Rubin BE: Nonoperative management of intrahepatic hemorrhage and hematoma following blunt trauma. *Surg Gynecol Obstet* 1979; 148:507-511.

See also pp 534–536 and 536–537.

Modified radical neck dissections necessitated by a retropharyngeal abscess

JAY H. KLARSFELD, MD; STEVEN H. SACKS, MD; ROBERT P. GREEN, MD

Since the advent of antibiotics, the incidence of retropharyngeal abscess has decreased significantly. Only recently has the topic received attention in the literature, after many years of relative obscurity. The classic presentation in the young child is to be considered in juxtaposition to that of patients of variable ages who are now presenting due to an underlying immunocompromised status. Occasionally, a patient presents in such an advanced condition that life-threatening complications ensue and require extraordinary therapeutic measures, as in the following example.

Case Report

A 14-month-old girl was transferred to

From the Department of Otolaryngology, Mount Sinai School of Medicine, New York, NY.

Address correspondence to Dr Green, Associate Clinical Professor, Mount Sinai School of Medicine, Department of Otolaryngology, 1 Gustave L. Levy Place, New York, NY 10029.

Mount Sinai Hospital with a diagnosis of retropharyngeal abscess. Two weeks prior to admission, nausea and vomiting developed, and she had a temperature of 103°F. Her pediatrician made the diagnosis of gastroenteritis and sent the child home on a diet of clear fluids. At that time, a mild degree of cervical adenopathy was noted. Three days later, she was seen with a persistent fever of 104°F; white blood cell count, 21,000/μL; erythrocyte sedimentation rate, 102 mm/h; and there were some bacteria in her urine. Her cervical adenopathy had persisted, but the remainder of her head and neck examination was normal. The child was sent home on oral trimethoprim and sulfamethoxazole for treatment of an apparent urinary tract infection.

During the next few days, the child remained febrile, the adenopathy progressed, and she became unable to tolerate anything by mouth. She was admitted to a local hospital for intravenous hydration. Following an otolaryngologic consultation and a lateral radiograph of the neck (Fig 1), the diagnosis of retropharyngeal abscess was made, and the patient was transferred to our institution.

At the time of transfer, the child was slightly lethargic, febrile (102°F), and breathing without compromise. The pharynx was erythematous and convex in appearance. Multiple, bilateral cervical nodes were palpable. The lateral neck films confirmed a bulging, grossly enlarged, retropharyngeal space. The medical history was unremarkable for recurrent infection or exposure to tuberculosis, and the patient had undergone routine immunization. She was taken to the operating room and, under general endotracheal anesthesia and via a peroral approach, a large right-sided retropharyngeal abscess was drained of frank pus, and a smaller left-sided retropharyngeal mass was opened, yielding a lesser amount of pus and containing obviously inflamed and hypertrophic lymphoid tissue. Following surgery, the endotracheal tube was removed, and the antibiotic coverage was switched from a combination of ampicillin and oxacillin to oxacillin alone, as the Gram stain and culture yielded only *Staphylococcus aureus.*

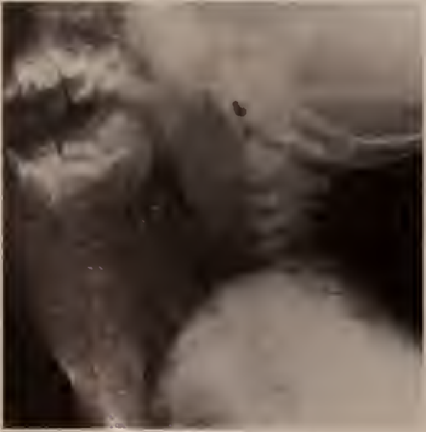


FIGURE 1. Lateral neck film demonstrating obvious retropharyngeal bulge.

During the following week, there was distinct clinical improvement, and oral intake was resumed. Daily temperature elevations in excess of 102°F, however, persisted. In addition, the patient's cervical adenopathy remained unchanged. She was returned to the operating room, where the retropharyngeal space was reopened transorally and large, necrotic nodes with scant pus were found.

On histologic analysis, the tissue was unremarkable. On the recommendation of the infectious disease consultant the antibiotic regimen was changed to cefotaxime and clindamycin. In addition, viral titers and immune response tests were performed, including immunoglobulin assays, lymphocyte counts, skin tests, phagocyte studies, and complement levels, all of which were unremarkable.

After a week of this new antibiotic regimen, it was apparent that the cervical adenopathy had not resolved and the temperature spikes persisted. A computed tomographic (CT) scan, performed with sedation and constant observation, revealed bilateral, abscessed, necrotic lymph nodes throughout both sides of the neck (Fig 2). Bilateral, modified radical neck dissections with preservation of all structures but vigorous removal of the major lymphatic chains was performed, yielding numerous frankly abscessed and necrotic nodes. The procedure consisted of raising subplatysmal flaps; dissecting and preserving the carotid sheath contents, sternocleidomastoid muscles, accessory nerves, phrenic nerves, brachial plexuses, and submaxillary glands; and removing the matted and abscessed nodal chains and infected fatty tissues, as is performed in the most conservative form of modified radical neck dissection. Suction drains were placed bilaterally and gradually advanced over six days. No growth was obtained on aerobic and anaerobic cultures. Gram stain revealed only inflammatory cells.

The child rapidly defervesced, maintained a stable airway, and was discharged after ten days. At one year's follow-up, she has no deficits and is without evidence of the above events, other than a well-camouflaged, long, horizontal cervical scar.

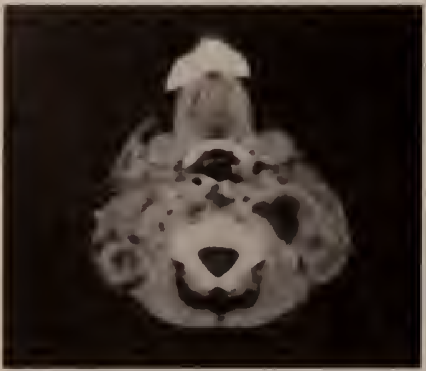


FIGURE 2. CT scan: axial section through midneck revealing multiple abscessed lymph nodes.

Discussion

To our knowledge, this is the only reported case of a retropharyngeal abscess that necessitated modified radical neck dissections. This is even more remarkable considering that reports of retropharyngeal abscesses date back to Galen in the second century AD.[1] Most retropharyngeal abscesses are initiated in children by an upper respiratory tract infection of the oral and nasopharyngeal lymphoid tissue. In adults, dental caries, tuberculosis, and trauma also play a role.[2,3]

The space between the visceral fascia (which is just posterior to the superior constrictor muscle and pharyngeal mucosa) and the alar fascia (which is just anterior to the prevertebral fascia) is anatomically the retropharyngeal space. The prevertebral space is a potential space posterior to the prevertebral fascia and anterior to the vertebral bodies. The "danger space" lies between these two spaces and is demarcated anteriorly by the alar fascia and posteriorly by the prevertebral fascia.[4] Extending from the base of the skull into the mediastinum, this space is the route by which both retropharyngeal and prevertebral abscesses spread to produce mediastinitis. The convergence of all fascial layers into the carotid sheath also provides a potential pathway for extension.[5,6]

The retropharyngeal lymph nodes are located in two groups on each side of the midline and undergo atrophy as maturation ensues.[7] The commonly noted sequestration of retropharyngeal abscesses to one side of the midline is probably secondary to an attachment of the superior constrictor muscle to the prevertebral fascia and is enhanced by the lateral placement of the relevant lymph nodes.[8]

Most of the lymphatic drainage from the nose, throat, and ears is carried directly to the deep cervical nodes, and only a small percentage is directed toward the retropharyngeal spaces, even in children.[9] Efferent lymphatics from the retropharyngeal spaces drain into the internal jugular nodes of the deep cervical chain, with cross anastamosis into other nodes of the cervical lymph system, hence, the frequently occurring cervical adenopathy.

In the preantibiotic era, retropharyngeal abscess was clearly a disease of early childhood.[10] Since the advent of antibiotics and the increased prevalence of immunocompromise in patients, the ratio of adult patients to children has dramatically increased.[11]

In adults who complain of fever, dysphagia, and possibly some difficulty breathing, the diagnosis may easily be suspected and possibly confirmed on pharyngeal or indirect examination. In young children, the symptom complex will be less clear, with fever, malaise, and poor appetite being quite nonspecific. With progression, cervical adenopathy; refusal to eat, with or without drooling; and airway compromise make the localization of the problem more apparent. Direct examination will be difficult, and a bulge viewed face-on can be missed. Neck stiffness due to prevertebral muscle spasm can cause obvious confusion with meningitis in a febrile child.[12] Rupture can result in immediate asphyxiation or serious life-threatening aspiration pneumonia. Spread to the great vessels in the neck or to the mediastinum can result in vessel rupture, septic thrombosis, or mediastinitis.[13-15] As in this case, spread of infection to other nodal chains can result in suppuration and disseminated abscess formation.

The lateral neck film characteristically demonstrates marked soft tissue enlargement in the retropharyngeal region, often associated with straightening of the cervical spine. The soft tissue shadow should not exceed one and a half times the width of the fourth cervical vertebral body before the age of one, and this lessens to less than one half the width of the fourth vertebral body after the age of one.[16] A true lateral position must be achieved, and it must be kept in mind that expiration in an infant can cause a thickened appearance imitating the presence of a retropharyngeal process.[17] CT scanning enhances one's diagnostic capability with regard to the primary abscess as well as associated complications, but with an infant who may require sedation and in the setting of a compromised airway, CT scanning should be used judiciously.[18] In this case, the CT scan was invaluable in fully delineating the extent of suppuration throughout the neck and in indicating the need for modified neck dissection.

After endotrachael intubation, in the infant or child, transoral drainage via a direct vertical incision is easily accomplished and is usually adequate. With inferior progression, or in the adult, where the extent of the process is often less well defined, an external approach is preferable. Cultures for aerobic and anaerobic organisms are mandatory. Diverse organisms, often of a resistant nature, require broad spectrum antibiotic coverage at least until culture results are available. At a minimum, coverage of the usual aerobic and anaerobic upper respiratory pathogens is essential.[11]

The case presented here is obviously quite unusual with regard to the massive cervical suppuration that required such extensive surgery. This report should not be interpreted as advocating modified radical neck dissections unless conventional therapy has clearly failed, as there is obvious risk to cranial nerves and major vascular structures, heightened by the disturbed anatomy and inflammatory response. Simple drainage of just the grossly abscessed nodes was thought to be inadequate, as we had observed mildly infected nodes progress to abscesses in spite of drainage of the primary site of infection and ongoing intravenous antibiotic therapy. Suppuration of cervical nodes requiring external drainage has been reported in conjunction with a retropharyngeal abscess, but not of this magnitude.[12] The underlying reasons for this progression are not clear. The child was, based on all of our sophisticated testing, immunologically normal, and the organisms involved are aggressive but common pathogens. In spite of adequate drainage of the primary retropharyngeal abscess and vigorous antibiotic therapy, expanding cervical sepsis proved difficult to contain. The amount of time from the true onset of the illness to the initiation of aggressive and directed management is obscure but clearly is an important consideration. How this time span compares to that which occurs in other cases of retropharyngeal abscess is difficult to determine.

Finally, it should be remembered that all swelling in the retropharyngeal space may not be caused by infection. Neoplasms such as cystic hygroma, hemangioma, lymphoma, and a variety of other tumors can occupy this space.[19] Direct trauma, in addition to possibly initiating an infection, can also cause an isolated hematoma. Such conditions should be readily distinguishable from a frank retropharyngeal abscess.

References

1. Davidson M: Abscesses of retropharyngeal spaces in adults. *Laryngoscope* 1949; 59:1146-1170.
2. Paparella MM, Shumrick DA: *Otolaryngology*, ed 2. Philadelphia, WB Saunders & Co, 1980, vol 3.
3. Husaru AD, Nedzelski JM: Retropharyngeal abscess and upper airway obstruction. *J Otolaryngol* 1979; 8:443-447.
4. Grodinsky M: Retropharyngeal and lateral pharyngeal abscesses; anatomic and clinical study. *Ann Surg* 1939; 110:177-199.
5. Frank I: Retropharyngeal abscess. *JAMA* 1921; 77:517-522.
6. Wong Y, Nootry G: Retropharyngeal space—a review of anatomy, pathology and clinical presentation. *J Otolaryngol* 1978; 7:528-536.
7. Seid AB, Dunbar JS, Cotton RT: Retropharyngeal abscesses in children revisited. *Laryngoscope* 1979; 89:1717-1724.
8. Johnson JT, Tucker HM: Recognizing and treating deep neck infection. *Postgrad Med* 1976; 59:95-100.
9. *Gray's Anatomy*, ed 35 (British). Philadelphia, WB Saunders & Co, 1973.
10. Brown J: Acute retropharyngeal abscess in children. *Laryngoscope* 1919; 29:9-12.
11. Barratt GE, Koopermann CF Jr, Coulthard SW: Retropharyngeal abscess—a ten-year experience. *Laryngoscope* 1984; 94:455-463.
12. Richards L: Retropharyngeal abscess. *N Engl J Med* 1936; 215:1120-1130.
13. Ramllo J, Harris U, White H: Empyema as a complication of retropharyngeal and neck abscess in children. *Ped Radiol* 1978; 126:843-846.
14. Mosher HP: Deep cervical abscess and thrombosis of internal jugular vein. *Laryngoscope* 1920; 30:365-375.
15. Wishart D: Retro-pharyngeal abscess; and erosion of internal carotid artery with pathological specimen. *Can Med Assoc J* 1923; 13:635-641.
16. Brenner GH: Variations in the depth of the cervical prevertebral tissue in normal infants studied by cinefluorography. *Am J Roentgenol* 1964; 91:573-577.
17. Gwinn JL, Lee FA, Canby JP: Retropharyngeal abscess. *Am J Dis Child* 1971; 121:237-238.
18. Endicott JN, Nelson RJ, Saraceno CA: Diagnosis and management decisions in infections of the deep fascial spaces of the head and neck utilizing computerized tomography. *Laryngoscope* 1982; 92:630-633.
19. McCook TA, Felman AH: Retropharyngeal masses in infants and young children. *Am J Dis Child* 1979; 133:41-43.

Fulminant lymphocyte-depleted Hodgkin disease in a homosexual man with HIV infection

DONALD COLBOURN, MD; HARRY STASZEWSKI, MD; STEVEN GOLDENBERG, MD;
VIRGINIA DONOVAN, MD; FRANK RAIO, MD

Recent reports have suggested a possible association of Hodgkin disease in patients at risk for acquired immunodeficiency syndrome (AIDS).[1-9] Usually, the pathologic subtypes of Hodgkin disease reported have been the mixed cellularity or nodular sclerosis types. In addition, most of the patients presented did not have human immunodeficiency virus (HIV) infections that had been confirmed with serologic or viral studies. We report on a patient with serologic evidence of HIV infection who had fulminant lymphocyte-depleted Hodgkin disease, which proved fatal.

CASE REPORT

A 43-year-old homosexual man was admitted to the Winthrop-University Hospital emergency room with a four-day history of fever, jaundice, and increasing mental confusion. He had been well until several months prior to admission, when he complained of mild anorexia and weight loss. No lymphadenopathy or organomegaly was detected at that time. T cell subset analysis at that time revealed an OKT4:OKT8 ratio of 1.06 (normal range, 1.2–3.8).

On admission, physical examination findings included a temperature of 103.2°F and a pulse rate of 100/min. The patient appeared acutely ill and was deeply jaundiced. Disorientation was present, but the patient remained alert. There was posterior cervical, bilateral supraclavicular, and axillary adenopathy, but no individual node was greater than 1 cm in diameter. Ascites and massive hepatosplenomegaly were demonstrable. Asterixis was present, but no focal neurologic abnormalities were detected.

Laboratory studies were remarkable for a severe pancytopenia. The blood urea nitrogen level was 92 mg/dL, and the creatinine level was 4.1 mg/dL. Total bilirubin was elevated at 11.8 g/dL, and hepatic enzymes were elevated to three times normal values.

A computerized axial tomographic (CAT) study of the abdomen revealed the presence of ascites and massive hepatosplenomegaly. There was no evidence of biliary tract obstruction or focal defects. The kidneys appeared normal, and there was a distinct absence of retroperitoneal adenopathy. A bone marrow aspiration and biopsy revealed a hypercellular marrow with megakaryocytic and histiocytic hyperplasia with occasional erythrophagocytosis. Stains and cultures for mycobacteria and fungi were negative. All stains and cultures of blood, urine, stool, and sputum for significant bacterial, fungal, mycobacterial, or protozoan organisms were negative. Antibody titers for Epstein-Barr virus, cytomegalovirus, and hepatitis A and B were not supportive of active infection. ELISA assay for HIV infection was positive in high titer and was confirmed by Western blot study.

The patient's condition deteriorated rapidly, with the development of renal and hepatic failure as well as severe metabolic acidosis. The bilirubin level continued to increase beyond 50 mg/dL. A diagnosis of a lymphoproliferative disorder, possibly malignant histiocytosis, was made. Heroic therapy with cyclophosphamide, vincristine, and prednisolone was administered intravenously. Eighteen hours later, a cervical lymph node biopsy was obtained (Fig 1). Progressive organ failure continued despite hemodialysis and blood product support, and the patient died nine days after admission. Postmortem examination demonstrated lymphocyte-depleted Hodgkin disease involving the liver, spleen, and bone marrow as well as the perisplenic and peripancreatic lymph nodes. Analysis of lymphocyte surface markers in lymph node sections by the immunoperoxidase technique demonstrated a lack of significant positivity, consistent with the diagnosis of Hodgkin disease.

DISCUSSION

It is apparent from recent reports that Hodgkin disease is yet another neoplastic disorder that occurs in patients at risk for AIDS.[1-9] Unusual presentations of Hodgkin disease with extranodal involvement and advanced stage at diagnosis have been characteristic of these reports. Most reports have been of the mixed cellularity subtype, though several patients with features of both mixed cellularity and lymphocyte-depleted Hodgkin disease have been presented.[8] While the occurrence of opportunistic infections characteristic of AIDS has suggested HIV infection in these patients, few cases with actual confirmation by serologic or viral studies have actually been published. It remains possible that some of these cases are the coincidental occurrence of Hodgkin disease in homosexual men.

Though the course of Hodgkin dis-

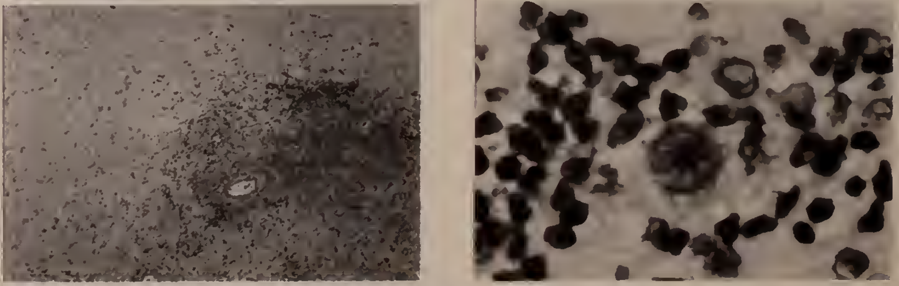


FIGURE 1. **Left,** Antemortem cervical lymph node biopsy showing marked lymphoid depletion and fibrosis (hematoxylin-eosin stain; original magnification × 40); **Right,** At higher magnification, a mononuclear Hodgkin cell admixed with benign lymphocytes (hematoxylin-eosin stain; original magnification × 1,000).

From the Oncology-Hematology Division, Department of Medicine (Drs Colbourn and Staszewski) and the Department of Pathology (Dr Donovan), Winthrop-University Hospital, Mineola, NY, and the Department of Medicine, Health Sciences Center, SUNY at Stony Brook, Stony Brook, NY (Drs Colbourn, Staszewski, Goldenberg, and Raio).

Address correspondence to Dr Staszewski, Director, Hematology Service, Oncology-Hematology Division, Winthrop-University Hospital, Winthrop-University Hospital Professional Building, 222 Station Plaza North, Mineola, NY 11501.

ease has been reported to be aggressive in most patients who are also at risk for AIDS, the disease usually responds to therapy. In most cases, the greatest morbidity and mortality have resulted from opportunistic infections or Kaposi's sarcoma, rather than from progressive Hodgkin disease. The role of the additional immunosuppression that inevitably occurs in the treatment of advanced Hodgkin disease is unclear in terms of the risk of activating opportunistic pathogens.

At least one group has cautioned against the use of steroids or combined modality therapy in male homosexuals with Hodgkin disease because of the sensitivity of the disease to treatment and the subsequent occurrence of overwhelming opportunistic infections in these patients.[2] Unfortunately, the fulminant nature of Hodgkin disease in patients such as the one described in this report may make such a course of action impossible to follow.

The present case has several unique features. No opportunistic infections either prior to the appearance of Hodgkin disease or during the hospital course appear to have contributed to the patient's demise. Fatal, fulminant lymphocyte-depleted Hodgkin disease presenting with pancytopenia and massive splenomegaly is an unusual occurrence in the more recent reports of this histologic subtype.[10] Unlike previous patients at risk for AIDS who had aggressive Hodgkin disease, this patient probably did not survive his malignancy long enough to develop opportunistic infectious complications.

Acknowledgment. The authors thank Dr Elaine S. Jaffe for reviewing the biopsy and autopsy reports.

REFERENCES

1. Unger PD, Strauchen JA: Hodgkin's disease in AIDS complex patients. Report of four cases and tissue immunologic marker studies. *Cancer* 1986; 58:821-825.
2. Baer DM, Anderson ET, Wilkinson LS: Acquired immune deficiency syndrome in homosexual men with Hodgkin's disease. Three case reports. *Am J Med* 1986; 80:738-740.
3. Schoeppel SL, Hoppe RT, Dorfman RF, et al: Hodgkin's disease in homosexual men with generalized lymphadenopathy. *Ann Intern Med* 1985; 102:68-70.
4. Scheib RG, Siegel RS: Atypical Hodgkin's disease and the acquired immunodeficiency syndrome [letter]. *Ann Intern Med* 1985; 102:554.
5. Ioachim HL, Cooper MC, Hellman GC: Hodgkin's disease and the acquired immunodeficiency syndrome [letter]. *Ann Intern Med* 1984; 101:876-877.
6. Robert NJ, Schneiderman H: Hodgkin's disease and the acquired immunodeficiency syndrome [letter]. *Ann Intern Med* 1984; 101:142-143.
7. Subar M, Chamulak G, Dalla-Favera R, et al: Clinical and pathological characteristics of AIDS associated Hodgkin's disease. *Blood* 1986; 68(suppl): 135a.
8. Prior E, Goldberg AF, Conjalka MS, et al: Hodgkin's disease in homosexual men. An AIDS-related phenomenon? *Am J Med* 1986; 81:1085-1088.
9. Mitsuyasu RT, Colman MF, Sun NC: Simultaneous occurrence of Hodgkin's disease and Kaposi's sarcoma in a patient with the acquired immune deficiency syndrome. *Am J Med* 1986; 80:954-958.
10. Kant JA, Hubbard SM, Longo DL, et al: The pathologic and clinical heterogeneity of lymphocyte-depleted Hodgkin's disease. *J Clin Oncol* 1986; 4:284-294.

Hypersensitivity reaction to anticonvulsants following head injury

JAMES M. HASSETT, JR, MD; THOMAS DOOLITTLE, MD; MICHAEL MOLLOY, BS; DAVID LALKA, PHD

Trauma is a complex state of bodily injury associated with physiologic changes that have the potential to alter hepatic drug clearance mechanisms. Responses to trauma include alteration in hepatic blood flow, reduction in hepatic microsomal activity, reduction in hepatic excretion processes, and changes in protein binding.[1,2] Moderate to severe brain injury commonly occurs in a variety of trauma mechanisms. Anticonvulsant prophylaxis after concussive head injury is indicated in patients who experience a single epileptic event that is not temporally associated with impact, or in patients with a significant risk of seizure. A patient's response to medication can be affected by the pre-existing, but clinically quiescent, hypersensitivity states. In this complex post-injury state, a patient's reaction to the most commonly used agents, phenytoin and phenobarbital, can have serious consequences. We describe a patient who experienced a hypersensitivity reaction to both agents.

CASE REPORT

A 17-year-old man was involved in an accident while riding a bicycle. The patient was transported to a regional hospital, where he was unresponsive to all but very painful stimuli. After the initial assessment, he was transferred to the Trauma Service of the Erie County Medical Center (ECMC). The patient experienced three episodes of emesis during transport. The initial examination revealed a Glascow Coma Scale of 12 with normal vital signs. Physical examination was essentially normal, except for a 4-cm laceration over the left temple, an abrasion of the left knee, a semiconscious state, and slowly reactive pupillary responses. Radiologic evaluations of the skull, cervical spine, chest, and abdomen were unremarkable. A computed tomographic (CT) scan of the brain demonstrated a right frontal contusion. The patient received both dexamethasone and phenytoin. The CT scan was repeated on the fourth day of hospitalization and again demonstrated the right frontal contusion. After an uneventful hospital stay, the patient was discharged on the sixth hospital day on continued therapy with phenytoin, 300 mg orally at bedtime.

On the eighth post-injury day, the patient returned with a 24-hour history of a maculopapular pruritic rash that involved his entire body. Physical examination revealed a temperature of 103°F (oral); blood pressure, 120/60 mm Hg; heart rate, 80/min; respiratory rate, 16/min, with diffuse, nonpitting edema of the extremities. He was readmitted, the phenytoin was discontinued, and he was given diphenylhydramine, 25 mg intramuscularly. The serum phenytoin level was 16.5 mg/dL. He was started on phenobarbital, 60 mg orally twice a day. After significant symptomatic improvement, he was discharged on the fourth hospital day (post-injury day 12).

On the 19th post-injury day, the patient returned to the hospital with a generalized maculopapular eruption that involved his

---

From the Departments of Surgery (Dr Hassett), Neurosurgery (Dr Doolittle), Pharmacy (Mr Molloy), and Pharmaceutics (Dr Lalka), State University of New York at Buffalo School of Medicine.

Address correspondence to Dr Hassett, Department of Surgery, Erie County Medical Center, 462 Grider St, Buffalo, NY 14215.

entire body. The new eruption was more severe than the previous one and occurred acutely on the day prior to admission. Physical evaluation revealed a temperature of 102.4°F; blood pressure, 100/60 mm Hg; heart rate, 82/min; respiratory rate 16/min, with a diffuse morbilliform rash. Dermatologic consultation confirmed a "drug eruption rash." The patient was started on hydroxyzine, 25 mg orally four times a day, and 0.1% triamcinolone acetonide, three times a day. The phenobarbital was discontinued, and the patient was discharged at the request of his family on the second hospital day. The cutaneous eruptions eventually cleared with symptomatic treatment. No further anticonvulsant therapy was administered, and the patient remained symptom free.

DISCUSSION

In a patient who has had a concussive head injury, the occurrence of a seizure further depresses the level of consciousness, increases the intracranial pressure, and may cause unnecessary death. The precise indications for and the dosage or duration of antiseizure chemotherapy are poorly defined.[3] The question of whether anticonvulsant medication should be given for prophylaxis against early, post-traumatic seizures is unanswered.[4] Prophylactic administration of phenytoin reduces the rate of late, post-traumatic seizures[4,5] and may decrease the frequency of early seizures.[4–6] Phenytoin and phenobarbital are the most frequently utilized agents.[7] Both are almost completely absorbed after oral administration in individuals with normal gastrointestinal physiologic responses.

From 85% to 90% of phenytoin is bound to serum albumin.[7] It is metabolized in the liver to p-(hydroxyphenyl-phenyl)-hydantoin (HPPH) via a pathway well described by Michaelis-Menten kinetics.[8] This metabolite has virtually no pharmacologic activity. It is rapidly conjugated with glucuronic acid and excreted in the urine[7,9,10] or through the gastrointestinal tract. Serum levels will peak four to eight hours after ingestion.

Phenytoin is oxidized by cytochrome P-450 mono-oxygenases to several oxidized products.[11,12] Arene oxides are reactive, electrophilic compounds, which are intermediates to such oxidative reactions. If they are not detoxified, they can covalently bind to cell macromolecules, resulting in cell death or mutation. They can also act as haptens, leading to secondary immune phenomena.

Ten percent of the patients who receive phenytoin will develop a rash that requires a change to another agent.[13] The eruption is usually accompanied by fever and occurs on the ninth or tenth day of treatment. The generalized skin reaction seen after phenytoin administration may be due to a hypersensitivity reaction[14] or a high body burden of phenytoin.[15]

Among patients in whom the rash develops, there is a lower capacity for metabolizing the phenytoin to p-hydroxyphenytoin without a change in the plasma protein binding. The appearance of the rash might be due to a slow rate of para-hydroxylation of the phenytoin, which forces the metabolic process toward an epoxide pathway.

Severe idiosyncratic reaction to phenytoin was first described as "phenytoin hypersensitivity" in 1936.[14] It can include lymphadenopathy, exfoliative dermatitis, eosinophilia, and hepatic necrosis.[13,16] The reaction appears to be a hypersensitivity that may also include hemolytic anemia, leukopenia, thrombocytopenia, diffuse vasculitis, splenomegaly, and severe hepatorenal failure.[17,18]

The frequency of phenytoin hypersensitivity is unknown.[14] It generally involves adolescents or young adults.[14,19] It has an antigenic component, with one of the metabolites acting as a hapten.[20,21] It would appear that the cytotoxicity of the phenytoin hypersensitivity reaction may be due to an inherent difficulty with epoxide hydrolase.[11,12] The genetic pattern of a possibly defective epoxide hydrolase pathway detoxification mechanism suggests an autosomal, inherited trait.[11]

The metabolism of phenobarbital is predominantly by hepatic microsomal enzymes.[7] The primary metabolite is p-hydroxyphenylbarbital, which has no known anticonvulsant properties and is excreted in the urine. An adverse reaction to phenobarbital occurs with a 4% maximum reported incidence of cutaneous reaction.[22] Phenobarbital administration is occasionally associated with a syndrome of hyperpyrexia, delirium, exfoliative dermatitis, and parenchymatous organ damage, which is frequently fatal.[23] The anecdotal clinical reports are remarkably similar to the phenytoin hypersensitivity reaction.[23,24] In vitro lymphocyte stimulation studies with anticonvulsant drugs suggest a cell-mediated hypersensitivity reaction to phenobarbital similar to that seen with phenytoin.[22]

In the majority of hypersensitivity reactions in which both phenytoin and phenobarbital have been implicated together, the interrelationships have generally been confused by the simultaneous administration of the two agents.[19,25–28] The structural similarities between phenytoin and phenobarbital have been considered to be the cause of the cross reaction.[22] In the patient presented here, there was a clear separation of the administration of the two drugs, with symptomatic improvement after discontinuation of the phenytoin and return of symptoms after administration of the phenobarbital.

A hypersensitivity reaction is independent of the traumatic event. However, traumatic injury has the potential to significantly alter drug pharmacokinetics through a variety of mechanisms. Studies in a rat trauma model indicate that hepatic microsomal activity (in vitro) is depressed during the post-traumatic period.[2] The normal hepatic response to injury results in an increase in the normal binding protein alpha-1-acid glycoprotein.[1,29] These alterations may increase the potential for idiosyncratic or hypersensitivity reactions. Environmental alteration may not be the only mechanism of altering pharmacogenetic conditions.[30] The trauma-induced alterations in hepatic metabolism, without a clinical change in liver function, can result in the creation of hypersensitivity-inducing ligands. This case clearly establishes the ability of both phenytoin and phenobarbital to induce similar hypersensitivity responses when administered separately to the same patient.

REFERENCES

1. Edwards DJ, Lalka D, Cerra F, et al: Alpha1-acid glycoprotein concentration and protein binding in trauma. *Clin Pharmacol Ther* 1982; 31:62–67.
2. Slaughter R, Hassett JM: Hepatic drug clearance following traumatic injury. *Drug Intell Clin Pharm* 1985; 19:799–806.
3. Rapport RL 2d, Penry JK: A survey of attitudes toward the pharmacologic prophylaxis of posttraumatic epilepsy. *J Neurosurg* 1973; 38:159–166.
4. Wyler AR, Ray MW: Anticonvulsant prophylaxis against posttraumatic seizures. *Contemp Neurosurg* 1985; 7:1–6.
5. Young B, Rapp RP, Norton JA, et al: Failure of prophylactically administered phenytoin to prevent early posttraumatic seizures. *J Neurosurg* 1983; 58:231–235.
6. Young B, Rapp RP, Norton JA, et al: Failure of prophylactically administered phenytoin to prevent late posttraumatic seizures. *J Neurosurg* 1983; 58:236–241.
7. Berman PH: Management of seizure disorders with anticonvulsant drugs: Current concepts. *Pediatr Clin North Am* 1976; 23:443–459.
8. Ludden TM, Allen JP, Valutsky WA, et al: Individualization of phenytoin dosage regimens. *Clin Pharmacol Ther* 1977; 21:287–293.
9. Olanow CW, Finn AL: Phenytoin: Pharmacokinetics and clinical therapeutics. *Neurosurg* 1981; 8:112–117.
10. Plaa GL: Acute toxicity of antiepileptic drugs. *Epilepsia* 1975; 16:183–191.
11. Spielberg SP, Gordon GB, Blake DA, et al:

Predisposition to phenytoin hepatoxicity assessed in vitro. *N Engl J Med* 1981; 305:722-727.

12. Spielberg SP, Gordon GB, Blake DA, et al: Anticonvulsant toxicity in vitro: Possible role of arene oxides. *J Pharmacol Exp Ther* 1981; 217:386-389.

13. Almeyda J, Levantine A: Drug reactions. XX. Cutaneous reactions to anticonvulsants. *Br J Dermatol* 1972; 87:646-649.

14. Haruda F: Phenytoin hypersensitivity: 38 cases. *Neurology* 1979; 29:1480-1485.

15. Wilson JT, Höjer B, Tomson G, et al: High incidence of a concentration-dependent skin reaction in children treated with phenytoin. *Br Med J* 1978; 1:1583-1586.

16. Van Oss CJ, Bronson PM, Border JR: Changes in the serum alpha glycoprotein distribution in trauma patients. *J Trauma* 1975; 15:451-455.

17. Hyman LR, Ballow M, Knieser MR: Diphenylhydantoin interstitial nephritis. Roles of cellular and humoral immunologic injury. *J Pediatr* 1978; 92:915-920.

18. Josephs SH, Rothman SJ, Buckley RH: Phenytoin hypersensitivity. *J Allergy Clin Immunol* 1980; 66:166-172.

19. Parker WA, Shearer CA: Phenytoin hepatotoxicity: A case report and review. *Neurology* 1979; 29:175-178.

20. Holland P, Mauer AM: Diphenylhydantoin-induced hypersensitivity. *J Pediatr* 1963; 66:322-332.

21. Robinson DS, MacDonald MG, et al: Sodium diphenylhydantoin reaction with evidence of circulating antibodies. *JAMA* 1965; 192:171-172.

22. Knutsen AP, Anderson J, Satayaviboon S, et al: Immunologic aspects of phenobarbital hypersensitivity. *J Pediatr* 1984; 105:558-563.

23. McGeachy TE, Bloomer WE: Phenobarbital sensitivity syndrome. *Am J Med* 1953; 14:600-604.

24. Shapiro PA, Antonioli DA, Peppercorn MA: Barbiturate-induced submassive hepatic necrosis. Report of a case and review of the literature. *Am J Gastroenterol* 1980; 74:270-273.

25. Crawford SE, Jones CK: Fatal liver necrosis and diphenylhydantoin sensitivity. *Pediatrics* 1962; 30:595-600.

26. Dubois EL: Toxic hepatic necrosis associated with taking dilantin, tridione and phenobarbital. *Am J Clin Path* 1950; 80:153-158.

27. Kahn HD, Faguet GB, Agee JF, et al: Drug-induced liver injury. In vitro demonstration of hypersensitivity to both phenytoin and phenobarbital. *Arch Intern Med* 1984; 144:1677-1679.

28. Pollack MA, Burk PG, Nathanson G: Mucocutaneous eruptions due to antiepileptic drug therapy in children. *Ann Neurol* 1979; 5:262-267.

29. Ting S, Dunsky EH: Diphenylhydantoin-induced hepatitis. *Ann Allergy* 1982; 48:331-332.

30. Vesell ES: Genetic host factors: Determinants of drug response [editorial]. *N Engl J Med* 1985; 313:261-262.

FROM THE LIBRARY

THE DIAGNOSIS AND TREATMENT OF LACERATIONS OF THE LIVER

Dr. G. Dencks gives the details of seven cases which were brought to the hospital at Friedrichshain, three of which recovered. In these three cases one resulted from a gunshot wound, one from a kick by a horse, while the third patient was run over. Of the four fatal cases, in two there were other severe accompanying lacerations. In the four fatal cases, the ruptures were subcutaneous, two having resulted from falls, and two having been crushed under wheels.

The conclusions reached from the observation of these cases do not very materially differ from those to be drawn from the study of a larger group of similar injuries. Thus it was observed that the right lobe is the part of the liver most often injured in subcutaneous ruptures, and that the tears are usually on the convex surface. Excluding the case of gunshot wound the left lobe was injured but once. In no case were isolated tears found on the concave surface.

As to the signs, tension of the abdomen and abdominal tendencies were constant manifestations. Neither was absent in a single case. The tension and rigidity were always general and never limited to one side. Tenderness, though likewise general, was found to be more marked over the right hypochondrium or epigastrium. In three cases there was neither hiccough eructation of gas nor vomiting.

These cases were all extremely severe, and in each large quantities of blood were found in the abdominal cavity. Dullness over the dependent parts of the abdomen was observed four times, and twice there was pain radiating towards the right shoulder blade.

The pulse could not be depended upon to indicate the degree of internal hemorrhage. Occasionally, in the most rapidly fatal cases, it was found to be of relatively good character.

The only therapeutic measure to be considered is laparotomy. In two cases the lacerations were sutured with catgut carried deeply into the liver substance. In the other cases, and this group includes the three which recovered, the bleeding was controlled by tampon. No ill effect ever attended this method of treatment. In one case the gauze strips were serviceable in draining an abscess which had formed between liver and diaphragm. The gauze tampon is to be regarded as the best means of controlling liver hemorrhage. It is the most reliable and at the same time the quickest of application. Suture is to be reserved for such wounds as have smooth, clean cut edges, easy of apposition.

Of greatest importance in cases of laceration of the liver is prompt diagnosis followed by immediate laparotomy. Even when the diagnosis cannot be absolutely made, if there is good ground to fear that this injury has occurred, exploratory laparotomy is indicated.—*Deutsche Zeitschrift f. Chirurgie*, 82 Band 4-6 Heft Mai, 1906.

(*NY State J Med* 1906; 6:379-380)

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Responses to "Death of a dentist"

TO THE EDITOR: Dr Bluestone's "Death of a dentist"[1] is a fine and well-written essay. We have lots of good mechanics who are dentists and physicians who are not doctors. They take care of diseases, not people with diseases. Of course, there are a few in each generation who, for whatever reasons, are sensitive to the needs of people. The applicants I have interviewed have often been broadly read and educated, not schooled, and my colleagues have rated them low "because their interests are too broad"! But I have persisted in swimming upstream at our meetings, and will die fighting for the principle of taking care of people with diseases.

MAURICE TULIN, MD
39 Hicks Lane
Great Neck, NY 11024

1. Bluestone NR: Death of a dentist. *NY State J Med* 1987; 87:356-357.

TO THE EDITOR: What can I say? Dr Bluestone's "Death of a dentist"[1] brought tears to my eyes, too.

ALFRED P. INGEGNO, MD
27 Eighth Ave
Brooklyn, NY 11217

1. Bluestone NR: Death of a dentist. *NY State J Med* 1987; 87:356-357.

TO THE EDITOR: "Death of a dentist"[1] is a masterpiece. I am enriched by having read it, and I thank Dr Bluestone for the time and effort that must have gone into writing it.

CHARLES S. HIRSCH, MD
Chief Medical Examiner
Suffolk County
Suffolk County Office Building
Hauppauge, NY 11788

1. Bluestone NR: Death of a dentist. *NY State J Med* 1987; 87:356-357.

TO THE EDITOR: I was touched by Dr Bluestone's[1] beautiful, warm story of the death of a dentist in the June issue of the *New York State Journal of Medicine.* It is a loving tribute to one who served her and his other patients with his skill and himself.

CHARLES A. PERERA, MD
Kendal at Longwood #17
Kennett Square, PA 19348

1. Bluestone NR: Death of a dentist. *NY State J Med* 1987; 87:356-357.

TO THE EDITOR: Dentists like Dr Bluestone's pal Jack are only rarely found. When I left dentistry for plastic surgery, it was because I felt I might not be appreciated. Dr Bluestone's essay[1] made me realize that, had I stayed, I might have been lucky enough to find a patient like her.

BARRY M. ZIDE, DMD, MD
New York University
Medical Center
333 East 34th St
New York, NY 10016

1. Bluestone NR: Death of a dentist. *NY State J Med* 1987; 87:356-357.

Moses Maimonides and air pollution

TO THE EDITOR: I enjoyed the review article by McCunney[1] and the accompanying editorial by Hinkle[2] on indoor air pollution. The medieval giant of Judiasm, Moses Maimonides (1135–1204), in his *Treatise on Asthma,*[3] one of his ten authentic medical works,[4] describes air pollution. The last chapter of the *Treatise on Asthma* deals with concise admonitions and aphorisms which Maimonides considered "useful to any man desirous of preserving his health and administering to the sick." The chapter begins as follows: "The first thing to consider . . . is the provision of fresh air, clean water and a healthy diet." Fresh air is described in some detail:

> . . . City air is stagnant, turbid and thick, the natural result of its big buildings, narrow streets, the refuse of its inhabitants . . . one should at least choose for a residence a wide-open site . . . living quarters are best located on an upper floor . . . and ample sunshine . . . Toilets should be located as far as possible from living rooms. The air should be kept dry at all times by sweet scents, fumigation and drying agents. The concern for clean air is the foremost rule in preserving the health of one's body and soul.

Let our air pollution control experts take cognizance of Maimonides' prophetic statements made nearly 800 years ago.

FRED ROSNER, MD
Department of Medicine
Queens Hospital Center Affiliation
of the Long Island Jewish
Medical Center
Jamaica, NY 11432

1. McCunney RJ: The role of building construction and ventilation in indoor air pollution: Review of a recurring problem. *NY State J Med* 1987; 87:203-209.
2. Hinkle LE Jr: Indoor air pollution. *NY State J Med* 1987; 87:196-197.
3. Rosner F: Moses Maimonides' *Treatise on Asthma. J Asthma* 1984; 21:119-129.
4. Rosner F: The medical writings of Moses Maimonides. *Clio Medica* 1981; 16:1-11.

Rare *Streptococcus pneumoniae* soft tissue abscess

TO THE EDITOR: *Streptococcus pneumoniae* usually produces infection in the respiratory tract. Major secondary complications are principally central nervous system infections, endocarditis, and osteomyelitis. Soft tissue abscess without evidence of systemic or contiguous infection is a rare event. The patient presented here demonstrates a chronic, solitary abscess in the soft tissue space of the buttock and thigh, of undefined origin.

Case Report. A 50-year-old man was admitted to Harlem Hospital Center on March 1, 1982, with a history of inability to walk since December 1981. The onset of this disorder had been sudden. After a visit to the emergency room on December 12, 1981, he was sent home with a diagnosis of muscle spasms.

In February 1982 swelling of the left posterior flank developed. Pus was periodically drained on an outpatient basis. There was no previous history of trauma, fever, diabetes, sickle cell disease, hypertension, tuberculosis, or drug or alcohol abuse. The patient worked as a security guard and had not recently travelled.

The physical examination was normal, except for a fluctuant abscess extending from the level of the third lumbar vertebra to the sacral area in the left flank. There was edema in the right thigh, with diminished power in the iliopsoas, hamstring, and extensor hallucis longus muscles. There was a surgical scar from a prior partial colectomy and anastomosis.

Incision and drainage of the left flank yielded 1,000 cc of pus on the fifth day of admission. A Gram stain revealed only neutrophils and no organisms. Some improvement in leg movement and function of the gluteal and thigh muscles was noted with the initiation of cefoxitin, 1 g intravenously every four hours, and oxacillin, 2 g intravenously every four hours. Spinal (lumbosacral) radiographs and a bone scan were normal and showed no evidence of osteomyelitis. A computed tomographic (CT) scan showed a large, soft tissue abscess that dissected along tissue planes overlying the paraspinal muscle groups, gluteal, and upper thigh muscles, with some extension into the obturator canal. Two thousand cc of pus was drained from this abscess on the sixth hospital day. All cultures grew *Streptococcus pneumoniae* type 15. Therapy was promptly changed to penicillin, two million units every four hours, and the patient steadily recovered, with no residual neurologic deficit.

Discussion. This patient had an extensive soft tissue abscess which grew a pure culture of *S pneumoniae* type 15. Despite clear evidence of motor weakness at the time of admission, no signs of epidural or subdural infection or of vertebral osteomyelitis could be found. The most likely explanation for the neurologic findings would appear to be compression of muscle groups by the abscess mass and, perhaps, compression of nerves of the obturator canal.

Soft tissue abscess caused by *S pneumoniae* is rare. The two cases reported previously from this institution occurred in drug addicts.[1] It was presumed that these individuals had injected the pneumococcus through contaminated drug paraphernalia. This patient was clearly not a drug addict and had not received any injections. The pathogenesis of the lesion is therefore obscure. In the absence of evidence of bone involvement, it must be assumed that a temporarily remote bacteremia had evolved into a chronic, indolent, deep tissue infection.

LAKSHMANAN SATHYAVAGISWARAN, MD
P.C.T. DICKINSON, MD
JAY DOBKIN, MD
Division of Infectious Diseases
Harlem Hospital Center
of Columbia University
506 Lenox Ave
New York, NY 10037

1. Lewis RJ, Richmond AS, McGrory JP: *Diplococcus pneumoniae* cellulitis in drug addicts. *JAMA* 1975; 232:54–55.

Isolated endometriosis in a Bartholin gland

TO THE EDITOR: Endometriosis of the vulva and perineum has been reported.[1-3] However, endometriosis of the Bartholin gland with recurrent cystic swelling of the gland has not been reported. We describe a patient with endometriosis in the area of the Bartholin gland, in whom exploratory laparotomy for a pelvic mass revealed leiomyomata uterii and no evidence of pelvic endometriosis.

Case Report. A 29-year-old woman, para 0-0-4-0, underwent excision of a cyst in the left Bartholin gland on April 8, 1986; prior symptoms included current swelling of the gland. Her history is pertinent for incision and drainage of the left Bartholin gland in 1978 and 1982, to reduce swelling. The swelling appeared to be more prominent during menses. She had had episodes of menorrhagia for several months prior to admission, associated with leiomyomata uterii.

A chest film and intravenous pyelogram performed before admission to the hospital were within normal limits. A physical examination was unremarkable. Pelvic examination revealed a 4 × 3-cm, rubbery, firm mass in the left Bartholin gland. The uterus was irregularly enlarged to the size of 14 weeks gestation by leiomyomata uterii.

Pathologic examination of the mass yielded a diagnosis of endometriosis. The formalin-fixed specimen consisted of four irregular pieces of rubbery tissue, the largest of which measured 4 × 2.5 × 1 cm. On cut sections, the larger pieces revealed gray to brown smooth surfaces. The microscopic examination revealed dense, collagenous fibrous tissue (Fig 1), with scattered nodular areas of endometriosis composed of variable admixtures of endometrial glands, stroma, inflammatory cells, extravasated red cells, and focal collections of hemosiderin-laden macrophages. Occasional glands were dilated and showed a flattened atrophic epithelium (Fig 2). In other glands there was evidence of active epithelial proliferation. Some nodules contained no recognizable epithelial components.

On May 26, 1986, an exploratory laparotomy and myomectomy were performed. There was no evidence of abdominal or pelvic endometriosis. All peritoneal reflections were normal. Both ovaries appeared normal. There were no nodules in the posterior cul-de-sac or uterosacral ligaments.

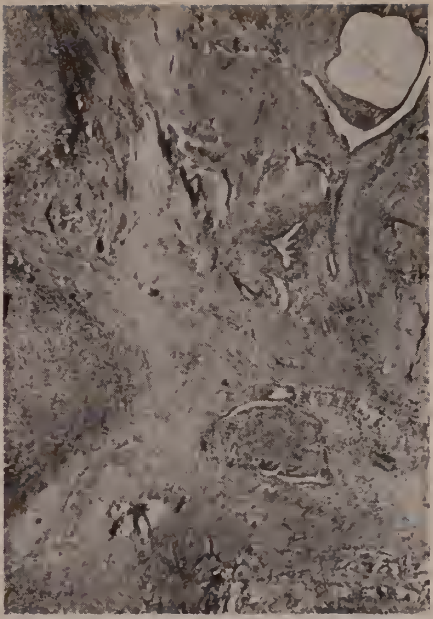


FIGURE 1. Low-power view of foci of endometriosis. Cystically dilated gland (bottom right) associated with extensive fibrous scarring and areas of hemorrhage and inflammatory cells (hematoxylin-eosin stain; original magnification × 30).

Discussion. Endometriosis of the Bartholin gland is a rare disease that may be responsible for recurrent enlargement of the gland, even in the absence of pelvic endometriosis. Although the absolute criteria for diagnosis of endometriosis are histologically demonstrable endometrial glands surrounded

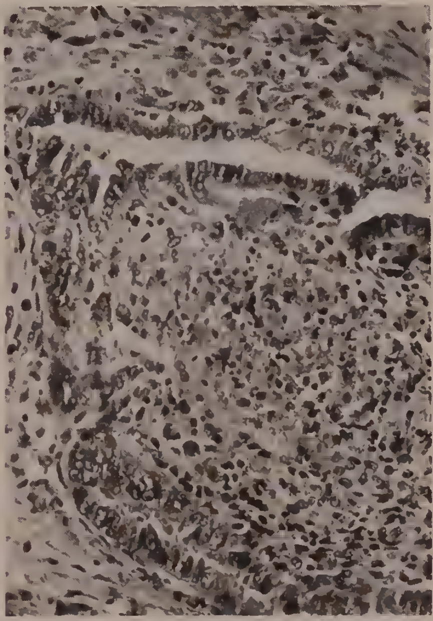


FIGURE 2. Close-up view showing proliferation of endometrial gland epithelium, inflammatory cell infiltrates, and extravasated red cells (hematoxylin-eosin stain; original magnification × 200).

by stroma, in longstanding cases extensive fibrosis may obliterate glands and stroma. Hemosiderin-laden macrophages and old blood pigment fibrosis may be the only evidence of endometriosis (Fig 1).

The origin of endometriosis in extra-abdominal pelvic sites is not fully understood. Ranney[4] states that hypotheses abound, but facts concerning the etiology of endometriosis are few. The popular theories include ceolomic metaplasia, lymphatic and vascular spread, and retrograde menstruation. Direct extension of endometriosis along the round ligament from preexisting pelvic endometriosis can explain endometriosis of the labia majora. An isolated focus of endometriosis in the Bartholin gland can, theoretically, be the result of ceolomic metaplasia, lymphatic and/or vascular spread. Hormonal stimulation can result in the growth of endometrial fragments that have reached the Bartholin gland via menstrual discharge. Endometrial fragments have the capacity to implant and grow on compatible tissue as demonstrated by the experiments of Ridley and Edward.[5] Because of this possibility, elective surgery of the cervix, vagina, or vulva should be avoided ten days prior to menstruation. Operative procedures on the lower genital tract can leave raw surfaces which can promote implantation endometriosis during subsequent menstrual bleeding.

STEPHEN MATSEOANE, MD
TOMMY HARRIS, MD
Department of Obstetrics and
Gynecology

EDWARD MOSCOWITZ, MD
Department of Pathology
Harlem Hospital Center
College of Physicians and Surgeons
of Columbia University
506 Lenox Ave
New York, NY 10037

1. Binder SS: Endometriosis of the vulva and perineum; report of a case. *Pacif Med Surg* 1965; 73:294–296.
2. Parmley TH: Screening techniques for vulvar and vaginal lesions. *Clin Obstet Gynecol* 1979; 22:683–688.
3. Ranney B: Endometriosis: Pathogenesis, symptoms, and findings. *Clin Obstet Gynecol* 1980; 23:865–874.
4. Ranney B: Etiology, prevention and inhibition of endometriosis. *Clin Obstet Gynecol* 1980; 23:875–882.
5. Ridley JH, Edward IK: Experimental endometriosis in the human. *Am J Obstet Gynecol* 1958; 76:783–789.

Tetracycline reduces the need for insulin

TO THE EDITOR: There have been scattered unsubstantiated reports that tetracycline reduces the need for insulin. Poltorak and Grinchenko and colleagues[1,2] have documented in animal studies that tetracycline binds insulin and influences its activity in some undetermined manner. Liver glycogen stores are also affected. Hiatt and Bonorris[3] postulated that oxytetracycline enhances the activity of insulin by interfering with epinephrine-induced glycemia, or increases the half-life of insulin. Those experiments were performed in pancreatectomized dogs.

Case Report. A 91-year-old woman with a long history of congestive heart failure and no history of diabetes was admitted to the hospital with a right pleural effusion involving almost 90% of the right lung volume. Clinical examination revealed a frail, pale woman in acute respiratory distress. Her heart rate was 100/min; blood pressure, 138/80 mm Hg; respiratory rate, 46/min. No breath sounds were heard over the right chest. Rales were heard in the lower half of the left lung field. Po_2 was 48 mm Hg on room air.

The patient initially improved with therapy that included diuretics, oxygen, and thoracenteses. Pleural fluid examination failed to reveal any malignant cells, and pleural biopsy was negative. Because the patient's systolic blood pressure (after diuresis) remained below 100, high doses of furosemide and captopril were not tolerated.

Because of continuous pleural drainage, one gram of tetracycline hydrochloride was instilled into the right pleural space. Two hours later, the patient became lethargic and then comatose. The blood glucose level at the time was 6 mg/dL. The patient was aroused after two intravenous boluses of 50 g of glucose. Approximately six hours later the patient again became stuporous. Blood glucose at this time was 23 mg/dL. Again she responded to intravenous glucose. For the next 48 hours, intravenous glucose solution was needed to stabilize the patient. She was eventually discharged with no recurrence of the pleural effusion. Her blood sugar level remained within normal limits.

Discussion. It is apparent that tetracycline has some direct hypoglycemic effect and probably also prolongs the insulin effect. Miller[4] reported a case of a patient with diabetes who became hypoglycemic after treatment with oral oxytetracycline. The profound hypoglycemia seen in the patient described here seems to support a hypothesis that tetracycline is either directly hypoglycemic or somehow prolongs the half-life of insulin. This case also demonstrates the need to be aware of adverse and unusual drug reactions. It is conceivable that tetracycline could be used in the future as adjunctive therapy in the treatment of diabetic patients.

VINCENT P. GARBITELLI, MD
71 East Williston Ave
East Williston, NY 11596

1. Poltorak VV, Grinchenko TS: Vliianie antibiotikov tetratsiklinovogo riada na uglevodnyĭ obmen (Effect of tetracycline series antiobiotics on carbohydrate metabolism). *Antibiotiki* 1978; 23:276–282.
2. Grinchenko TS, Poltorak VV, Rubinshtein ML: Vliianie antibiotikov tetratsiklinovogo riada na funktisiiu beta-kletok insuliarnogo apparata podzheludochnoi zhelezy (Effect of tetracycline series antiobiotics on beta cell function of pancreatic islets). *Antibiotiki* 1978; 23:1097–1102.
3. Hiatt N, Bonorris G: Insulin response in pancreatectomized dogs treated with oxytetracycline. *Diabetes* 1970; 19:307–311.
4. Miller JB: Hypoglycaemic effect of oxytetracycline [letter]. *Brit Med J* 1966; 2:1007.

BIOSTATISTICS IN CLINICAL MEDICINE

Second Edition. By Joseph A. Ingelfinger, MD, Frederick Mosteller, PhD, Lawrence A. Thibodeau, PhD, James H. Ware, PhD. 339 pp, illustrated. New York, Macmillan Publishing Co Inc, 1987. $29.95 (softcover)

This group of authors, distinguished in both medicine and biostatistics, has presented an introduction to statistical concepts and methods, with physicians as their intended audience. The book purports to prepare "physicians to use and understand applications of probability and statistics for the care of the individual patient." That goal is effectively pursued with a richness of clinical examples, used to illustrate the diverse statistical procedures discussed.

The emphasis throughout is placed upon the intuitive aspects of statistical concepts, rather than the mathematical aspects, but quantitative rigor is not sacrificed. Cogent discussions of the statistical concepts being illustrated are, however, sometimes sacrificed to lengthy discussions of the clinical problems presented as examples. Highlighting with boxes and prudent repetition of critical points are judiciously applied for emphasis.

A lucid presentation of the rules of probability as applied to problems of diagnosis should make these notions more palatable to motivated physicians, who may have been frustrated with lengthy discussions of coin tosses in other texts. Effective presentations of odds and likelihood ratios and decision trees are welcome additions to an introductory text. Requisite chapters on the basic descriptive statistics of central tendency and variability are freshly presented with the promised relevance to clinical care. The role of the common distributions in statistical inference, including the normal, Student's t, binomial, and Poisson, is also well handled. Omission of the exponential distribution in this setting is unfortunate. There is a good chapter on chi-squared analysis, and the discussion of regression analysis is enhanced with examples of common computer results of such analyses.

Hypothesis testing is afforded only cursory attention as a topic; however, one of the highpoints of the book is a chapter devoted to P values. Issues surrounding the application of P values to clinical questions are seriously discussed, including the several ambiguities of the word "significance," and the choice of the 0.05 level. The role of power in the interpretation of P values receives admirable treatment. Another highpoint is a chapter on clinical trials, in which problems associated with the design and execution of clinical trials are discussed, as are problems associated with interpretation of clinical trials. An accompanying chapter on the application of clinical trial results is less effective.

A new chapter on life table analysis represents the only important addition to this second edition. It is perhaps the most disappointing chapter in the book, providing a barely adequate introduction to this important and rapidly developing statistical technique. The contrast between the elementary treatment of life tables and the scholarly exposition of odds illustrates an unevenness of presentation, which can be a minor distraction. This is not an easy text to use, and the extent to which it has the potential to influence the delivery of patient care is problematic. However, for the physician who is seriously desirous of gaining a better understanding of statistical procedures, but whose mathematical grasp does not exceed algebra, this book is a superb introduction to probabilistic thinking. The relevance of these biostatistical concepts to a broad spectrum of clinical problems is presented both literately and authoritatively. Most of the material is complete and current, and some discussions cannot be found elsewhere.

OLIVER GLIDEWELL
Montoursville, PA 17754

THE DEVELOPMENT OF AMERICAN PHYSIOLOGY. SCIENTIFIC MEDICINE IN THE NINETEENTH CENTURY

By W. Bruce Fye, MD. 308 pp. Baltimore, Md, The Johns Hopkins University Press, 1987. $35.00 (hardcover)

This book is a timely discussion of the role of physiology in the maturation of American science and medicine. It is timely not only because 1987 marks the hundredth anniversary of the founding of the American Physiological Society, but because at present there has been a loss of the perception of physiology as an essential foundation of medicine and because major concern with reductionist thought is fragmenting the discipline and causing a loss of integrative thought so characteristic of physiology.

Following the account of the development of American physiology and the parallel revolution in American medical education given in the introduction, the author describes the careers of the men considered most responsible for these developments. Four were chosen: First, John Call Dalton, Jr: pioneer vivisector (founder of experimental teaching) and America's "First Professional Physiologist." He was founder of the Departments of Physiology at Buffalo, Vermont, what became the Long Island College of Medicine in Brooklyn, what is now the State University of New York Health Science Center at Brooklyn, and finally what is now Columbia University College of Physicians and Surgeons. A second was S. Weir Mitchell, Philadelphia's often frustrated physiologist and triumphant reformer. He provided much of the energy and drive required for organizing the American Physiological Society. The third was Henry P. Bowditch, a man of the highest prestige carrying the heritage of Carl Ludwig to America; Dalton carried Claude Bernard's. The author refers to Bowditch as the prototypical full-time physiologist and educational reformer. A thesis of the book is that American physiology attained stature when the country's institutions became able and willing to support full-time professional physiologists. The fourth leader described by the author was H. Newell Martin, who was involved in establishing physiology at the Johns Hopkins University and in developing the initial research ethic required for modern medicine.

The fifth chapter is entitled, "the American Physiological Society: a Profession Defining Itself." Although the men previously mentioned were the leaders, others were also involved. The first meeting was held in 1887 in the new physiological laboratory of the College of Physicians and Surgeons; Professor John G. Curtis was host. There were 28 original members. Some of the younger members, with a few others who were elected within the next few years, really brought American physiology to a position of preeminence after the first World War and by the 1930s. This book, however, treats only the earlier years, a quarter of the 19th century, and an early decade or two of the 20th. This chapter, like the preceding four, contains many details of the concerns, the struggles, and the attainments of the early days.

The sixth and final chapter deals with the influence of physiology on American medicine; the title is "A Scientific Prescription for American Medicine." Obviously the author deals with more than the history of the early development. There is an account of the 1910 Flexner report and its effects. The principles essential to high attainment are delineated but as the subtitle indicates, it is an account of physiology and "Scientific Medicine in the Nineteenth Century." The reader is left with the job of applying the teaching of history to today's problems.

The book concludes with appendices listing the institutions involved in the early meetings of the American Physiological Society. There is a bibiography of 28 manuscript collections and 286 individual references plus an index. The author has contributed a valuable reference work and a most interesting account of our history.

CHANDLER McC. BROOKS, PhD
State University of New York
Health Science Center
at Brooklyn
Brooklyn, NY 11203

MODERN MEDICINE AND JEWISH ETHICS

By Fred Rosner, MD. 405 pp. Hoboken, NJ, KTAV Publishing House Inc, 1986. $22.50 (hardcover)

The burgeoning developments in interventional therapeutic capabilities during the past three decades have spawned an impressive, though often less definitive, growth in general medical ethics. An early landmark in the development of Jewish medical ethics as a discrete discipline was the appearance in 1959 of a book by that name written by the present reviewer's father, Sir Immanuel Jakobovits, whose brief foreword graces the current work. By now the subject has taken off and has attracted many able writers and thinkers, medical and rabbinical, whose continuing contributions have assured the interested reader of numerous literary resources.

Fred Rosner, MD, has achieved well-earned preeminence in the secular and religious communities as a remarkably prolific and gifted exponent of the teachings an ancient religion brings to bear on many of today's medical dilemmas. As Chief of Medicine at a leading New York hospital and Professor of Medicine at the State University of New York Health Science Center at Stony Brook, he has direct contact with many of the medico-moral issues of which he writes. In addition, as a notable figure in numerous prominent regional and national medical ethics "think tanks," he is uniquely qualified; indeed, his is a virtual household name among the evergrowing cadre of religiously concerned physicians.

Modern Medicine and Jewish Ethics presents 26 topics in as many chapters culled in large part from previous essays by Dr Rosner. Virtually all the major areas of concern are touched upon. The discussions manifestly demonstrate how Jewish religious values and mores may be intelligently applied to even the most technically advanced situations. Analyses range from fetal surgery and sex-organ transplants to mechanical hearts and the appropriate use of artificial life supports. Fascinating questions regarding the allocation of scarce medical resources, smoking in public places, the definition of death, and animal experimentation are among the many thought-provoking essays which will appeal to a wide variety of readers. Conversely, several topics, such as mental health issues, are mentioned only parenthetically, and the Jewish attitudes to homosexual and other "alternate" sexual behaviors—so important in these days of the AIDS epidemic—are omitted altogether.

In recent years there has been a surge of interest at the grass roots level of the Jewish community in Tay-Sachs disease and in its prevention by mass screening. The implications of such screening and the potentially detrimental concerns which it may foster in the minds of poorly informed young men and women are sympathetically reviewed. Although the author thinks that, on balance, the risk-benefit ratio warrants such screening, the pros and cons are fairly presented. Indeed, an appealing feature of the book in general is its penchant for presenting all sides of an argument fairly and fully, and yet not leaving the reader so unguided as to be unsure of the author's position.

The division between ethics and ritual is intentionally vague in the Jewish tradition, so the inclusion of chapters on priestly defilement, embalming and cremation, and the therapeutic efficacy of prayer should engender no surprise, though they may be of less universal application than the remaining 23 sections. Otherwise, little reference is made to several important ritualistic Jewish concepts relevant to medical practice, such as the Sabbath and kosher food regulations.

The author notes that "Today, many physicians and patients do not feel firmly bound by religious precepts. Religious guidance is available, however, to those who seek it" (page 217). This book is not designed to draw readers to a religious theory of medical ethics. Those who seek, however, an authentically Jewish viewpoint on a kaleidoscope of issues will be richly rewarded by the breadth, erudition, and documentation contained within its pages.

JULIAN JAKOBOVITS, MD
Johns Hopkins University
School of Medicine
Baltimore, MD 21205

CARDIAC ARRHYTHMIAS: SELF-LEARNING

By Edward K. Chung, MD, 200 pp, illustrated. New York, Yorke Medical Books, 1986. $40.00 (hardcover)

This publication, a self-learning companion volume to the *Manual of Cardiac Arrhythmias*, should prove useful for primary care physicians, internists, medical residents, and cardiac care nurses. However, because of a lack of sufficient depth and thoroughness in certain areas, and because of some questionable interpretations in the text, it would not be suitable for cardiologists or cardiology trainees.

The book consists of nine chapters covering A-V block, intraventricular block, sinus arrhythmias, A-V junctional arrhythmias, ventricular arrhythmias, differential diagnosis of cardiac arrhythmias; Wolff-Parkinson-White Syndrome; cardiac arrhythmias related to artificial pacemakers, and miscellaneous ECG findings.

The omission of examples of some important and not uncommon arrhythmias is regrettable. These include Mobitz II type A-V block in Chapter 1, bradycardia (phase 4) related to bundle branch block in Chapter 2, Wenckebach type S-A block in Chapter 3, concealed junctional premature beats in Chapter 5, and torsades de pointes in Chapter 6. A number of cases are described wherein the author's interpretation of the arrhythmia can be questioned. For example, the diagnosis of "variant" of Wenckebach A-V block or "variant" of Mobitz II A-V block is questionable in patients with 2:1 A-V block and normal QRS complexes since the level of A-V block cannot be ascertained by surface electrocardiography (case #16). In case #43, the left anterior hemiblock is clearly rate related, and the QRS complexes without LAH do not suggest the presence of LVH. In case #54, S-A block of 4:3, 3:2, and 2:1 is unquestionably present, and the diagnosis of Mobitz II S-A block is very doubtful.

"Chung's Phenomenon," referred to in cases #138 and #191, inappropriately ascribes to Dr Chung credit for first recognizing the finding of "alteration of the P wave configuration of the sinus beat (the 4th and the 9th beats) immediately following APCs. This finding has been termed 'aberrant atrial conduction.' " Actually, this phenomenon was first reported by Sir Thomas Lewis in 1921 and again described in detail by Scherf and Schott in *Extrasystoles and Allied Arrhythmias* (1953).

Despite some of the shortcomings mentioned above, this is the first book of which the reviewer is aware that combines electrocardiographic diagnosis, an assessment of clinical significance of the arrhythmias, and a suggested therapeutic approach.

YUAN KUANG KU, MD
Long Island College Hospital
Brooklyn, NY 11201

BEAT THE ODDS

By Harold S. Solomon, MD. 311 pp, illustrated. New York, Villard Books, 1986. $19.95 (hardcover)

This book for the lay public is a comprehensive approach to awareness and the need for behavior change. It specifically addresses many aspects of personal health, including safety, macroenvironmental factors, and lifestyle factors such as substance abuse which contribute to the nation's leading causes of disability and death. In addition to educating the reader regarding the need to reduce personal health risks, it offers recommendations and methods to achieve this goal.

The author's background in internal medicine and hypertension supports this fine effort to reach people with an approach to wellness. The text has been written in terminology that the average layperson should comprehend with little difficulty. The book is an excellent resource for the physician who believes that patient education and knowledge is the key to compliance. The physical quality of the book is impressive, the print size allowing for ease of reading by all ages. A few typographical errors were noted. Lay readers may feel overwhelmed with the amount of information presented and annoyed with frequent repetition. A few terms were poorly defined (eg, ischemia, maximum heart rate). The organization of the text is simple and easy to follow. The chapter on a physician's perspective on exercise appears out of place and would seem more appropriately placed within the section offering strategies for change. Greater emphasis could have been placed on precautions concerning exercise and additional information given on the theory behind it.

The book's comprehensive perspective relates lifestyle to many disease processes. All recommendations made are consistent with the guidelines established by recognized professional organizations, such as the American Dietetic Association, the American College of Sports Medicine, and the American Heart Association. The author's objectives to educate the lay population regarding the need to modify one's life if the "odds are to be beat" are clear. The strategies offered and the opportunity for feedback on one's personal progress are what distinguishes this book from others available. *Beat the Odds* should be a part of any health educator's library.

MARY CARDAMONE
Geisinger Medical Center
Danville, PA 17822

TAKING THE WORLD IN FOR REPAIRS

By Richard Selzer, MD. 239 pp, New York, William Morrow & Co, Inc, 1986. $15.95 (cloth)

Unfortunately, I have not read any of the four books written by Dr Selzer prior to this one. His writing technique is superb but the subjects he selected for the 12 short stories in this work are odd and may be distasteful to some people. The first story is dull. It describes the daily life in a monastery he visits. While there, he finds that one of the monks has Grave's disease, and the abbot develops a carbuncle which is surgically treated by the author. The other stories involve, for example, a slaughterhouse, a laundry, a bee stuck on a tart, and a man who becomes a part of the life cycle of a guinea worm.

The last story bears the same title as the book and I found it to be the most interesting. It tells of a trip to Peru by a health care team sponsored by an organization called "International Plastic Surgeons Inc." It was founded in 1969 and serves indigent patients in third-world countries. Dr Selzer is at his best in depicting the emotional impact on the children whose deformities are corrected by this group. This book will depress those readers who are seeking pleasure and relaxation. However, those interested in creative thinking and good writing will find this book worth reading.

JULIUS E. STOLFI, MD
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

THE MEDICARE SYSTEM OF PROSPECTIVE PAYMENT. IMPLICATIONS FOR MEDICAL EDUCATION AND PRACTICE

Edited by Mohan L. Garg, ScD, and Barbara A. Barzansky, PhD. 229 pp. New York, Praeger Publishers Inc, 1986. $38.95 (hardcover)

Basically this well-referenced book argues the age-old battle between taking an action for the collective good of society versus acting for the benefit of the individual. Medicare's prospective payment system (PPS), utilizing the diagnosis related groups (DRGs) for reimbursement, is the decision that is the crux of the 15 chapters in this book that consider the implications of PPS/DRG on medical practice and education.

People in the health care industry do not have to be told that the changes ensuing from the implementation of the PPS/DRG scheme are complex, fundamental, and somewhat unpredictable. With solid historical and political background data tied into supporting evidence from the literature, the authors present their perspectives with reasoned prose and speculative predictions. Everyone may not agree with some of the optimistic projections, since the disaster trends appear more frequently in professional journals. However, the foreword does note that "not infrequently out of turmoil comes opportunity."

Topics covered include items on health care cost inflation and regulatory efforts to curtail the upward spiral, changing the practice behavior patterns of physicians, how to improve clinical decision making by physicians, new roles for physicians in hospital decision making, the impact on teaching hospitals and graduate medical education, whether or not technology development will be retarded, the role of professional review organizations, what will happen to quality assurance, utilization review strategies, legal issues including constitutionality and regulations, consideration of the morality or immorality of PPS/DRG, and the need to tighten the horizontal and vertical organization of teaching hospitals.

While a good deal of the material does seem to dwell on the academic locus of the teaching hospital and graduate medical education, the importance to the physician in his/her private office should not be negated by the titles of the chapters. For example, Schmitt and Berner discuss strategies to improve clinical decision making by physicians. They believe that PPS/DRG makes it necessary to upgrade the manner in which a physician decides to choose one form of therapy over another. Using illustrations of branching logic, analysis styles, and clinical algorithms, the authors make a case for cost containment forcing judgments into behavior practices in medical care. In a related chapter, Garg, Coulson, and Barzansky review the multiple variables of a physician's practice that influence the cost of health care. Citing research, they reiterate the fact that physicians are usually unaware of what medication costs or how much the hospital charges for lab tests or other services. Since PPS/DRG calls for an examination of the utilization of resources, the difficult task of altering medical practice patterns to contain cost expenditures must be undertaken if hospitals are to remain financially solvent. In addition to making better cost conscious decisions and using limited resources, Schroeder forecasts that new equipment in the future will only be technology that saves money while it flashes, blinks, whistles, and prints out fancy diagrams. Komaroff raises the critical issue of whether high quality care can be achieved if one has to have an accountant at one's side tabulating the cost as one sutures a wound. When does quality assurance take precedence? He does posit that the PPS/DRG system must do more to include severity of illness gradations in its payment mechanism to account for variances in clinical care within the same DRG. Loudly and clearly, the implications of PPS/DRG on the US health care delivery system appear to be punctuated with dollar signs. Although the PPS/DRG plan currently applies only to inpatients covered under Medicare, other third party payers are jumping on the bandwagon. Furthermore, the federal legislation opens the door for similar cost containment measures for other facilities, ambulatory care, psychiatric care, home care, long term care, and so on.

Callahan considers the ethical morality of the PPS/DRG plan and decides that it is not immoral to try to curtail costs. However, he does point out that conflicting loyalties arise for the physician who owes his/her patient the best care but also has a concern to keep the hospital from going bankrupt.

Freymann feels that the public will insist on good medicine rather than "Mac Medicine" even though it may cost a bit more. He sums up the situation strongly: "Beyond the balance sheet, beyond the quarterly dividends, the doctor's authority is inviolable in the end—if they exert it. Socrates said it 24 centuries ago: 'For the true physician is also a ruler having the human body as a subject, and is not a mere moneymaker.' "

To provide insight into the many facets and implications of the PPS/DRG cost containment scheme, this book is a sound investment of your time and energy.

ALLEN D. SPIEGEL, PhD
SUNY Health Science Center
at Brooklyn
Brooklyn, NY 11203

A DIABETIC DOCTOR LOOKS AT DIABETES: HIS AND YOURS

By Peter Lodewick, MD. 232 pp, illustrated. New York, Bantam Books, 1984. $3.95 (paperback)

Family physicians are always on the lookout for new patient education aids. Most commonly these take the form of materials supplied by the pharmaceutical houses or national medical societies. The lay press and major medical journals are also a good source for these materials. Occasionally, a book such as *A Diabetic Doctor Looks at Diabetes: His and Yours* comes along which succeeds in achieving its stated goal of providing patients with a better understanding of this multisystemic disease. Written by Peter A. Lodewick, MD, an endocrinologist and an insulin-dependent diabetic, this book gives its readers a detailed, precise, comprehensive overview of diabetes mellitus. Gearing his book toward diabetic patients and their families, the author makes a conscious effort to provide emotional encouragement to the nearly six million Americans suffering from this chronic disease.

The book is broad-ranging in content, and is written in a clear, concise style that is very readable. Dr Lodewick, president of the American Diabetic Association, New Jersey affiliate, guides the reader through this complex disease. By beginning with the basic definitions of diabetes mellitus and continuing through the different types of the disease and its natural course, the author makes readers aware of why therapeutic compliance is so important. Symptoms and signs are well described, and the diagnostic tools and latest treatment modalities available to physicians are covered. To help clarify his information, Dr Lodewick makes use of numerous case histories, many of them reflecting his own experiences as a patient with diabetes. I was particularly pleased to see chapters on glycohemoglobin and foot care. Other chapters in this book discuss diabetes and pregnancy, impotence, alcohol, and travel. The author concludes his book by answering the ten questions he hears most often, and by providing a list of recommended readings.

A Diabetic Doctor Looks at Diabetes: His and Yours comes across as a compassionate and upbeat guide to living an active and successful life with diabetes. While technically correct and contemporary in its tone, the book strives to provide an emotional lift to the reader. The author is successful in achieving that goal, mainly due to his insights from the dual perspective of physician and patient. As such he appears unafraid to discuss the emotional issues faced by newly diagnosed diabetics.

Although many instructional texts on diabetes are available, *A Diabetic Doctor Looks at Diabetes: His and Yours* is a valuable asset to its readers. It is written for the nonprofessional and offers both medical and emotional advice. Its price is not prohibitive, and both patients and primary care physicians will find this a helpful and enjoyable book to read.

DAVID BRECHER, MD
Mease Clinic Countryside
Safety Harbor, FL 33572

AMA's socioeconomic survey

The American Medical Association's Center for Health Policy Research released its most recent Socioeconomic Monitoring Survey Report. According to the report, the major problems facing physicians are the increasing cost of liability insurance and the growing number of claims (*JAMA* 1987; 258:455).

More specifically, the report states that annual liability insurance premiums averaged $10,000 in 1985. Paying $23,000, on average, obstetricians/gynecologists claimed the highest rates. During 1985, the average annual rate of claims rose to 10.1/100 physicians, compared to 8.2/100 for the previous four years. In addition, close to 9% of physicians faced at least one claim in 1985.

Increasing power of medical malpractice insurers

Malpractice insurers are setting guidelines and rules that will govern how physicians practice medicine. Failure to abide by these guidelines can result in the loss of liability coverage. What are the medical, legal, and economic ramifications of such a system? Mark Crane examined these issues in a special report for *Medical Economics* (1987; 64:23–29).

The arrangement is as follows: In exchange for supplying malpractice insurance, the insurers want to dictate terms regarding certain medical practices, including drug prescription, the decision to perform specific procedures, referral to specialists, and record-keeping practices. The insurers' goal is to reduce the risk associated with patient care, particularly involving those specialties and procedures associated with high risk and, ultimately, higher insurance losses. The "insurability guidelines," which have been adopted by almost half of the companies included in a recent survey by the Physician Insurers Association of America, focus on the practice of obstetrics and anesthesia, the diagnosis of breast cancer, and the issue of informed consent.

Penalties for defying the guidelines can range from surcharges of up to $25,000 to loss of coverage. Insurers may also refuse to renew coverage if physicians perform a procedure for which they are not appropriately trained. Compliance with the rules is generally determined after a claim is filed. Although companies will defend a client even if his or her practices were found to deviate from the rules, loss of coverage commonly results.

As liability insurance premiums skyrocket for obstetricians, as well as for family practitioners who deliver babies, one company has instituted the following guidelines: High-risk patients should be referred to an obstetrician; referral is required for patients with insulin-dependent diabetes, severe eclampsia or pre-eclampsia, multiple gestation, history of previous cerclage, hydatiform mole, serious cardiac or hematologic diseases, or confirmed intrauterine growth retardation. Furthermore, physicians must use "a prenatal scoring system for every patient to determine risk and appropriate management in six areas: cesarean sections, use of instruments, management of hypertension, monitoring of fetal distress, management of pre- and postmature deliveries, and administration of oxytocin." For example, the guidelines state that only a physician can administer oxytocin, the rate of delivery of which is set in the guidelines, and patients receiving oxytocin must have electronic fetal monitoring.

Another insurance company has applied stringent requirements to anesthesiologists and has achieved a dramatic reduction in paid claims. As a result, the company was able to reduce premiums for anesthesiologists. The guidelines included requiring the presence of an anesthesiologist or a certified nurse-anesthetist in the operating room at all times, and requiring the use of two simultaneous methods of pulse and blood pressure monitoring, as well as predetermined standards governing the use of regional and general anesthesia.

These insurance company-sponsored programs have been in effect for, at most, two years; their impact on malpractice claims, liability premiums, and medical practice and patient care is not yet known. Notes author Crane, "The financial impetus for the insurers is compelling, and the legal impact on physicians could be far-reaching."

Health effects of radon exposure

Increasing awareness of the sometimes high levels of radon in homes has led to widespread media attention and numerous studies on the health effects of radon exposure. Despite the limited data available on radon levels in North American homes, the results of preliminary studies suggest that exposure to excessive radon levels may be responsible for several thousand cases of lung cancer each year. The Council on Scientific Affairs of the American Medical Association (AMA) has reviewed the data on radon exposure, evaluated the resulting health effects, and developed three basic recommendations (*JAMA* 1987; 258:668-672).

Radon 222 is a radioactive gas that forms from the decay of radium 226. Radium is a mineral irregularly distributed throughout the earth's crust and present in earth-based materials such as soil and rock. As this radium decays, the radon gas rises through the soil to enter the atmosphere. If allowed to enter a home through joints or cracks in the foundation, radon can accumulate to hazardous levels. When inhaled, the radon continues to decay, thus irradiating pulmonary tissues.

The US Environmental Protection Agency (EPA) has recommended the following: concentrations in homes should not exceed 0.02 WL (working level) of radon decay products ("equivalent to 4pCi of radon per liter of air assuming 50% equilibrium"). Investigators have concluded that the average radon level in US homes is 1.5 pCi/L, although large variations have been documented. One to three percent of homes had levels that exceeded 8 pCi/L.

"A primary determinant of the level of radon in a house is the makeup of the earth under and around the house, especially the pressure of radon gas in the soil and pressure differentials between the soil and the house." Ventilation is another major determinant of radon levels; ex-

---

NEWS BRIEFS are compiled and written by Vicki Glaser.

hausting indoor air with fans or using a heat exchanger can reduce radon levels. Energy-saving techniques, including insulating, weatherstripping windows and doors, and caulking, virtually seal a house and can increase the level of radon gas.

Another important source of radon is the water supply. Water may account for as much as 35% of the increased level of radon inside a house as compared to outside. Radon escapes from the water during routine activities such as washing, drinking, and cleaning. Regions with the highest water levels of radon are the Appalachian-Piedmont area and New England.

The health effects associated with exposure to radioactive decay products are not well understood. Previous studies have focused on the risks to uranium miners. "A conservative position of protection against adverse effects of radiation assumes that the risk of developing radiogenic malignant neoplasms is proportional to radiation exposure." However, the risks of exposure to low-level radiation are not clear. One investigator determined that the average person suffers exposures of about 0.2 WLM (working-level-month)/year. This would lead to somewhere between 1,000 and 20,000 cases of lung cancer each year in the US. Other estimates suggest that 10,000 cases of cancer per year may be due to radon in homes.

The Council on Scientific Affairs has recommended that the AMA play an active role in the education of physicians, other health care professionals, and the public as to the potential risks of high radon levels in homes and other buildings. In addition, the Council recommended that the AMA should catalyze the organization of a conference to address the issue of radon. Other participants would include the Environmental Protection Agency, the Department of Energy, and the National Institute of Environmental Health Sciences. Finally, according to the Council, it is imperative that accurate measures be obtained of ambient radon levels in a sample of homes, workplaces, and commercial buildings.

Carpal tunnel syndrome: cause and treatment

Two recent reports have focused on carpal tunnel syndrome, which is characterized by numbness and/or burning pain on the palmar surface of the first three digits of the middle hand. The disorder results from compression of the median nerve at the carpal tunnel. Risk factors for carpal tunnel syndrome include hypothyroidism, rheumatoid arthritis, pregnancy, diabetes mellitus, acromegaly, amyloidosis, neuromas, and stress on the nerve caused by repeated flexion and extension at the wrist.

Barnhart and Rosenstock reported a cluster of seven patients with carpal tunnel syndrome, all of whom were grocery checkers (*West J Med* 1987; 147:37–40). As grocery checking requires highly repetitive motions involving the wrist, and no other risk factors were apparent, the authors concluded that in this patient group, carpal tunnel syndrome was probably a result of job-related activity. A review of the literature supported this conclusion.

Surgical decompression of the median nerve at the carpal tunnel is the definitive mode of treatment. Conservative therapeutic alternatives include splinting of the wrist, injection of hydrocortisone into the carpal canal, and the use of nonsteroidal anti-inflammatory agents.

Ellis and colleagues have demonstrated a correlation between the clinical symptoms of carpal tunnel syndrome (CTS) and the specific activity of erythrocyte glutamic oxaloacetic transaminase (EGOT), an enzyme that is released into the serum following tissue injury (*South Med J* 1987; 80:882–884). The authors reported that "the lower the EGOT activity the more severe and more numerous were the signs and symptoms of CTS." This finding is based on a study of 35 selected patients with carpal tunnel syndrome, who had the more common, idiopathic, nonspecific form of the syndrome, with no known predisposing disorders.

Pyridoxamine, an active form of vitamin B_6, acts as a coenzyme with EGOT; EGOT cannot function without the help of vitamin B_6. Thus, the EGOT test can be used to determine a person's vitamin B_6 status. Ellis treated the 35 patients with 100–200 mg/day of vitamin B_6. Following four weeks of this therapy, the vitamin deficiency was corrected. Symptoms of carpal tunnel syndrome improved or resolved following 12 weeks of therapy. Improvements included reduced swelling of hands, improved flexion of finger joints, increased pinch pressure, and diminished paresthesia in the fingers. Ellis noted improved results on electromyographic tests following vitamin B_6 therapy, "but not a return to normal even when clinical improvement was significant."

The recommended dietary allowance (RDA) of vitamin B_6 is 2.2 mg/day for men, 2.0 mg for women, and 2.6 mg for pregnant women. "I recommend that 50 mg of vitamin B_6 be given daily, beginning with diagnosis of pregnancy," states the author. "In the presence of edema of pregnancy, 200 mg is preferrable to prevent CTS in the third trimester of pregnancy." For the general population, Ellis concluded, "the US RDA of 2 mg for vitamin B_6 is marginal for CTS."

CPMC seeking participants for three multicenter trials

Columbia-Presbyterian Medical Center (CPMC) in New York is participating in three national, multicenter trials to study new treatment methods for Parkinson disease, stroke, and sudden cardiac death. The Neurological Institute at CPMC will test the effectiveness of two drugs—deprenyl and tocopherol—in delaying the progressive, debilitating effects of Parkinson disease.

The institute is also involved in the five-year, Asymptomatic Carotid Atherosclerosis Study, which will include 1,500 patients nationwide. The study will determine whether a surgical approach to stroke prevention, using the carotid endarterectomy, is more effective than treatment with aspirin. Eligible patients will exhibit significant narrowing of the carotid artery due to atherosclerosis, with no symptoms of stroke.

The Cardiac Arrhythmia Suppression Trial will encompass 138 hospitals and 5,000 patients in the United States, Canada, and Sweden. Researchers will study the effectiveness of three antiarrhythmic drugs—encainide, flecainide, and morcizine—in preventing sudden cardiac death.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Leon Benton, MD, St Louis, Missouri. Died April 17, 1987; age 78. Dr Benton was a 1936 graduate of Deutsche Univerzita Medizinishe Fakulta, Praha, Czechoslovakia. He was a member of the Medical Society of the County of Ulster and the Medical Society of the State of New York.

George Blinick, MD, Boca Raton, Florida. Died June 6, 1987; age 79. Dr Blinick was a 1932 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Academy of Medicine, the New York Obstetrical Society, the New York County Medical Society, and the Medical Society of the State of New York.

Bernard Bloom, MD, Kiamesha Lake. Died September 10, 1986; age 66. He was a 1948 graduate of Chicago Medical School, Chicago. Dr Bloom was a member of the Sullivan County Medical Society and the Medical Society of the State of New York.

Frances L. Hacker Bogatko, MD, Rome. Died June 27, 1987; age 95. Dr Bogatko was a 1917 graduate of the Medical College of Pennsylvania, Philadelphia. She was a member of the New York County Medical Society and the Medical Society of the State of New York.

Jean Pierre Bonin, MD, Sidney. Died June 6, 1987; age 58. Dr Bonin was a 1962 graduate of Faculte de Medicine de l'Universite de Lausanne, Lausanne, Switzerland. He was a member of the Delaware County Medical Society and the Medical Society of the State of New York.

Eugene Braude, MD, New York. Died May 31, 1987; age 87. He was a 1925 graduate of Medizinische Fakultaet der Ludwig Maximilians Universitaet, Muenchen, Bayern, Germany. Dr Braude was a member of the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Lubomyr Bylow, MD, Port Crane. Died June 1987; age 75. Dr Bylow was a 1945 graduate of Charles Univerzita Fakulta of Ped-Gen Medicine, Praha, Czechoslovakia. Dr Bylow was a member of the American Psychiatric Association, the Broome County Medical Society, and the Medical Society of the State of New York.

Lawrence Thomas Cacioppo, MD, St Augustine, Florida. Died June 25, 1987; age 87. Dr Cacioppo was a 1924 graduate of State University of New York Health Science Center at Brooklyn. He was a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Otolaryngology. His memberships included the American Academy of Otolaryngology, the American Academy of Facial Plastic and Reconstructive Surgery, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Alexander Cameron, MD, Sarasota, Florida. Died May 8, 1987; age 92. Dr Cameron was a 1919 graduate of New York University College of Medicine, New York. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Gerard Ross Carroll, MD, Highland Falls. Died June 6, 1987; age 66. He was a 1952 graduate of Faculty of Medicine University of Glasgow, Glasgow, Scotland. Dr Carroll was a Diplomate of the American Board of Family Practice and a member of the Medical Society of the County of Orange and the Medical Society of the State of New York.

June G. Corey, MD, Suffern. Died June 21, 1987; age 38. She was a 1974 graduate of New York Medical College, New York. Dr Corey was a Diplomate of the American Board of Family Practice and a member of the American Academy of Family Practice, the Medical Society of the County of Rockland, and the Medical Society of the State of New York.

Marie Leonore Cote, MD, Newburgh. Died June 6, 1987; age 67. She was a 1945 graduate of Albany Medical College of Union University, Albany. Dr Cote was a Diplomate of the American Board of Pediatrics and a member of the American Academy of Pediatrics, the Medical Society of the County of Orange, and the Medical Society of the State of New York.

Clarence De La Chapelle, MD, New York. Died June 13, 1987; age 90. Dr De La Chapelle was a 1922 graduate of New York University School of Medicine, New York. He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the Academy of Medicine, the New York Pathological Society, the International Academy of Pathology, the American Association of Pathologists and Bacteriologists, the New York County Medical Society, and the Medical Society of the State of New York.

Felice J. De Stefano, MD, Brooklyn. Died July 5, 1987; age 73. Dr De Stefano was a 1938 graduate of Creighton University of Medicine, Omaha. Dr De Stefano was a Qualified Fellow of the International College of Surgeons and a member of the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Harry David Fein, MD, New York. Died July 21, 1987; age 75. He was a 1936 graduate of New York University School of Medicine, New York. Dr Fein was a Fellow of the American College of Physicians and the American College of Gastroenterology and a Diplomate of the American Board of Internal Medicine. His memberships included the Academy of Medicine, the New York Gastroenterological Society, the American Gastro-Enterological Association, the New York County Medical Society, and the Medical Society of the State of New York.

Louis Joel Feit, MD, New York. Died May 2, 1987; age 79. He was a 1934 graduate of New York Medical College. Dr Feit was a Fellow of the American College of Surgeons, a Qualified Fellow of the International College of Surgeons, and a Diplomate of the American Board of Otolaryngology. His memberships included the American Academy of Otolaryngology, the American Academy of Facial Plastic and Reconstructive Surgery, the New York County Medical Society, and the Medical Society of the State of New York.

Mayer Fisch, MD, New York. Died May 7, 1987; age 61. Dr Fisch was a 1948 graduate of New York Medical College, New York. He was a Diplomate of the American Board of Psychiatry and Neurology and a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Joseph Osias Fisher, MD, Portland, Oregon. Died May 30, 1987; age 89. Dr Fisher was a 1927 graduate of the University of Maryland School of Medicine, Baltimore. He was a member of the American Academy of Family Practice, the American Geriatrics Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

Jesse Morris Frankel, MD, Brooklyn. Died June 23, 1987; age 87. He was a 1923 graduate of New York University School of Medicine, New York. Dr Frankel was a Fellow of the American College of Surgeons, a Diplomate of the American Board of Obstetrics and Gynecology, and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Meyer Friedenson, MD, Yonkers. Died January 19, 1987; age 82. Dr Friedenson was a 1927 graduate of Yale University School of Medicine, New Haven. He was a Fellow of the American College of Physicians, a Diplomate of the American Board of Internal Medicine, and a member of the New York County Medical Society and the Medical Society of the State of New York.

Antonio R. Ginorio, MD, Fort Lauderdale, Florida. Died July 15, 1987; age 69. He was a 1944 graduate of Loyola University Stritch School of Medicine, Maywood, Illinois. Dr Ginorio was a Diplomate of the American Board of Orthopedic Surgery and a member of the American Academy of Orthopaedic Surgeons, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Joseph G. Glasser, MD, New York. Died June 12, 1987; age 77. Dr Glasser was a 1937 graduate of Medizinische Fakultaet der Universitaet Basel, Basel, Switzerland. He was a Fellow of the American Academy of Family Practice and a member of the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Meyer E. Golob, MD, New York. Died July 10, 1987; age 75. Dr Golob was a 1938 graduate of New York Medical College, New York. He was a Fellow of the American Psychiatric Association and a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Henry Abraham Gozan, MD, Coconut Creek, Florida. Died July 6, 1987; age 75. He was a 1935 graduate of State University of New York Health Science Center at Brooklyn. Dr Gozan was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the American Fertility Society, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Julius Hauer, MD, Brooklyn. Died July 5, 1986; age 86. Dr Hauer was a 1927 graduate of Deutsche Univerzita Medizinische Fakulta, Praha, Czechoslovakia. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Frank Manley Higgins, MD, Roswell, Georgia. Died July 11, 1987; age 84. Dr Higgins was a 1928 graduate of Queens University Faculty of Medicine, Kingston, Ontario, Canada. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Margot Hoffman, MD, San Jose, California. Died June 15, 1987; age 88. Dr Hoffman was a 1927 graduate of Schlesische-Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Breslau, Germany. Dr Hoffman was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Albert W. Horowitz, MD, North Miami Beach, Florida. Died June 5, 1987; age 76. He was a 1937 graduate of Universitaet Wien, Medizinische Fakultaet, Wien, Austria. Dr Horowitz was a Fellow of the American College of Surgeons and the American College of Obstetricians and Gynecologists, and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Harriet Hosmer, MD, Buffalo. Died July 13, 1987; age 91. She was a 1921 graduate of the Cornell University Medical College, New York. Dr Hosmer was a member of the Academy of Medicine, the American Academy of Family Practice, the American Medical Women's Association, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Matthew H. Jaffe, MD, Vestal. Died July 10, 1987; age 75. Dr Jaffe was a 1937 graduate of New York University School of Medicine, New York. He was a Diplomate of the American Board of Urology and a member of the American Urological Association, the Broome County Medical Society, and the Medical Society of the State of New York.

Samuel F. Jenkins, MD, New York. Died April 20, 1987; age 91. Dr Jenkins was a 1928 graduate of New York University School of Medicine, New York. He was a Diplomate of the American Board of Pediatrics and a member of the American Public Health Association, the New York County Medical Society, and the Medical Society of the State of New York.

Sanford Kaminester, MD, San Diego, California. Died June 3, 1987; age 83. He was a 1927 graduate of State University of New York Health Science Center at Brooklyn. Dr Kaminester was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, a Diplomate of the American Board of Obstetrics and Gynecoloy, and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Aaron Kelman, MD, Richmond Hill. Died July 5, 1987; age 69. He was a 1945 graduate of Middlesex University School of Medicine, Waltham, Massachusetts. Dr Kelman was a member of the American Rheumatism Association, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Lionel Klein, MD, Great Neck. Died May 21, 1987; age 74. He was a 1938 graduate of State University of New York Health Science Center at Brooklyn. Dr Klein was a Qualified Fellow of the International College of Surgeons. His memberships included the New York State Society of Occupational Medicine, the American Occupational Medical Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Adalbert Kroo, MD, New York. Died May 5, 1987; age 86. Dr Kroo was a 1929 graduate of Facolta di Medicina e Chirurgia dell'Universita di Napoli, Napoli, Italy. Dr Kroo was a member of the American Academy of Family Practice, the New York County Medical Society, and the Medical Society of the State of New York.

Harry George LaForge, MD, Buffalo. Died May 31, 1987; age 85. He was a 1934 graduate of State University of New York at Buffalo School of Medicine, Buffalo. Dr LaForge was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. His memberships included the Medical Society of the County of Erie and the Medical Society of the State of New York.

Tadeusz J. Lewandowski, MD, Snyder. Died June 2, 1987; age 74. Dr Lewandowski was a 1939 graduate of Akademia Medyczna Wydzial Lekarski, Poznan, Poland. Dr Lewandowski was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

William W. Marmar, MD, New York. Died April 15, 1987; age 87. Dr Marmar was a 1925 graduate of Tufts University School of Medicine, Boston. He was a member of the American Academy of Family Practice, the New York County Medical Society, and the Medical Society of the State of New York.

William F. Meinhardt, MD, Saratoga Springs. Died June 5, 1987; age 58. Dr Meinhardt was a 1953 graduate of University of Pennsylvania School of Medicine, Philadelphia. He was a Diplomate of the American Board of Internal Medicine and a member of the New York State Society of Internal Medicine, the Medical Society of the County of Saratoga, and the Medical Society of the State of New York.

Francis P. Melomo, MD, Brooklyn. Died May 31, 1987; age 81. He was a 1930 graduate of State University of New York Health Science Center at Brooklyn. Dr Melomo was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Saul L. Meylackson, MD, New York. Died May 18, 1987; age 96. Dr Meylackson was a 1914 graduate of Fordham University School of Medicine, New York. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Alfred Adam Mitchell, MD, Elmira. Died June 20, 1987; age 73. Dr Mitchell was a 1938 graduate of State University of New York at Buffalo School of Medicine, Buffalo. He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. He was a member of the Medical Society of the County of Chemung and the Medical Society of the State of New York.

James R. Montgomery, MD, Naples, Florida. Died June 15, 1987; age 82. Dr Montgomery was a 1931 graduate of Case Western Reserve University School of Medicine, Cleveland. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

George E. Murphy, MD, New York. Died July 16, 1987; age 69. Dr Murphy was a 1943 graduate of the University of Pennsylvania School of Medicine, Philadelphia. He was a member of the Academy of Medicine, the New York Pathological Society, the American Association of Pathologists and Bacteriologists, the American Rheumatism Association, the Society for Experimental Biology and Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Imre Ney, MD, Brooklyn. Died May 14, 1987; age 73. Dr Ney was a 1938 graduate of Facolta di Medicina e Chirurgia dell'Universita di Bologna, Bologna, Italy. Dr Ney was a Fellow of the American College of Anesthesiologists and a member of the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Vincent A. Parnell, MD, Bayside. Died July 13, 1987; age 72. He was a 1943 graduate of Creighton University School of Medicine, Omaha. Dr Parnell was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. His memberships included the Medical Society of the County of Queens and the Medical Society of the State of New York.

Abraham Harold Perman, MD, Bronx. Died June 15, 1987; age 83. Dr Perman was a 1935 graduate of Rush Medical College, Chicago. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

William Alfred Petry, MD, Rochester. Died June 17, 1987; age 81. He was a 1932 graduate of McGill University Faculty of Medicine, Montreal, Quebec, Canada. Dr Petry was a Diplomate of the American Board of Internal Medicine and a member of the Academy of Medicine, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Jerome Pincus, MD, Cedarhurst. Died July 23, 1987; age 66. He was a 1944 graduate of State University of New York Health Science Center at Brooklyn. Dr Pincus was a member of the New York Cardiological Society, the New York State Society of Internal Medicine, the American Geriatrics Society, the American Public Health Association, the American Society of Internal Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

Daniel F. Primeau, MD, Kenmore. Died May 27, 1987; age 60. He was a 1950 graduate of State University of New York at Buffalo School of Medicine, Buffalo. Dr Primeau was a Diplomate of the American Board of Obstetrics and Gynecology and a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Russell Elsworth Reitz, MD, New Hartford. Died May 22, 1987; age 72. He was a 1940 graduate of State University of New York at Buffalo School of Medicine, Buffalo. Dr Reitz was a Diplomate of the American Board of Ophthalmology. His memberships included the American Academy of Ophthalmology and Otolaryngology, the Pan American Association of Ophthalmology, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Andries I. Roodenburg, MD, Rochester. Died June 23, 1987; age 77. He was a 1938 graduate of Faculteit der Geneeskunde Universiteit van Amsterdam, Amsterdam, Netherlands. Dr Roodenburg was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the Academy of Medicine, the American Rheumatism Association, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Stephan F. Saxl, MD, New York. Died July 17, 1987; age 89. Dr Saxl was a 1923 graduate of Universitaet Wien Medizinische Fakultaet, Wien, Austria. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Joseph F. Shanaphy, MD, Staten Island. Died July 10, 1987; age 66. He was a 1945 graduate of State University of New York Health Science Center at Brooklyn. Dr Shanaphy was a Fellow of the American College of Surgeons and the American College of Obstetricians and Gynecologists, a Qualified Fellow of the International College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. His memberships included the American Fertility Society, the Richmond County Medical Society, and the Medical Society of the State of New York.

Harold L. Siegelbaum, MD, Suffern. Died July 3, 1987; age 65. He was a 1946 graduate of New York Medical College, New York. Dr Siegelbaum was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Medical Society of the County of Rockland and the Medical Society of the State of New York.

Victor Stern, MD, Westport, Connecticut. Died June 20, 1987; age 79. Dr Stern was a 1933 graduate of Universitaet Wien Medizinische Fakultaet Wien, Austria. He was a member of the American Geriatrics Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

S. Henry Thaler, MD, Bal Harbor, Florida. Died May 5, 1987; age 83. Dr Thaler was a 1931 recipient of registrable qualification granted by the Society of Apothecaries of London, England. Dr Thaler was a Qualified Fellow of the International College of Surgeons and a member of the Pan American Association of Ophthalmology, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Joseph C. Todoro, MD, Hamburg. Died May 22, 1987; age 65. He was a 1947 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. Dr Todoro was a Diplomate of the American Board of Ophthalmology. His memberships included the American Academy of Ophthalmology and Otolaryngology, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Burton W. Wilcke, MD, Rensselaer. Died May 28, 1987; age 78. Dr Wilcke was a 1935 graduate of University of Iowa College of Medicine, Iowa City. He was a member of the American Academy of Family Practice, the Medical Society of the County of Rensselaer, and the Medical Society of the State of New York.

Willard H. Willis, MD, New Hartford. Died May 29, 1987; age 77. He was a 1936 graduate of Ohio State University College of Medicine, Columbus. Dr Willis was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the New York State Society of Internal Medicine, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Charles C. Wolcott, MD, Bronxville. Died June 8, 1987; age 95. Dr Wolcott was a 1917 graduate of the University of Michigan Homeopathic Medical School, Ann Arbor. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Otolaryngology. His memberships included the American Academy of Otolaryngology; the American Broncho-Esophagological Association; the American Laryngological, Rhinological and Otological Society; the Medical Society of the County of Westchester; and the Medical Society of the State of New York.

NOTICE TO ALL PHYSICIANS

MSSNY SYMPOSIUM

"FOCUS ON WOMEN PHYSICIANS"

Saturday November 7, 1987
8:30 AM – 3:30 PM
(*Luncheon Included*)

Where: **THE WARWICK HOTEL**
54th Street & Avenue of the Americas
New York City: (212) 247-2700

Program Topics:

• **BREAST CANCER**	VICKI L. SELTZER, MD, *Author of Every Woman's Guide to Breast Cancer*
• **CHALLENGES FACING WOMEN PHYSICIANS** ***. . . A Welcome to a Crowded Field***	JUDITH LORBER PhD, *Author of Women Physicians: Careers, Status & Power*
• **AIDS**	JOYCE WALLACE, MD, discusses her Greenwich Village practice, consisting largely of AIDS patients (more and more of whom are women)
• **WORKING IN ORGANIZED MEDICINE**	PALMA FORMICA, MD, President-Elect, Medical Society of New Jersey
• **WOMEN PHYSICIANS AND STRESS:** ***Patterns of Drug and Alcohol Abuse from Medical School to Retirement***	MATILDA RICE, MD, Assistant Clinical Professor of Psychiatry, SUNY HSC, Stony Brook
• **LIFESTYLE PANEL**	Women physicians look at lifestyle issues from different perspectives, including dual physician marriages and marriages with non-physicians

CME Credit: Application has been made for 6 Category I CME credits.

Registration Fees:

Members of the MSSNY	$50
Nonmembers	$65
Student or resident members of the MSSNY	$30
Nonmember students or residents	$40

MSSNY will refund the difference between the member and nonmember registration fee to any registrant whose completed membership application and dues payment are received by the symposium date.

TO REGISTER, Please complete the form on the reverse side of this page.

REGISTRATION FORM

Please complete and return to:

FOCUS ON WOMEN PHYSICIANS

Division of Membership Support Services
Medical Society of the State of New York
420 Lakeville Rd, Lake Success, NY 11042
(516) 488-6100, Ext. 252

NAME (please print) ______________________________

ADDRESS ______________________________

CITY ____________________ **STATE** ____________________ **ZIP** ____________

PHONE NUMBER (   ) ____________________

Enclosed is a check made payable to MSSNY in the amount checked below:

(   ) $30—Medical student member of MSSNY

(   ) $30—Resident member of MSSNY and ____________________ County Medical Society

(   ) $40—Nonmember student or resident

(   ) $50—Other MSSNY member (through ____________________ County Medical Society)

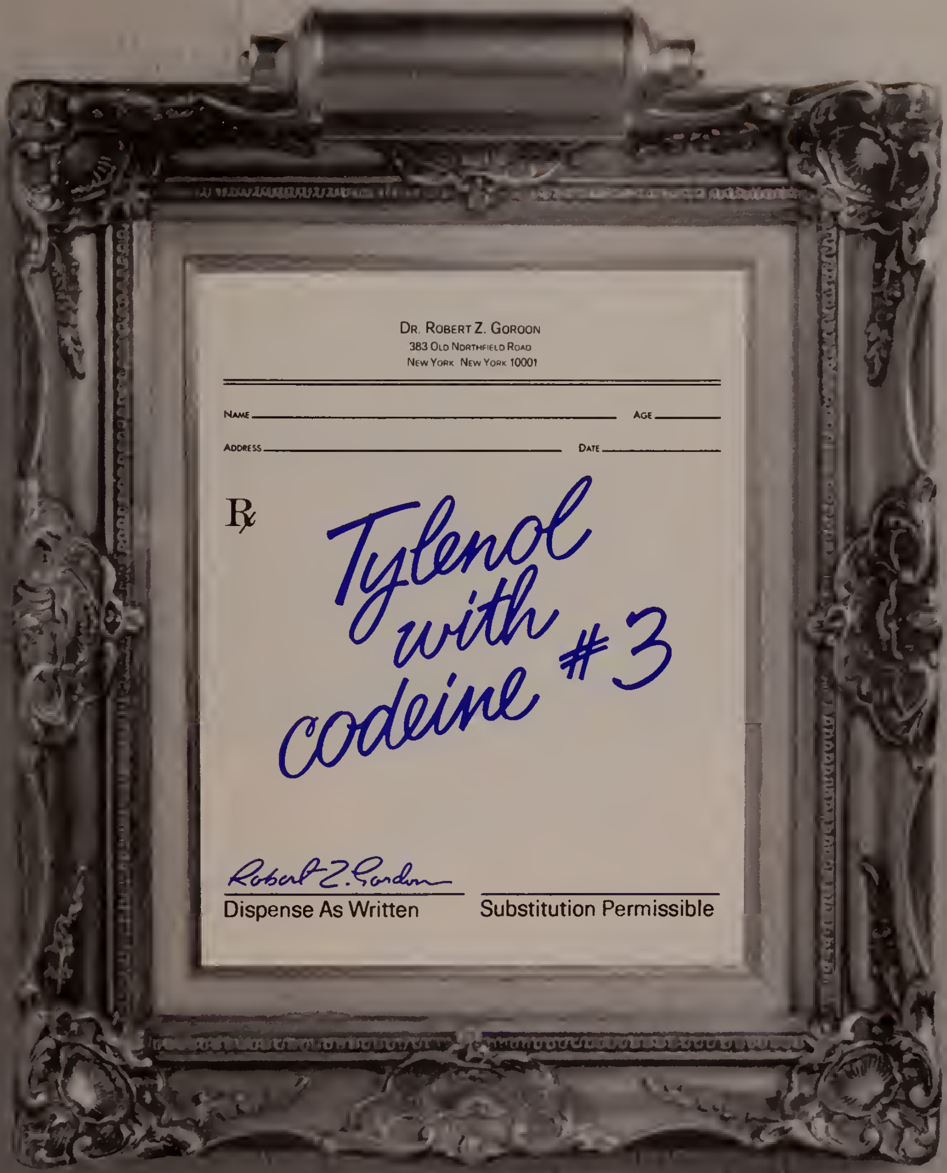
(   ) $65—Other nonmember

(   ) Please send me membership information and an application form for membership through ____________________ County Medical Society

MSSNY WILL REFUND THE DIFFERENCE BETWEEN THE MEMBER AND NONMEMBER REGISTRATION FEE FOR ANY REGISTRANT WHOSE COMPLETED MEMBERSHIP APPLICATION FORM AND DUES PAYMENT ARE RECEIVED BY THE SYMPOSIUM DATE

(   ) I cannot attend at this time. Please keep me informed of future programs.

REFUNDS WILL BE GIVEN IF REQUESTED UP TO ONE WEEK PRIOR TO SYMPOSIUM



Dr. Robert Z. Gordon
383 Old Northfield Road
New York New York 10001

Name ____________ Age ______

Address ____________ Date ______

℞

Tylenol with codeine #3

Robert Z. Gordon

Dispense As Written ____________ Substitution Permissible

CREATE A SIGNED ORIGINAL

to be sure your patients get...

Tylenol®
ACETAMINOPHEN
with codeine
phosphate

TABLETS Ⓒ III ELIXIR Ⓒ V

Tablets: Contain Codeine Phosphate*: No. 3 — 30 mg;
No. 4 — 60 mg — plus Acetaminophen 300 mg.
Elixir: Each 5 mL contains 12 mg Codeine Phosphate* plus
120 mg Acetaminophen (Alcohol 7%).
*Warning: May be habit forming.

© McNEILAB INC. 1987

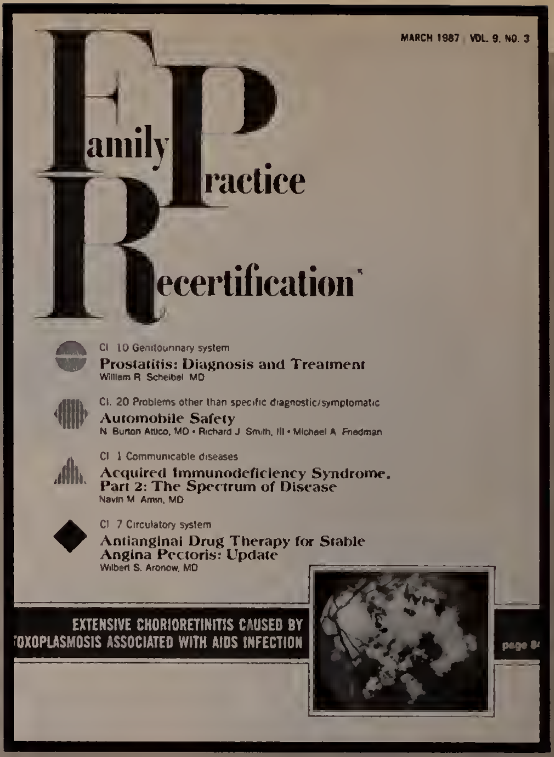
TYCT 6445-A

The complete journal for family practice physicians

- Reaches 79,000 family physicians monthly
- Presents the most commonly seen patient problems in family practice
- Written by physicians for physicians
- The most current clinical updates in:

Cardiology	Ob/Gyn
Diabetes	Psychiatry
Pediatrics	Gastroenterology

- Provides 20 hours of CME Category 1 Credit



MARCH 1987 VOL. 9, NO. 3

Family Practice Recertification

Cl 10 Genitourinary system
Prostatitis: Diagnosis and Treatment
William R Scheibel MD

Cl. 20 Problems other than specific diagnostic/symptomatic
Automobile Safety
N Burton Attico, MD • Richard J Smith, III • Michael A Friedman

Cl 1 Communicable diseases
Acquired Immunodeficiency Syndrome, Part 2: The Spectrum of Disease
Navin M Amin, MD

Cl 7 Circulatory system
Antianginal Drug Therapy for Stable Angina Pectoris: Update
Wilbert S. Aronow, MD

EXTENSIVE CHORIORETINITIS CAUSED BY TOXOPLASMOSIS ASSOCIATED WITH AIDS INFECTION

page 8

PRACTICAL ▪ CLINICAL ▪ EDUCATIONAL ▪ CURRENT

(*continued from p 4A*)

FLORIDA

Nov 12–15. **Flexible Fiberoptic Sigmoidoscopy in the Diagnosis of Colorectal Diseases.** 15 Cat 1 Credits. Buena Vista Palace, Lake Buena Vista. Contact: Seminars & Symposia, Inc, 132 East 70th Street, New York, NY 10021. Tel: (212) 517-7520.

Nov 13–14. **The Distal Radioulnar Joint—Problems and Procedures.** 12¼ Cat 1 Credits. Hotel Royal Plaza, Lake Buena Vista. Contact: American Society for Surgery of the Hand, 3025 Parker Rd, Suite 65, Aurora, CO 80014. Tel: (303) 755-4588.

ILLINOIS

Nov 5–7. **11th National Conference on Correctional Health Care.** Palmer House and Towers, Chicago. Contact: Jodie Manes, National Commission on Correctional Health Care, 2000 North Racine, Suite 3500, Chicago, IL 60614. Tel: (312) 528-0818.

MARYLAND

Nov 9–11. **Recent Advances in the Biomedical Significance of TRH.** Hyatt Regency Hotel, Bethesda. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd Street, New York, NY 10021.

MASSACHUSETTS

Nov 2–5. **Broadening the Skills of the General Care Internist, VI.** Ritz-Carlton Hotel, Boston. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

Nov 4–6. **Gynecologic Microsurgery and Laser.** 21 Cat 1 Credits. University of Massachusetts Medical Center, Worcester. Contact: Registrar, The American College of Obstetricians and Gynecologists, 600 Maryland Ave SW, Suite 300 East, Washington, DC 20024-2588. Tel: 1-800-633-8811.

AROUND THE WORLD

MEXICO

Nov 30–Dec 2. **Infertility and Reproductive Endocrinology.** 16 Cat 1 Credits. Acapulco Princess, Acapulco. *Dec 3–5.* **Advanced Colposcopy.** Acapulco Princess, Acapulco. Contact: American College of Gynecologists, Registrar, 600 Maryland Ave SW, Suite 300 East, Washington, DC 20024-2588. Tel: 1-800-533-8811.

SINGAPORE

Nov 9–14. **First International Congress on Oral Cancer and Jaw Tumors.** Singapore. Contact: Dr N. Ravindranthan, Organizing Secretary, First International Congress on Oral Cancer and Jaw Tumors, Department of Oral Surgery, Faculty of Dentistry, National University of Singapore, Kent Ridge, Singapore 0511. □

Lactaid. Real Milk for Lactose Intolerant Patients.

LACTOSE THE PROBLEM. If your patients drink milk and have indigestion, gas or diarrhea even up to twelve hours afterwards, it may be due to the lactose in milk and dairy foods. One out of five people in the United States may have this problem.

LACTAID THE SOLUTION. Lactaid "Specially Digestible" lactose reduced milk is great tasting lowfat milk with all the protein, calcium and nutritional benefits. With Lactaid, your patients can continue or resume real milk consumption without later discomfort since the lactose is converted into simple sugars which are fully digestible.

DAIRY FOODS ENJOYED ANYTIME—ANYWHERE. Prescribe one to two Lactaid Tablets with any other milk or dairy food, and let your patients enjoy their favorite foods without discomfort. Lactaid Liquid Enzyme can be added to a quart of any milk to hydrolyze up to 99 percent of the lactose if desired.

Lactaid®

For more information on Lactaid products — or to receive your FREE Lactaid Patient Starter Kit including brochures and product samples — call our Lactaid Hotline at:

1-800-257-8650

Monday–Friday, 9 a.m.–4 p.m.

EMERGENCY! MEDICAL CLAIMS OVERLOAD!

The
*EMC*EXPRESS*™
System

TO THE RESCUE!

If claims processing paperwork can get your office all backed up, you need to call in GE Information Services' EMC*EXPRESS rescue squad.

The **EMC*EXPRESS System** is a computerized medical claims system which enables you to deliver medical claims to participating carriers within hours of treating a patient. One phone call is all it takes to have your computer speed all the information needed to process your claims to **multiple** carriers, including Medicare, commercial and private.

Working with the software already in your office, the **EMC*EXPRESS** System helps you:

- Reduce your administrative costs for processing claims
- Reduce the number of claims rejected because of incorrect or incomplete information
- Speed up your claims payment—**and that can put new life into your cash flow.**

The EMC*EXPRESS System and Your Office

The EMC*EXPRESS System accommodates a broad range of practice sizes and a wide variety of office computer systems, from easy-to-use claims entry systems to the most sophisticated practice management systems.

So whatever the size of your office, whatever the magnitude of your paperwork problem, we recommend the **EMC*EXPRESS** System as your treatment of choice.

For more than 20 years, GE Information Services has been helping companies around the world do business better. And now GE has teamed up with authorized system vendors to bring rapid, accurate, and cost-effective electronic medical claims communications to the healthcare field.

To have the EMC* EXPRESS rescue squad come to your aid, call an authorized EMC*EXPRESS system vendor or contact us directly at 1-800-433-3683, extension 7321. We'll send help your way before you can say, "Emergency!"

INFORMATION SERVICES
USA

The
EMC*EXPRESS™
System

PARTICIPATING CARRIERS

Empire Blue Cross and Blue Shield
Medicare B and Private Business Claims
Metropolitan Life Insurance Co.
(Includes Empire NY State Employees)

NEIC ACTIVE PARTICIPANTS

Aetna Life & Casualty
Allstate Life Insurance Co.
Benefit Trust Life
CNA Insurance Companies
Confederation Administration Life Insurance Co.
Confederation Life Insurance Co.
Connecticut Life Insurance Co.
Connecticut General Life Insurance Co. (CIGNA)
Equitable Life Assurance Society of the U.S.
General American Life Insurance Co.
The Great-West Life Assurance Co. of America
Gulf Group Services
The Hartford Insurance Group
John Hancock Mutual Life Insurance Co.
Liberty Life Assurance Co.
Lincoln National Life Insurance Co.
Massachusetts Mutual Life Insurance Co.
Metropolitan Life Insurance Co.
Mutual of Omaha Insurance Co.
New England Mutual Life Insurance Co.
New York Life Insurance Co.
Pacific Mutual Life Insurance Co.
Philadelphia American Life Insurance Co.
Philadelphia Life Insurance Co.
Phoenix Mutual Life Insurance Co.
Pilot Life Insurance Co.
Principal Mutual Financial Group
Provident Life and Accident Insurance Co.
Prudential Insurance Co.
State Mutual Insurance Co.
The Travelers Insurance Co. (includes Railroad Retirement)
Unionmutual Stock Life Insurance Co.

AUTHORIZED SYSTEM VENDORS

Annson Systems, Inc.	312 564-8310
Articulate Publications, Inc.	800 872-2282
Artificial Intelligence, Inc.	206 271-8633
CALYX	800 558-2208
Colwell Systems, Inc.	800 637-1140
Coopervision	800 772-CVIS
EDP Systems, Inc.	214 881-8454
Health America Systems	312 362-3730
LDS, Inc.	913 648-7111
MOS, Inc.	800 323-6671
Physicians' Micro Systems, Inc.	206 441-8490
Physicians' Office Computer	213 603-0555
Prism Data Systems	800 428-2310
ProSource Systems	800 645-5609
Santiago Data Systems, Inc.	800 652-3500
UNIVAIR, Inc.	314 426-1099
Westland Software House, Inc.	818 992-0081

NEW VENDORS AND CARRIERS ADDED REGULARLY. FOR MOST RECENT LISTINGS, CALL 1-800-433-3683.

INFORMATION SERVICES
USA

401 N. WASHINGTON ST., ROCKVILLE, MD 20850
NYSJM1087

SIGNATURE LOANS—$5,000 to $60,000

DOCTORS ONLY. No collateral, simple interest, 6 years to repay. No points, no fees, no prepay penalties. Use for any reason. Residents, D.O.'s, start-up welcome. For appointment and info call:

(NY) NORTHSTAR FUNDING (212) 323-8076
or MEDIVERSAL 1-800-331-4952 Dept. #135

EQUIPMENT LEASING—$10K-$10 MILLION
NORTHSTAR FUNDING (212) 323-8076 Apply by Phone

REAL ESTATE FOR SALE OR RENT—CONT'D

LOOKING FOR A GOOD PROFESSIONAL LOCATION? Set up your office in prestigious North Syosset. Bring the family with you to this large center hall colonial with new country kitchen and baths. Separate entry to office, new circular driveway. Many extras SD#2 walk to all. $379,000. Pat Randazzo, Coldwell Banker, (516) 364-6460.

FOR RENT: Medical Office Kew Gardens, suitable for group or solo practice. Completely furnished and equipped, spacious, Picker x-ray, physical therapy treatment rooms. Separate wing of private house, tree lined street, excellent neighborhood. Retired Orthopedist, phone (718) 849-8844.

DOCTORS OFFICE FOR RENT. Fully furnished and equipped. East 3rd Street, Cortelyou Road Brooklyn, 2 blocks away from Ocean Parkway. No doctor in immediate area. Great future, also apartment above the office available. Call (718) 436-6751.

MISCELLANEOUS

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuarial certification and employee statements of participation—partnership agreements and professionals incorporation—No insurance required—references upon request—Wachstock and Dienstag Attorneys at Law, 122 Cutter Mill Road, Great Neck, NY 11021 (516) 773-3322.

1988 CME CRUISE/CONFERENCES on Medicolegal Issues & Risk Management—Caribbean, Mexico, Alaska, China/Orient, Europe, New England/Canada, Trans Panama Canal, South Pacific. Approved for 24-28 CME Cat. 1 Credits (AMA/PRA) and AAFP prescribed credits. Distinguished lecturers. Excellent Group Rates on Finest Ships. Registration limited. Prescheduled in compliance with IRS requirements. Information: International Conferences, 189 Lodge Ave., Huntington Station, NY 11746. (516) 549-0869.

MISCELLANEOUS—CONT'D

HAVE YOU RECEIVED ANY COMMUNICATION FROM THE OFFICE OF PROFESSIONAL MEDICAL CONDUCT? If affirmative, contact Susan Kaplan, Attorney-at-Law, (212) 877-5998. Practice limited to the crucial legal problems directly affecting your license to practice medicine—with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professions), and as an Assistant District Attorney in Nassau County. Susan Kaplan, Esq., 165 West End Avenue, Suite 27P, New York, N.Y. 10023.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta, GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

ADVICE, COUNSEL & REPRESENTATION of physicians and other health care professionals in licensure and professional conduct matters, audit preparations, reimbursement issues and practice-related litigation. David E. Ruck, Esq., former Chief, Criminal Division, Office of the New York State Special Prosecutor for Medicaid Fraud Control and Alain M. Bourgeois, Esq., former Acting Justice, Supreme Court of the State of New York. Bourgeois & Ruck, 101 Park Avenue, New York, New York 10178. Telephone 212-661-8070.

PORTABLE RADIOLOGY SERVICE: abdominal + pelvic ultrasound, cardiac echos including 2D + m-mode portable x-rays and portable osteoporosis bone densitometry screening. NYC and suburbs. Schedule weekly/bi-weekly in your office or visits to your patient's home. New portable hi-tech equipment. Hi-quality like standard office images, far superior to hospital portable images. No lead shielding needed for x-rays. University Radiology Service, (212) 265-7392 or (718) 339-5363.

PROFESSIONAL MISCONDUCT ATTORNEYS. William L. Wood, Jr., formerly Executive Director of the New York State Office of Professional Discipline and Anthony Z. Scher, formerly Director of Prosecutions. Our recent tenure as chief enforcement officers for the regulation of New York State's one-half million licensed professionals has given us experience which allows us to represent physicians in professional misconduct proceedings, malpractice, license restoration, controlled drug proceedings, insurance company reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practitioners. Wood & Scher, Attorneys at Law, One Chase Road, Scarsdale, New York 10583. Telephone (914) 723-3500.

Added Control For America's Coughs

The expectorant action of guaifenesin plus the Advantage of Codeine!

Robitussin and codeine. Each 5 ml (*1 teaspoonful*) contains: Guaifenesin, USP, 100 mg; Codeine Phosphate, USP, 10 mg (*warning: may be habit forming*); alcohol, 3.5 percent; in a palatable, aromatic syrup

Robitussin A-C®Ⓥ

A·H·ROBINS

Pharmaceutical Division, Richmond, Virginia 23261-6609

©A.H. Robins Company 1985

TIME
to subscribe

Consider a few features of
The Western Journal of Medicine:

- **Epitomes of Progress** — Each month this section highlights the major advances in a different specialty with 15 or 20 brief epitomes by experts in the field.
- **Clinical Investigation** — Five major clinical research societies coordinate important new research findings, emphasizing clinical value.
- **Socioeconomics** — WJM is often the first to predict, examine and evaluate socioeconomic changes, trends.
- **Yearly Special Issue** — Each December's issue is devoted to a topic vital to physicians: clinical nutrition (1979), cost of care (1980), geriatrics (1981), occupational/environmental (1982), cross-cultural medicine (1983), personal health maintenance (1984), high-tech medicine (1985), medical informatics (1986).

NYSJM 10/87

Enclosed is $________ to cover ________ subscriptions.
Subscription renewal ☐ Regular subscription ☐
Foreign subscription ☐

TO: ________________________________

Street ________________________________

City/State/Zip ________________________________

Specialty ________________________________

Gift Card to read "From________________________________"

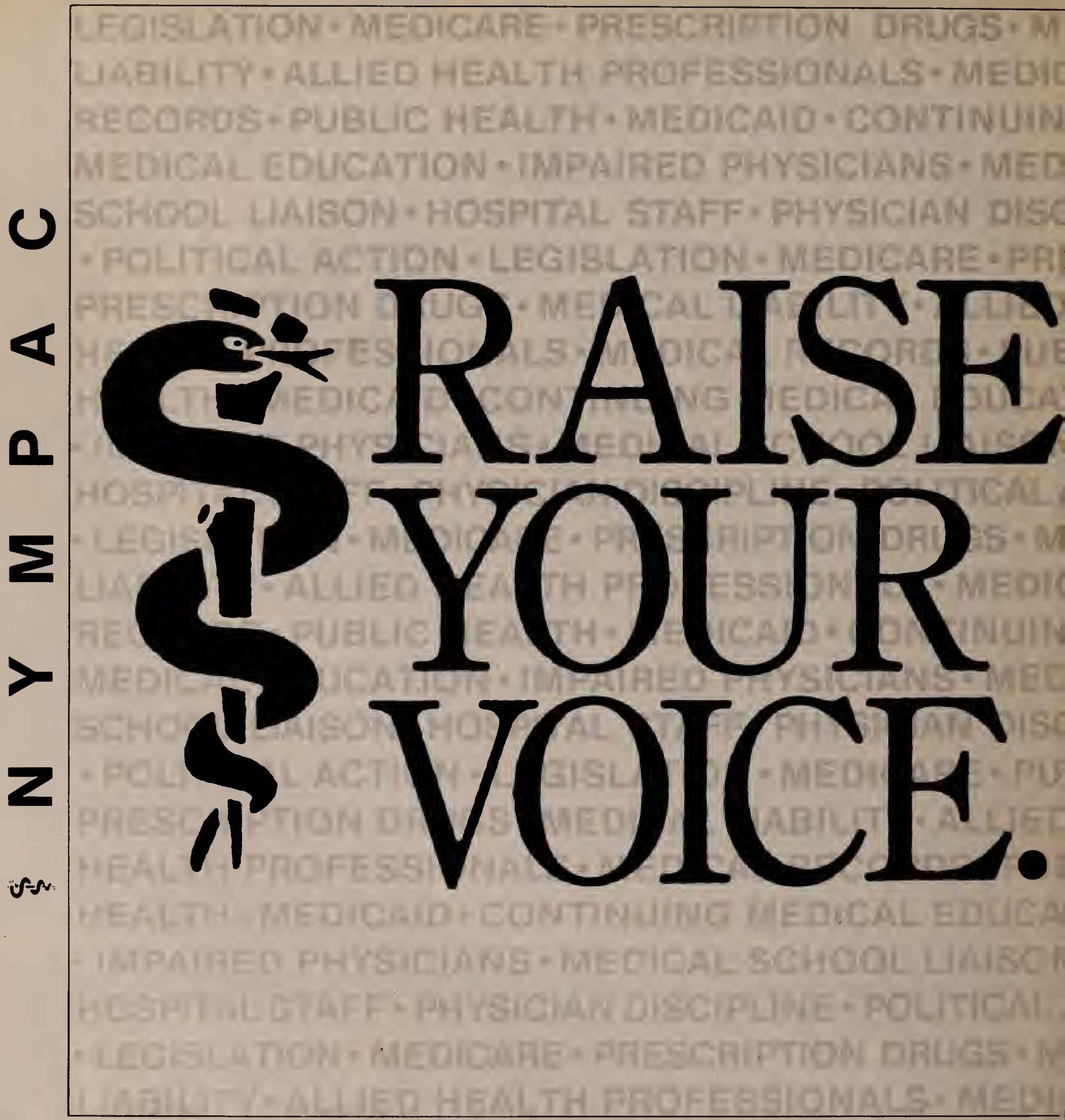
The Western Journal of Medicine

Please mail this coupon with
enclosed payment to:

Circulation Department
P.O. Box 7602, San Francisco,
California 94120-7602

Subscriptions, 12 issues Per Year
U.S. and Canada $30 1 year,
$50 2 years

All other countries $50 1 year,
$90 2 years



NYMPAC
LEGISLATION • MEDICARE • PRESCRIPTION DRUGS • M
LIABILITY • ALLIED HEALTH PROFESSIONALS • MEDIC
RECORDS • PUBLIC HEALTH • MEDICAID • CONTINUIN
MEDICAL EDUCATION • IMPAIRED PHYSICIANS • MED
SCHOOL LIAISON • HOSPITAL STAFF • PHYSICIAN DISC
• POLITICAL ACTION • LEGISLATION • MEDICARE • PR
RAISE
YOUR
VOICE.

New York Medical Political Action Committee
An Arm of the Medical Society of the State of New York
420 Lakeville Road, Lake Success, NY 11042 • 516-488-6100

Political Action for Physician

A Clinical Opportunity for Smoking Intervention

You can play a special role in reaching smokers. Encouraging parents not to smoke can improve the health of the entire family.

Take a few minutes to explain that children of parents who smoke are often more prone to respiratory infections. Tell them that young children of parents who smoke are more likely to become smokers themselves.

The minutes you spend can make a difference now, and in the years ahead.

For a free copy of *Clinical Opportunities for Smoking Intervention: A Guide for the Busy Physician*, complete the form below.

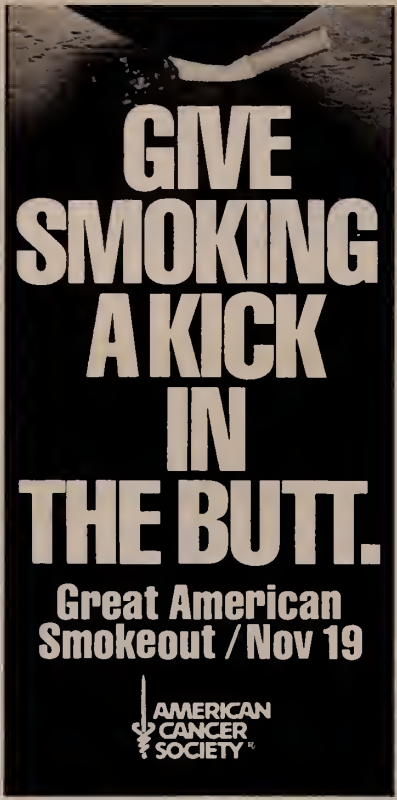
Mail to:
The National Heart, Lung,
and Blood Institute
Smoking Education Program
National Institutes of Health
Building 31, Room 4A 18
Department P-26
Bethesda, MD 20892

Name

Specialty

Address

City


GIVE SMOKING A KICK IN THE BUTT.
Great American Smokeout / Nov 19
AMERICAN CANCER SOCIETY

MISCELLANEOUS—CONT'D

SIGNATURE LOANS—$5,000 to $60,000 doctors only. No collateral, simple interest, 4, 5, or 6 year terms. No points, no fees, no prepay penalties. No use restrictions. Residents, D.O.'s, new practitioners welcome. For application and info call: (NY) Northstar Funding (212) 323-8076 or Mediversal 1-800-331-4952 Dept. #135.

PROFESSIONAL MISCONDUCT ADVOCACY with dignity and concern. George Weinbaum, Esq. former deputy director of the New York State Attorney General's office for Medicaid Fraud Control and also former Director of Medicare and private insurance investigations for Empire Blue Cross/Blue Shield is pleased to announce the opening of his office for the practice of health law. Attention is devoted to misconduct avoidance, disciplinary proceedings, reimbursement maximization, controlled drug issues and medical staff representation among other physician needs. George Weinbaum, Esq., 3 Barker Avenue, White Plains, New York 10601. Telephone (914) 686-9310 office hours, (212) 621-7776-24 hours.

LEASE YOUR NEXT AUTOMOBILE—from the people who know their profession and yours. Just as your patients seek the advice of a professional, so should you! All leases are custom tailored to your needs. A wide variety of services are available. Our rates are highly competitive. We can make the acquisition of your next vehicle inexpensive, swift and easy. All makes and models leased. Madison Leasing Corp., 1983 Marcus Ave., Lake Success, NY 11042, (516) 775-5200.

EB TRANSCRIPTION SPECIALISTS offering expert work with professional service to fit your needs. All medical specialties. Letters and correspondence, patients charts and manuscript typing. Fast turn around time, free pick up and delivery in the New York area and competitive rates. For additional information call Emily at (718) 849-1987.

Doctor . .

Do you, or a physician you know, have a problem with alcohol, drugs, or emotional illness?

If so, please contact the Medical Society of the State of New York's Committee for Physicians' Health, whose function is to assist, *confidentially.*

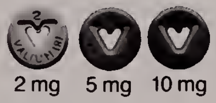
Telephone: 516-488-7777.

Index to Advertisers

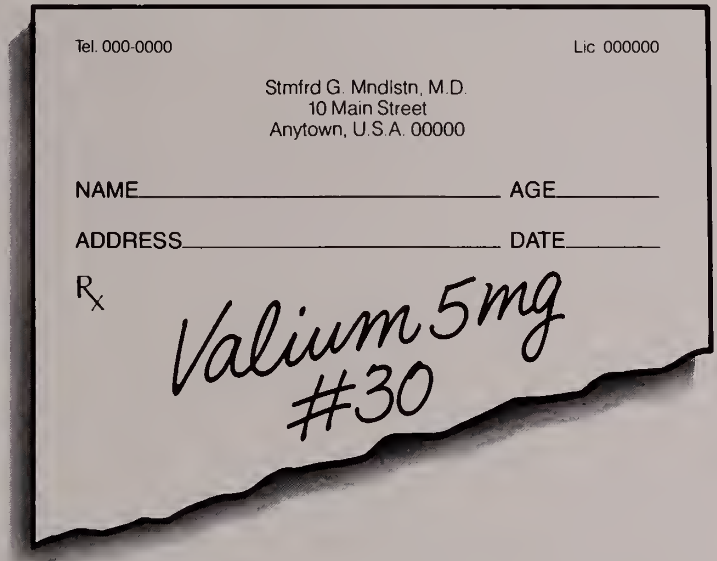
AMA Advisers, Inc.	5A
St. Clare's Hospital	1A
Classified Advertising	6A, 8A, 19A, 24A
Family Practice Recertification Magazine	16A
General Electric Information Services	18A, 19A
Knoll Pharmaceuticals	3A, 4A
Lactaid	17A
Eli Lilly and Company	10A
McNeil Laboratories	15A
Milstein Properties Corp.	8A
A. H. Robins Company	7A, 20A
Roche Laboratories	3rd & 4th Cover
Sarantos and Company, Inc.	8A
Upjohn Company	2nd Cover

Do Not Substitute

VALIUM® Ⓘⓥ
brand of
diazepam/Roche



The One You Know Best



Tel. 000-0000 Lic 000000

Stmfrd G. Mndlstn, M.D.
10 Main Street
Anytown, U.S.A. 00000

NAME_______________ AGE______

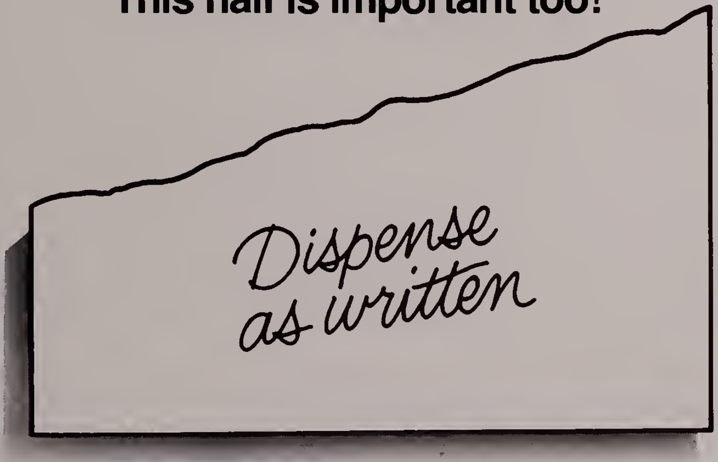
ADDRESS_______________ DATE______

℞

Valium 5mg
#30

Are you writing only half a prescription for it?

This half is important too!



Dispense
as written

Be sure to write a complete prescription.

Specify "Dispense as written."

ROCHE®
Roche Products Inc.
Manati, Puerto Rico 00701

Copyright © 1987 by Roche Products Inc.
All rights reserved.

State flag of New York

Flag It.

To complete your prescription,
be sure to specify
"Dispense As Written."
This "flags" both pharmacist and patient
that you want the *brand* to be dispensed.
And it protects your decision.

VALIUM®
brand of
diazepam/Roche Ⓘⓥ

scored tablets

The one you know best.

The cut out "V" design is a registered trademark
of Roche Products Inc.

ROCHE®

STACKS

NEW YORK STATE JOURNAL OF MEDICINE

DECEMBER 1987 • Volume 87, Number 12

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA
DEC 21 1987
M.D.S.

Contents

COMMENTARIES

Sleep deprivation in physician training 637
EMANUEL E. GARCIA, MD

Renal adenocarcinoma 638
BARRY N. NOCKS, MD

Acoustic neuromas: Yesterday, today, and tomorrow 640
STEPHEN G. HARNER, MD

IN MEMORIAM

Henry Irwin Fineberg, MD 642

RESEARCH PAPERS

Cancer screening and prevention: Knowledge, attitudes, and practices of New York City physicians 643
DONALD H. GEMSON, MD, MPH; JACK ELINSON, PhD

Deprenyl versus placebo in Parkinson disease: A double-blind study 646
ABRAHAM N. LIEBERMAN, MD; GOVINDAN GOPINATHAN, MD; ANDREAS NEOPHYTIDES, MD; SON HOO FOO, MD

REVIEW ARTICLE

Lacunes: The pervasive strokes 650
EMILIO D. SORIA, MD; EDWARD J. FINE, MD; MARGARET W. PAROSKI, MD

HISTORY

The medical writings of Moses Maimonides 656
FRED ROSNER, MD

CASE REPORTS

Amebic liver abscess without obvious risk factors 662
ASHOK PATEL, MD; SUDHAKAR A. REDDY, MD; FRANCIS U. STEINHEBER, MD; B. KHURANA, MD

Renal adenocarcinoma in the pediatric age group 663
THOMAS KAUFMANN, MD, NORWIN H. BECKER, MD

Childhood acoustic neuroma 665
KENNETH F. MATTUCCI, MD; WALTER M. GLASS, MD; MICHAEL SETZEN, MD; WILLIAM LEVIN, MD

LETTERS TO THE EDITOR

Limb salvage with sarcomas 667
MUHAMMAD ABU HENA, MD

Koplik spots and echo 9 virus 667
DAVID ANNUNZIATO, MD

Measles rash 667
HELMUT A. MIKK, MD

Strategic planning for medical societies 667
RANDALL D. BLOOMFIELD, MD

ESSAY REVIEW

Doctors in Science and Society. Essays of a Clinical Scientist 669
PASCAL JAMES IMPERATO, MD

LEADS FROM EPIDEMIOLOGY NOTES 671

BOOK REVIEWS 674

NEWS BRIEFS 678

MEDICAL SOCIETY NEWS 680

OBITUARIES 682

1987 INDEX 685

GUIDELINES FOR AUTHORS 698

MEDICAL MEETINGS AND LECTURES 4A

**Annual CME Assembly, April 22–24, 1988, New York Hilton, New York City
(See January 1988 issue for Preliminary Program)**

Motrin® 800 TABLETS mg

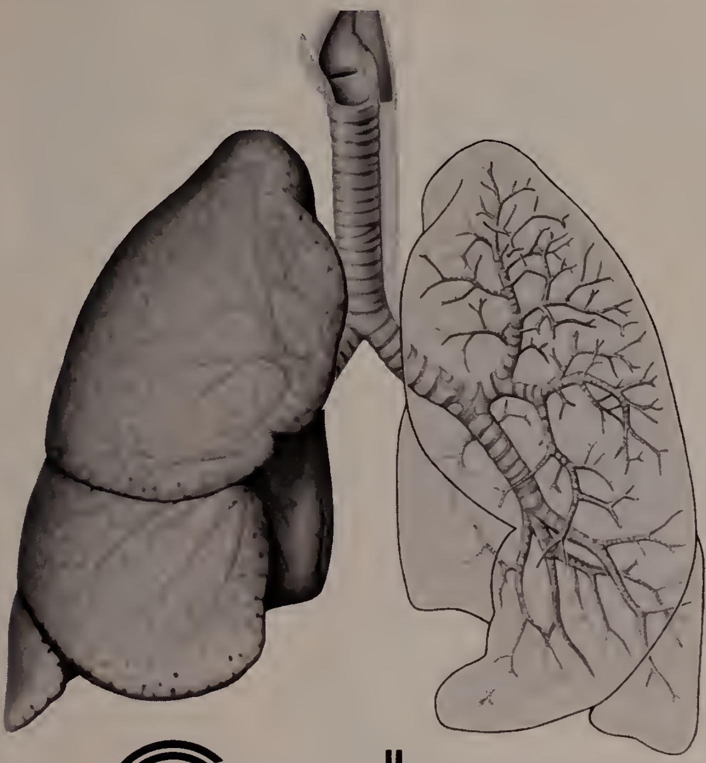
ibuprofen

Extra strength
Convenience
Economy

Upjohn
A Century
of Caring
1886·1986

©1986 The Upjohn Company

Consider the causative organisms...



Ceclor® cefaclor

250-mg Pulvules® t.i.d. offers effectiveness against the major causes of bacterial bronchitis

Haemophilus influenzae and *Streptococcus pneumoniae*
(ampicillin-susceptible and ampicillin-resistant)

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

Ceclor® (cefaclor)

Summary. Consult the package literature for prescribing information.

Indication: Lower respiratory infections, including pneumonia, caused by *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Streptococcus pyogenes* (group A β-hemolytic streptococci).

Contraindication:
Known allergy to cephalosporins.

Warnings:
CECLOR SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY. POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS

Administer cautiously to allergic patients.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea. Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.

Precautions:
- Discontinue Ceclor in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of nonsusceptible organisms.
- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- Ceclor should be administered with caution in the presence of markedly impaired renal function. Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk. Exercise caution in prescribing for these patients.

Adverse Reactions: (percentage of patients)
Therapy-related adverse reactions are uncommon. Those reported include:
- Gastrointestinal (mostly diarrhea): 2.5%.
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
- Hypersensitivity reactions (including morbilliform eruptions, pruritus, urticaria, and serum-sickness-like reactions that have included erythema multiforme [rarely, Stevens-Johnson syndrome] or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever): 1.5%; usually subside within a few days after cessation of therapy. Serum-sickness-like reactions have been reported more frequently in children than in adults and have usually occurred during or following a second course of therapy with Ceclor. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.
- Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.
- As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely.
- Rarely, reversible hyperactivity, nervousness, insomnia, confusion, hypertonia, dizziness, and somnolence have been reported.
- Other: eosinophilia, 2%; genital pruritus or vaginitis, less than 1%; and, rarely, thrombocytopenia.

Abnormalities in laboratory results of uncertain etiology
- Slight elevations in hepatic enzymes.
- Transient fluctuations in leukocyte count (especially in infants and children).
- Abnormal urinalysis; elevations in BUN or serum creatinine.
- Positive direct Coombs' test.
- False-positive tests for urinary glucose with Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (glucose enzymatic test strip, Lilly). [061787L]

PA 0709 AMP

©1987, ELI LILLY AND COMPANY CR-5005-8-849318

Lilly

Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285.

Eli Lilly Industries, Inc
Carolina, Puerto Rico 00630

NEW YORK STATE JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD	JOHN T. PRIOR, MD
ELIZA H. CALDWELL, MD	GITA S. SINGH*
JOSEPH F. MURATORE, MD	STANFORD WESSLER, MD

*Medical student

Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Consulting Editor and Book Review Editor	RICHARD B. BIRRER, MD
Consulting Editor	NAOMI R. BLUESTONE, MD, MPH
Consulting Editor	CARL POCHEDLY, MD
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Managing Editor	CAROL L. MOORE
Advertising Director	ROBERT MILLER
Consulting Medical Writer	VICKI GLASER
Editorial Assistant	MILDRED J. AREMANN
Secretary	ELIZABETH J. SOMERS
Librarian	ELLA ABNEY
Assistant Librarian	ELEANOR BURNS

ASSOCIATE EDITORIAL BOARD 1987

MICHAEL E. BERLOW, MD	FLORENCE KAVALER, MD
RANDALL BLOOMFIELD, MD	ROBERT LANDESMAN, MD
ROBERT D. BRANDSTETTER, MD	JAMES M. MORRISSEY, ESQ
JOHN S. DAVIS, MD	STEPHEN NORDLICHT, MD
CHARLES D. GERSON, MD	JOSEPH SCHLUGER, MD
MYLES S. GOMBERT, MD	BJORN THORBJARNARSON, MD
ALFRED P. INGEGNO, MD	RODRIGO E. URIZAR, MD

NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. United Media Associates, Inc, 16 Bruce Park Ave, Greenwich, CT 06830. Telephone: (203) 661-9702.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

SAMUEL M. GELFAND, MD,	*President*
JOHN A. FINKBEINER, MD,	*Past-President*
CHARLES D. SHERMAN, JR, MD,	*President-Elect*
DAVID M. BENFORD, MD,	*Vice-President*
JOHN H. CARTER, MD,	*Secretary*
GEORGE LIM, MD,	*Assistant Secretary*
MORTON KURTZ, MD,	*Treasurer*
ROBERT A. MAYERS, MD,	*Assistant Treasurer*
CHARLES N. ASWAD, MD,	*Speaker*
SEYMOUR R. STALL, MD,	*Vice-Speaker*

Councilors

Term Expires 1988

JAMES H. COSGRIFF, JR, MD,	Erie
RICHARD A. HUGHES, MD,	Warren
ANTONIO F. LASORTE, MD,	Broome
SIDNEY MISHKIN, MD,	Nassau

Term Expires 1989

ROBERT E. FEAR, MD,	Suffolk
STANLEY L. GROSSMAN, MD,	Orange
THOMAS D. PEMRICK, MD,	Rensselaer
RALPH E. SCHLOSSMAN, MD,	Queens

Term Expires 1990

STUART I. ORSHER, MD, (*elected to serve until 1988*)	New York
DUANE M. CADY, MD,	Onondaga
WILLIAM A. DOLAN, MD,	Monroe
ROBERT E. GORDON, MD,	Kings

Resident Councilor (*representing the resident physician membership*)

KATHLEEN E. SQUIRES, MD,	New York

Student Councilor (*representing the medical student membership*)

MICHAEL PACIOREK,	Onondaga

Trustees

RICHARD D. EBERLE, MD, (*Chairman*)	Onondaga
EDGAR P. BERRY, MD,	New York
JAMES M. FLANAGAN, MD,	Wayne
ALLISON B. LANDOLT, MD,	Westchester
DANIEL F. O'KEEFFE, MD,	Warren
BERNARD J. PISANI, MD,	New York
VICTOR J. TOFANY, MD,	Monroe

Executive Vice-President	DONALD F. FOY
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	GEORGE J. LAWRENCE, JR, MD
Director, Division of Policy Coordination	IRMA A. ERICKSON
Director, Division of Scientific Publications	PASCAL JAMES IMPERATO, MD
Director, Division of Communications	EDWARD A. HYNES
Director, Division of Computer Services	IVAN H. NAJMAN
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Governmental Affairs	GERARD L. CONWAY, ESQ
General Counsel	DONALD R. MOY, JD
Director, Division of Management and Meeting Services	ROBERT MILLER
Director, Division of Membership Support Services	EUNICE M. SKELLY
Director, Division of Scientific and Educational Activities	PETER B. FARNSWORTH, MD
Director, Division of Socio-Medical Economics	MARIO V. MENGHINI
Medical Director, Committee for Physicians' Health	BARTON A. HARRIS, MD
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York.

Copyright © 1987, Medical Society of the State of New York. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $30.00 ($35.00 for all other countries). The single copy rate is $5.00 Domestic/Canada, and $6.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.

A better alternative for hypertensives who are going bananas...

Spare your patients the extra cost—in calories, sodium and dollars.

Spare your patients the rigors of dietary K+ supplementation.

DYAZIDE®

25 mg Hydrochlorothiazide/50 mg Triamterene/SKF

Effective antihypertensive* therapy...without the bananas

DAW
'DYAZIDE' AS WRITTEN.

Before prescribing, see complete prescribing information in SK&F CO. literature or PDR. The following is a brief summary.

* **WARNING**

This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

Contraindications: Concomitant use with other potassium-sparing agents such as spironolactone or amiloride. Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: The bioavailability of the hydrochlorothiazide component of 'Dyazide' is about 50% of the bioavailability of the single entity. Theoretically, a patient transferred from the single entities of triamterene and hydrochlorothiazide may show an increase in blood pressure or fluid retention. Similarly, it is also possible that the lesser hydrochlorothiazide bioavailability could lead to increased serum potassium levels. However, extensive clinical experience with 'Dyazide' suggests that these conditions have not been commonly observed in clinical practice. Angiotensin-converting enzyme (ACE) inhibitors can elevate serum potassium; use with caution with 'Dyazide'. Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids, and during concurrent use with amphotericin B or corticosteroids or corticotropin[ACTH]). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Cumulative effects of the drug may develop in patients with impaired renal function. Thiazides should be used with caution in patients with impaired hepatic function. They can precipitate coma in patients with severe liver disease. Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic and hemolytic anemia have been reported with thiazides. Thiazides may cause manifestation of latent diabetes mellitus. The effects of oral anticoagulants may be decreased when used concurrently with hydrochlorothiazide; dosage adjustments may be necessary. Clinically insignificant reductions in arterial responsiveness to norepinephrine have been reported. Thiazides have also been shown to increase the paralyzing effect of nondepolarizing muscle relaxants such as tubocurarine. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. Triamterene has been found in renal stones in association with the other usual calculus components. Therefore, 'Dyazide' should be used with caution in patients with histories of stone formation. A few occurrences of acute renal failure have been reported in patients on 'Dyazide' when treated with indomethacin. Therefore, caution is advised in administering nonsteroidal anti-inflammatory agents with 'Dyazide'. The following may occur. transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia is uncommon with 'Dyazide', but should it develop, corrective measures should be taken such as potassium supplementation or increased dietary intake of potassium-rich foods. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Concurrent use with chlorpropamide may increase the risk of severe hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function. Thiazides may add to or potentiate the action of other antihypertensive drugs. Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances; postural hypotension (may be aggravated by alcohol, barbiturates, or narcotics). Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and respiratory distress including pneumonitis and pulmonary edema, transient blurred vision, sialadenitis, and vertigo have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components. Rare incidents of acute interstitial nephritis have been reported. Impotence has been reported in a few patients on 'Dyazide', although a causal relationship has not been established.

Supplied: 'Dyazide' is supplied as a red and white capsule, in bottles of 1000 capsules; Single Unit Packages (unit-dose) of 100 (intended for institutional use only); in Patient-Pak™ unit-of-use bottles of 100.

BRS-DZ:L45

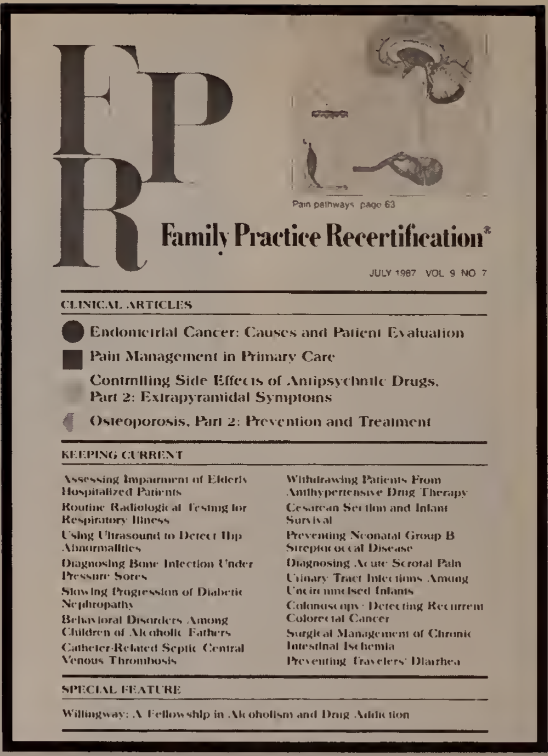
** Not for initial therapy. See brief summary.*

a product of
SK&F CO.
Cidra, P.R. 00639

SK&F Co., 1987

The complete journal for family practice physicians

- Reaches 79,000 family physicians monthly
- Presents the most commonly seen patient problems in family practice
- Written by physicians for physicians
- The most current clinical updates in:
  - Cardiology
  - Diabetes
  - Pediatrics
  - Ob/Gyn
  - Psychiatry
  - Gastroenterology
- Provides 20 hours of CME Category 1 Credit


FPR

Pain pathways page 63

Family Practice Recertification®

JULY 1987 VOL 9 NO 7

CLINICAL ARTICLES

● Endometrial Cancer: Causes and Patient Evaluation

■ Pain Management in Primary Care

Controlling Side Effects of Antipsychotic Drugs, Part 2: Extrapyramidal Symptoms

Osteoporosis, Part 2: Prevention and Treatment

KEEPING CURRENT

Assessing Impairment of Elderly Hospitalized Patients

Routine Radiological Testing for Respiratory Illness

Using Ultrasound to Detect Hip Abnormalities

Diagnosing Bone Infection Under Pressure Sores

Slowing Progression of Diabetic Nephropathy

Behavioral Disorders Among Children of Alcoholic Fathers

Catheter-Related Septic Central Venous Thrombosis

Withdrawing Patients From Antihypertensive Drug Therapy

Cesarean Section and Infant Survival

Preventing Neonatal Group B Streptococcal Disease

Diagnosing Acute Scrotal Pain

Urinary Tract Infections Among Uncircumcised Infants

Colonoscopy: Detecting Recurrent Colorectal Cancer

Surgical Management of Chronic Intestinal Ischemia

Preventing Travelers' Diarrhea

SPECIAL FEATURE

Willingway: A Fellowship in Alcoholism and Drug Addiction

PRACTICAL ■ CLINICAL ■ EDUCATIONAL ■ CURRENT

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

JANUARY 1988

AROUND THE STATE

MANHATTAN

Jan 11–13. **Molecular Basis of the Immune Response.** Vista International Hotel. Contact: Conference Director, New York Academy of Sciences, 2 East 63rd St, New York, NY 10021.

NASSAU

Jan 23–24. **Transitional Cell Carcinoma of the Urinary Tract.** 16 Cat 1 Credits. Long Island Jewish Medical Center, New Hyde Park. Contact: Ann J. Boehme, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

AROUND THE NATION

ARIZONA

Jan 12–15. **Third International Conference on Prevention of Human Cancer: Chemoprevention.** The Arizona Cancer Center, Tucson. Contact: Mary Humphrey, Conference Coordinator, Arizona Cancer Center, Arizona Health Sciences Center, Tucson, AZ 85724.

Jan 27–30. **Clinical Recognition and Management of Heart Disease—1988.** University Medical Center, Tucson. Contact: American College of Physicians, PO Box 7777-R-0510, Philadelphia, PA 19175.

CALIFORNIA

Jan 14–17. **Advanced Seminars in Diagnostic Imaging.** 20 Cat 1 Credits. Ritz-Carlton Hotel, Laguna Niguel. *Jan 18–21.* **Chest Imaging 1988.** 22 Cat 1 Credits. Hotel del Coronado, Coronado. *Jan 21–23.* **Practical Approach to Duplex Imaging.** Hotel Inter-Continental, San Diego. *Jan 25–28.* **Annual Update of Nuclear Radiology.** Hotel del Coronado, Coronado. Contact: Dawne Ryals, Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

Jan 16–18. **The Fifth Annual Mohs Surgery Conference of The Skin Cancer Foundation.** San Diego Princess Resort, San Diego. Contact: Mitzi Moulds, Executive Director, The Skin Cancer Foundation, 245 Fifth Ave, Suite 2402, New York, NY 10016. Tel: (212) 725-5176.

Jan 19–23. **Intensive Course in Geriatric Medicine and Board Review.** Beverly Hilton Hotel, Beverly Hills. Contact: American College of Physicians, 4200 Pine St, Philadelphia, PA 19104.

(*continued on p 15A*)

88
NEW YORK

American College of Physicians presents 69th Annual Session March 3–6, 1988 Jacob K. Javits Convention Center

Medicine's foremost educational opportunity in the nation's most exciting city

☐ More than 200 presentations for a curriculum suited to your needs
☐ Critical findings in Molecular Biology and Clinical Medicine in six State of the Art lectures
☐ Pre-Session courses on critical care, geriatrics and infectious diseases
☐ Clinical controversies featured in 12 panel discussions
☐ Analyses that confront the social, ethical and environmental issues surrounding today's dilemmas
☐ Rewarding activities planned especially for family and guests

Request Your 1988 Scientific Program Guide

The Scientific Program Guide—with schedules, course and faculty lists, registration and housing information, and the family and guest program—enables you to plan the most valuable, personalized Session possible. Because many popular programs fill up quickly, you'll want to schedule your curriculum well in advance. Reserve your copy now by filling out and returning the request form.

YES, mail me the '88 Scientific Program Guide **NY12**

PLEASE PRINT
NAME
ADDRESS
CITY/STATE/ZIP
Telephone toll-free: (800) 523-1546 ext. 3675
In Pennsylvania: (215) 243-1200 ext. 3675
☐ ACP Member ACP# ______
☐ Nonmember

American College of Physicians
4200 Pine Street
Philadelphia, PA 19104

Classified Advertising

PHYSICIANS WANTED

EMERGENCY MEDICINE POSITIONS—Full/part time emergency medicine physicians sought by multi state professional association for openings in metropolitan NY, PA, MD, DC, FL, New England and throughout U.S. Contact or send CV to Liberty Healthcare Corporation, 399 Market Street, Suite 400, Philadelphia, PA 19106, (215) 592-7400 or outside Pa (800) 331-7122.

ARIZONA-BASED PHYSICIAN RECRUITMENT firm has quality opportunities coast to coast. Available positions in most primary care and surgical specialties to include OB/GYN, Orthopedics, ER, and ENT. "Quality Physicians for Quality Clients since 1972." Call 602-990-8080; or send CV to: Mitchell & Associates, Inc., PO Box 1804, Scottsdale, AZ 85252.

NEW YORK, Western—Seeking primary care trained physicians for full time Emergency Department positions. Moderate volume. Attractive hourly compensation, plus malpractice insurance. Director position available. Contact: Emergency Consultants, Inc., 2240 S. Airport Rd., Room 42, Traverse City, MI 49684; 1-800-253-1795 or in Michigan 1-800-632-3496.

NEW YORK, Buffalo—Seeking primary care trained physicians for full-time emergency department positions. University affiliated hospital. Moderate volume. E.D. Attractive hourly compensation plus malpractice insurance. Contact: Emergency Consultants, Inc., 2240 S. Airport Rd., Room 42, Traverse City, MI 49684; 1-800-253-1795 or in Michigan 1-800-632-3496.

LOCUM TENENS PHYSICIAN—Join a comprehensive physician support service with a major medical center in south central Montana. Locum physicians provide primary care coverage (excluding routine OB) for physicians in rural Montana and Wyoming. Assignments vary in length. Reimbursement for expenses, malpractice, health insurance, CME. Call Locum Tenens Coordinator, 1-800-325-1774, or send C.V. to 1500 Poly Drive, Suite 103, Billings, MT 59102.

MULTI-SPECIALTY GROUP PRACTICE has an excellent opportunity for a General Internist. Located in the Southern Tier of New York State, this area provides many cultural & recreational activities and is an excellent family oriented community. Please submit C.V. to: Allen D. Alt, M.D., Chenango Bridge Road, Binghamton, New York 13901 or call collect (607) 648-4101.

FAMILY PRACTICE OPPORTUNITY—Rural hospital based practice seeks to add board certified/eligible family practitioner to established group presently composed of Pediatrician, Ob-Gyn & General Surgeon. Salary negotiable, excellent fringe benefits. Beautiful area. Contact Dr. T. Lavell, Delaware Valley Hospital, Walton, NY 13856, (607) 865-6526.

PHYSICIANS WANTED—CONT'D

LOCUM TENENS positions available in all specialties throughout the country. Work one to fifty-two weeks while you travel (expenses paid) and enjoy an excellent guaranteed income. Malpractice insurance, housing & transportation provided. Contact: Locum Medical Group, 30100 Chagrin Blvd., Cleveland, Ohio 44124 or call (800) 752-5515.

ALBANY, NY—OFFICE SPACE TO SHARE with internist and surgeon. Located close to I-90 and Northway 87, facing the hospital across the street. Free parking. (518) 465-6068.

BUSY INTERNIST Pompano Beach, Florida, seeking associate. Leading to full partnership in short period of time Call collect (305) 941-5100.

GENERAL INTERNIST—UPSTATE NEW YORK. Busy Internist seeking a board certified/eligible internist to join him. Excellent fringe benefits, group coverage, excellent salary with opportunity to become partner. Please reply Dept. 452 c/o NYSJM.

PRACTICES AVAILABLE

FOR SALE-PED-PED ALLERGY—well established office in Nassau County, New York. B.E. or B.C. pediatrician with interest or boards in allergy. Will introduce and remain as long as needed. Also arrange hospital appointments. Send resume or write for appointment. Reply Dept. 543 c/o NYSJM.

PRACTICES WANTED

PRACTICE APPRAISAL AND BROKERAGE. Don't just close the door! Your practice may be able to be sold to a physician anxious to follow you. Our professional staff can provide you with a realistic appraisal and assistance in the transfer of your practice. Call: Physicians International, Practice Appraisal and Brokerage Division, 4-NY Vermont Street, Buffalo, NY 14213, (716) 884-3700.

EQUIPMENT

50% OFF PREVIOUSLY OWNED MEDICAL—Laboratory, x-ray, ultrasound equipment. Buy, Sell, Broker, Repair. Appraisals by certified surgical consultants. Medical Equipment Resale & Repair, 24026 Haggerty Road, Farmington Hills, MI 48018. 1-800-247-5826. (313) 477-6880.

MEDICAL EQUIPMENT: Hewlett-Packard EKG page writer. Digitek and Seralyzer Chemistry machines, x-ray cassettes and hangers. Coulter white and red cell counter. All at 50 to 60% reduction. Cambridge EKG machine. All in good condition. Call: (718) 332-4654 or (718) 896-0373.

LIBRIUM®
chlordiazepoxide HCl/Roche Ⓒ̸IV
5-mg, 10-mg, 25-mg capsules
Before prescribing, please consult complete product information, a summary of which follows:

Indications: Management of anxiety disorders; short-term relief of anxiety symptoms, acute alcohol withdrawal symptoms, preoperative apprehension and anxiety. Usually not required for anxiety or tension associated with stress of everyday life. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended.

Contraindications: Known hypersensitivity to the drug.

Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage. Withdrawal symptoms (including convulsions) reported after abrupt cessation of extended use of excessive doses are similar to those seen with barbiturates. Milder symptoms reported infrequently when continuous therapy is abruptly ended. Avoid abrupt discontinuation; gradually taper dosage.

Usage in Pregnancy: **Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically. Due to isolated reports of exacerbation, use with caution in patients with porphyria.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral—Adults.* Mild and moderate anxiety disorders and symptoms, 5 or 10 mg *t.i.d.* or *q.i.d.*; severe states, 20 or 25 mg *t.i.d.* or *q.i.d.* *Geriatric patients:* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)

Supplied: Librium® (chlordiazepoxide HCl/Roche) *Capsules,* 5 mg, 10 mg and 25 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in boxes of 4 reverse-numbered cards of 25, and in boxes containing 10 strips of 10. Libritabs® (chlordiazepoxide/Roche) *Tablets,* 5 mg and 10 mg—bottles of 100 and 500; 25 mg—bottles of 100. With respect to clinical activity, capsules and tablets are indistinguishable.

ROCHE
Roche Products Inc.
Manati, Puerto Rico 00701

Do not substitute

Librium®

brand of

chlordiazepoxide HCl/Roche Ⓒⅳ

Nobody does it better.

ROCHE®

Copyright © 1986 by Roche Products Inc. All rights reserved.

Please see adjacent page for summary of product information.

NEUROIMAGING UPDATE 1988

Paradise Island, Bahamas—Jan. 31, 1988-Feb. 5, 1988

MUHLENBERG HOSPITAL
PLAINFIELD, NJ 07061
Dept. of Radiology and
Medical Education
A Regional Teaching Hospital
Affiliated with UMDNJ-Rutgers
Medical School

INTENDED AUDIENCE

This conference is designed for radiologists, neurosurgeons and other physicians with interests in various diagnostic modalities.

COURSE DESCRIPTION & OBJECTIVES

The program will provide comprehensive and intensive review of the basic principles, practical application and analysis of the current imaging techniques encompassing computed tomography, digital subtraction angiography, and magnetic resonance imaging. Emphasis will be on the very exciting new technology MRI which will be highlighted throughout the course.

In addition, daily sessions for unknown cases of interest and free paper presentations will be conducted with active participation of the faculty and registrants together. All of the registrants will be encouraged to submit interesting proven cases or free papers.

The distinguished faculty members will address interdisciplinary approaches in the management of intracranial and intraspinal degrangements with new insights and perspectives on recent developments in neurodiagnostic modalities.

Telephone Now for Course Registration, Hotel & Travel Arrangements

ACCREDITATION

Muhlenberg Regional Medical Center is accredited by the Accreditation Council for Continuing Medical Education through the Medical Society of New Jersey and sponsors continuing medical education for physicians. The course "Neuroimaging Update, 1988," February 1-5 meets the criteria for 20 credit hours in Category 1 of the Physicians Recognition Award of the American Medical Association.

TOPICS OF PRESENTATION

Basic Physics of MRI, MR Flow Imaging, Reduction of Motion-Induced Artefacts in Neuro-MR Scanning, PACS, Posterior Fossa and Brainstem Lesions, MRI of Arterio-Venous Malformation, Understanding Cerebral Hemorrhage with MRI, CT and MRI of Stroke, Vascular Disease, Extra-Axial Brain Tumors, Intra-Axial Brain Tumors, CT and MRI of Cerebral Trauma, Sellar and Parasellar Abnormalities, MRI of Movement Disorders, Intracranial Infectious Disease, Imaging of Myelopathic Patient, CT and MRI of Spinal Disc Disease, Radiologic Evaluation of Acute and Chronic Spinal Cord Injury, Radiculopathy of Non-Disc Disease Origin, CT and MRI of Postoperative Spine, Clinical Aspect of Dementia, Multiple Sclerosis—Clinical Diagnosis, The Painful Red Eyes, Occular Ischemia in Carotid Artery Disease, Diagnosis and Treatment of Extracranial Carotid Aneurysm, Management of Progressive Cervical Myelopathy

Neuroimaging Update '88
c/o PERSPECTIVE TRAVEL, INC.
40 Stirling Road
Watchung, N.J. 07060
(201) 756-6600

DISTINGUISHED FACULTY

S. Howard Lee, MD, Course director

Rosalie Burns, MD, Professor & Chairperson—Dept. of Neurology, Medical College of Pa., Philadelphia, Pa.

Roger Countee, MD, Clinical Professor of Neurosurgery, Univ. of Medicine & Dentistry of N.J.—R.W. Johnson Med. School

Kenneth R. Davis, MD, Professor and Chief of Radiology, Harvard Medical School, Boston, Ma.

Sadek Hilal, MD, Professor of Radiology, Columbia Presbyterial College of Medicine, Chief of Neuroradiology—Neurological Institute of New York

Herbert I. Goldberg, MD, Professor of Radiology, University of Pennsylvania School of Medicine, Philadelphia, Pa.

Seungho Howard Lee, MD, Clinical Professor of Radiology, University of Medicine & Dentistry, R. W. Johnson Medical School of N.J.

Robert M. Quencer, MD, Professor of Radiology, University of Miami School of Medicine, Miami, Fl.

Krishina C.V.G. Rao, MD, Clinical Professor of Radiology, University of Maryland, Chairman of Department of Radiology, Prince George Hospital, Cheverly , MD.

Theodore Villafana, PhD, Professor of Radiology, Dept. of Diagnostic Imaging, Temple University Medical School, Philadelphia, Pa.

COURSE REGISTRATION

Physicians in Practice	**$475.00**
Physicians in Training	**$300.00**

In New York City, for the Newest and Finest Medical Space, Remember:

THE 4 ESSENTIAL REQUIREMENTS

1. LOCATION!
Liberty Residences at Battery Park City

2. LOCATION!
Windsor Pavilion
(3rd Avenue & 32nd Street)

3. LOCATION!
205 East 64th Street (3rd Avenue)

AND

4. LOCATION!
Normandie Pavilion
(3rd Avenue & 95th Street)

For Further Information Contact Lester Schwalb
(212) 708-0846
Milstein Properties Corp.

M.D.s: WRITING TROUBLE?

THE COMPETITION TO GET PUBLISHED IS **TIGHT** • **THE KEY:** EDITORS AT THE TOP JOURNALS WANT A CLEAR, PRECISE, WELL-WRITTEN *AS WELL AS* CLINICALLY CREDIBLE PAPER!

CONTACT

MICHAEL L. FRIEDMANN
PHYSICIANS' AUTHOR'S EDITOR
(201) 461-6125

PRIVATE CONSULTATIONS FOR EDIT, REWRITE, ORGANIZATION, STYLE • CASE REPORTS, REVIEW ARTICLES, ORIGINAL RESEARCH, GRANT PROPOSALS, ETC.

NY-NJ-CONN METRO

COURTESY DAVID S. ARSENAULT

Medical Society of the State of New York

ANNUAL CME ASSEMBLY

Newer Approaches to Habit Abuse & Dependency

April 22–24, 1988

and the

Annual Meeting of the House of Delegates

April 21–24, 1988

The New York Hilton
Avenue of the Americas at 53rd/54th Streets
New York City

HIGHLIGHTS . . .

- 25 Sessions
- Panel Discussions
- Technical Exhibits
- Two Receptions
- President's Reception & Dinner Dance

CLIP & MAIL

Director of Front Office Operations
The New York Hilton
1335 Avenue of the Americas
New York, N.Y. 10019

Phone: 212-586-7000

for hotel use only

NAME ____________________ TITLE ____________
FIRM ____________________ PHONE ____________
STREET ____________________________________
CITY/STATE/ZIP ____________________________
REMARKS ___________________________________
(please print)

NOTE: Reservations must be received no later than March 30, 1988 and will be held only until 6 p.m. on day of arrival unless guaranteed.

ARRIVAL DATE ____________________
(check-in time is 3 p.m.)
☐ 2-4 p.m. ☐ 4-6 p.m. ☐ 6-8 p.m. ☐ after 8 p.m.

DEPARTURE DATE ____________________
(ckeckout time is 1 p.m.)

HAVE A CREDIT CARD? To take advantage of this credit card PAYMENT GUARANTEED reservation, please complete the information below.

☐ MasterCard
☐ Visa
☐ American Express
☐ Diners Club
☐ Carte Blanche

Expiration Date ____________

____
Credit Card No.

Signature ____________________

MEDICAL SOCIETY OF THE STATE OF NEW YORK
Annual Meeting of the House of Delegates
April 21–24, 1988
Annual CME Assembly
April 22–24, 1988

Circle Preferred rate:
Singles: $135 $150 $160
Doubles/Twins: $160 $175 $185
Executive Tower: $205 Single/Double
Suites: (Parlor & 1) $365 $400
Suites: (Parlor & 2) $465 $500

Rates subject to 8¼% N.Y. State sales tax; $2.00 per room per night, and 5% N.Y.C. occupancy taxes. (If room at the rate requested is unavailable, one at the nearest available rate will be reserved.)

GENERAL INFORMATION

Don't miss this opportunity to gain Category 1 credits over a weekend when you are free from office hours. Bring your family to New York City to enjoy the many sights.

All physicians, members of the allied professions, and their guests are invited to attend the Medical Society of the State of New York's Annual CME Assembly to be held April 22–24, 1988, at the New York Hilton. The two and one-half days of meetings will provide the individual physician with a possible 18 credits out of a total of 93 Category 1, CME credit hours being offered. The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians. Over 125 outstanding speakers, including a number from out-of-state, will present papers at 25 sessions.

The theme of the CME Sessions will be "Newer Approaches to Habit Abuse and Dependency". A few of the subjects to be presented will be, Human Abuse Through the Ages: "From the Womb to the Tomb"; Managing Life — Style Abuses of the G.I. Tract; Wake Up! Sleep Apnea Can Kill You; AIDS and Self-Induced Skin Diseases. For details, see the Preliminary Program which appears in the January 1988 issue of the *New York State Journal of Medicine.* Our program will offer the clinician timely approaches in diagnosis and treatment of diseases in a wide range of medical specialties.

Here are comments from some of the attendees at the 1987 CME Assembly: "The best program I have attended in 25 years; "The speakers were stimulating and gave scholarly presentations", "The subjects were timely and practical". "The sessions were well organized and informative".

Professional representatives from more than 75 companies will be in the Rhinelander Gallery Exhibit Hall on Friday and Saturday to discuss their products and services. The Technical exhibits will include drug products, office management systems, medical and surgical suppliers, health insurance plans, pension plans and more.

SPECIAL EVENTS

Thursday, April 21

The Annual Dinner Dance, in honor of our President, Samuel M. Gelfand, M.D., will be held in the Trianon Ballroom of the New York Hilton. The reception will be held in the Mercury Ballroom starting at 7:00 p.m. All attendees are welcome. Tickets are $75.00 per person and should be ordered in advance from the Medical Society.

THE WOOLWORTH BUILDING

— *Courtesy David S. Arsenault*

Friday, April 22 and Saturday, April 23

A reception for physicians, their guests, the allied professions and exhibitors will be held both days in the Exhibit Hall between 5:00 p.m. and 6:00 p.m. A free drawing for a color TV set will take place at both receptions.

HOTEL RESERVATIONS

Physicians must make their own reservations with the New York Hilton no later than March 30, 1988. After that date, rooms are on an availability basis only. If making reservations by telephone, please mention that you are attending the Medical Society Convention for billing at the special rate.

REGISTRATION

CME Registration will take place in the Exhibit Hall on Friday, April 22 and Saturday, April 23 from 8:00 a.m.–5:00 p.m., and on Sunday, April 24, registration will take place on the 2nd floor Promenade from 8:00 a.m.–12:30 p.m. There is a $25.00 registration fee for MSSNY members or members of any state medical society.

House of Delegates registration will take place on the 2nd and 3rd floor Promenades at the following hours: Wednesday, April 20 from 12 noon–6:00 p.m.; Thursday, April 21, Friday, April 22 from 8:30 a.m.–5:30 p.m., Saturday, April 23 from 8:30 a.m.–5:00 p.m., and Sunday, April 24 from 8:30 a.m.–12 noon. All MSSNY members are invited to attend the meetings.

CME MEETING HOURS

Friday, April 22 —	8:30am – 1pm
	2pm – 5pm
Saturday, April 23	8:30am – 1pm
	2pm – 5pm
Sunday, April 24	8:30am – 1pm

EXHIBIT HOURS

Friday, April 22 —	9am – 6pm
Saturday, April 23	9am – 6pm

1988 ANNUAL CME ASSEMBLY

Medical Society of the State of New York
The New York Hilton, New York City

Newer Approaches to Habit Abuse and Dependency

FRIDAY, APRIL 22

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Plenary Session: Human Abuses Through the Ages: "From the Womb to the Tomb," *Sutton South/Regent Parlor*
2:00 p.m. - 5:00 p.m.	Women's Medical Society/Occupational Health, *Gramercy B*
	Cardiovascular Diseases, *Sutton South*
	Physical Medicine & Rehabilitation/Family Practice/Orthepedics, *Gramercy A*
	Emergency Medicine/Critical Care, *Murray Hill B*
	Pathology/Pediatrics, *Bryant Suite*
	General Surgery/Radiology, *Regent Parlor*
	Neurosurgery, *Murray Hill A*
	Medical Liability Mutual Insurance Co., *Rendezvous Trianon*

SATURDAY, APRIL 23

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Internal Medicine (All Day), *Regent Parlor*
	Ophthalmology (All Day), *Sutton South*
	Hand Surgery/Neurology (All Day), *Sutton Parlor North*
	Psychiatry (All Day), *Gramercy B*
	Urology/Radiology, *Nassau A*
	Dermatology, *Murray Hill A*
2:00 p.m. - 3:00 p.m.	Medical Technology
2:00 p.m. - 5:00 p.m.	Internal Medicine, *Regent Parlor*
	Ophthalmology, *Sutton South*
	Hand Surgery/Neurology, *Sutton Parlor North*
	Psychiatry, *Gramercy B*
	Chest Diseases/Otolaryngology, *Murray Hill B*
3:00 p.m. - 5:00 p.m.	Computers in Medicine, *Nassau A*

SUNDAY, APRIL 24

8:30 a.m. - 9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m. - 1:00 p.m.	Allergy & Immunology, *Gramercy B*
	Obstetrics and Gynecology, *Murray Hill A*
	Gastroenterology/Colon and Rectal Surgery, *Nassau A*
	Orthopedics/Family Practice/Physical Medicine & Rehabilitation/Anesthesiology, *Regent Parlor*
	Sleep Disorders—Apnoea, *Nassau B*

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis. The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians.

□

Technical Exhibits

United Nations Headquarters Overlooking the East River
Photo courtesy of David S. Arsenault

Advance Registration Form

1988 Annual CME Assembly, April 22–24
The New York Hilton, New York City

Complete and return this form to the Medical Society address as indicated below.

Name____
PLEASE PRINT LAST FIRST

Address____

City/State/Zip____

Medical Specialty______________________________________

REGISTRATION FEE *Please check categories*

☐ Active Member*	$25.00	☐ Nurse	☐ Lab Technician
☐ Non-Member Physician*	$50.00	☐ Physician Assist.	☐ PT/OT
☐ Allied Profession	$10.00	☐ Dentist	☐ Other______ Specify

NO FEE *Please check category*

- ☐ MSSNY Life Member
- ☐ Resident/Intern
- ☐ Medical Students
- ☐ Medical Assist.
- ☐ Speaker
- ☐ Spouse/Family
- ☐ News Media/Medical Writer

REMITTANCE ENCLOSED $__________

* of any state medical society

Please make check payable to:
Medical Society of the State of New York, and return this form with remittance to: Division of Meeting Services/MSSNY, 420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042.

Important: Your ID Badge will be held at the MSSNY Registration Desk located in the Exhibit Hall, on the second floor of the New York Hilton.

NEW YORK STATE
JOURNAL OF MEDICINE

December 1987 • Volume 87, Number 12

COMMENTARIES

Sleep deprivation in physician training

> Sleep that knits up the ravell'd sleave of care,
> The death of each day's life, sore labour's bath,
> Balm of hurt minds, great nature's second course,
> Chief nourisher in life's feast
>
> —Shakespeare, *Macbeth*

Professional organizations and bureaucracies can have an uncanny way of undermining the very values that define the work of their members. Medicine, for example, has as its fundamental aim the relief of suffering and the lessening of human misery. As an organized profession, however, it sometimes indulges in certain practices that are harmful both to its members and to the patients it serves. As a physician I feel compelled to call attention to these practices because I believe their perpetuation constitutes no less an inhumanity than their original imposition. I refer, in short, to the nearly ubiquitous custom of requiring doctors-in-training, ie, hospital interns and residents, to work continuously every third or fourth day for 34 to 40-plus hours at a stretch without an assured period of sleep. During my internship I personally averaged approximately one and a half hours of sleep per 36-hour period. The consequences of such a system are, to my mind, far-reaching.

Let me begin with the medical housestaff, the interns and residents who staff most of our teaching hospitals around the clock. The routine pressures, stresses, and demands of caring for a sizable number of patients in today's hospital are not inconsiderable. A high degree of diligence, thoroughness, exquisite attention to detail, as well as a fair amount of intellectual and manual skill are the minimum requirements for competence, constantly demanded and daily employed. Patients admitted to hospitals nowadays are generally more ill than ever before, frequently on the brink of death; and technology makes possible a wider and ever-burgeoning array of therapeutic interventions. Lapses or oversights can sometimes prove fatal. Emergencies necessitating quick and abundant action occur rather frequently. The atmosphere is, to say the least, highly charged.

Immersed in this veritable cauldron of suffering, surrounded by death, compelled to energized vigilance and plain hard work, residents are regularly and systematically deprived of "sore labour's bath, balm of hurt minds" [1]—the much-needed refuge of sleep. It is interesting to note that a common form of torture employed in oppressive political regimes is routinely seen in the training of designated servants to humanity.

The physical effects of exhaustion—slow-wittedness, decreased strength, and impaired dexterity—during the period of sleep deprivation seem obvious. But there are other consequences as well. Curious things happen to a resident's psyche. The hurt, frustration, resentfulness, and intense anger that inevitably follow from what seems to be an endless, claustrophobic cycle of duties are ultimately directed against patients. This is irrational, unfortunate, but true. The patient comes to be regarded more as a "torture machine," ceaselessly bothering residents with demands for care, than as a suffering human being. The goal of treatment becomes disposal (getting the patient out of the hospital) instead of palliation, an attitude that has been well documented.[2] Sympathy and compassion, under the haze of heavy-lidded eyes, give way to hostility and disinterest.

Anyone falling within earshot of a group of residents at the luncheon table would be privy to the black humor directed towards patients. The patient has become the enemy, when the real enemy is the senseless system.[2]

By no means do I imply that we residents routinely indulge in cruelties. Our gallows humor is insurance against this. However, sympathy and tolerance for suffering certainly lessen. During the years that are critical for the cultivation of the appropriate relationship to patients, disease, and death, the poison of an adversarial attitude is introduced and allowed to flourish.

There are other consequences, perhaps more readily apparent. Demoralization, despair, and apathy beset residents. Breakdowns occur. Tempers shorten and outbursts of rage and rudeness flare, to the detriment of nurses and other hospital staff, including colleagues. I know of some residents who have retched, and others who have succumbed to tears during their sleepless stints. And I know of several cases where the muddied thinking and general dullness of mental faculties under conditions of sleep deprivation have resulted in suboptimal treatment. In one such instance, a fatality nearly occurred. It should be obvious that someone who has not slept for 24 or 30 or 34 or 40 hours may not be the person to be relied on for clear, rational decision making, especially where life and death may be at stake. Any sensible individual would cringe at the idea of entrusting his life to a groggy airline pilot. Does one somehow feel more secure being admitted to the hospital under the care of a bone-weary intern who, with the mere slip of his pen, might jeopardize one's existence?

For those of us who survive the training process intact, scars remain. Even in the best of us there would seem to be an ingrained sense of superiority to patients. Furthermore, a sense of entitlement that often begins in medical school continues to develop.[3] After passage through such an onerous period we come to believe that we actually deserve to have the accoutrements of the good life. We become entrenched members of an elite, a breed of royalty, forming an exclusive fraternity. And it is this which explains in part the public's mounting dissatisfaction with physicians.

Sadly, the misery continues to be revisited upon others. Fresh medical school graduates are themselves subjected to the same senseless hazing. The victim has become the persecutor. "They should go through what we went through" is the attitude that prevails.

There is an attempt to justify the present on-call system by citing its value in building character, teaching residents to handle emergencies, and helping students master the details of medicine when a patient remains acutely ill over a long period of hours or days. This is hogwash. Enough emergencies occur both day and night to test our mettle. And education actually suffers greatly. I have learned little, if anything, while groggy. Worse yet, the desire to learn and study, to plumb the depths of medical knowledge during off-hours, is utterly extinguished by the fatigue, and the repulsion against all medical material which we come to feel. The detailed, comprehensive histories and physical exams which we strove so hard to master in medical school become embarrassingly skeletal. The system tends to turn generally compassionate people into cynical automata, at least temporarily; it inspires regression and decompensation rather than healthy growth.

At least one solution does exist, one that requires no additional expenditure of scarce financial resources (not that this should stand in the way), and one that a number of progressive institutions have begun to employ with considerable success. It is simply the use of a shift system, or, as we know it in medical circles, the "night-float." Essentially, it guarantees a period of sleep for each resident on or off call. For example, during my on-call day (the day when I am responsible for admitting patients), I would work until 11 PM, at which time I would be relieved by a fresh resident whose task for the month would be working nights. And at 7 or so the next morning I would return to resume my responsibilities and relieve the night worker, refreshed and possibly even enthusiastic.

I am convinced that this simple change would confer tremendous benefit on medical training. Residency would by no means be devoid of tribulations, but it would prove to be a much more tolerable and humane experience, as implementation has already demonstrated. The solace of assured sleep would make all the difference in the world.

I would like to emphasize yet another consequence of the current process. After we pass through an internship we tend to ignore or deny its damages, preferring instead to preserve only the moments that have given satisfaction, for example, the instances when our work has "paid off" in a "cure." Dismal memories recede, and with them our desire to effect change. In this way we contribute to perpetuating the system, a marked transgression of our own Hippocratic vows, thereby committing future physicians to needless torment and patients to poorer care. This may explain why the advantageous night float system has not yet been adopted on a wide scale across the country.

Recently, *The New York Times*, *Newsweek*, and *Sixty Minutes* touched on some of the points I raise. Even a few of my patients have uneasily asked whether I was one of those "36-hour" doctors. My impression, however, is that the public remains generally ignorant of the process and how it affects them. In no other profession would such reckless practices be tolerated. As a physician, I call on the public to exert its influence on the medical profession to bring about an end to the useless and harmful system I have described. The result would be better medical care and perhaps even the disappearance of the distasteful relics of residency—elitism and entitlement.

EMANUEL E. GARCIA, MD
2120 Race St
Logan Square
Philadelphia, PA 19103

(*Dr Garcia has recently completed his internship in a Philadelphia hospital.*)

1. Shakespeare W: *Macbeth*, II, ii, lines 38–41.
2. Mezrahi J: *Getting Rid of Patients. Contradictions in the Socialization of Physicians.* New Brunswick, NJ, Rutgers University Press, 1986.
3. Dubovsky JL: Coping with entitlement in medical education. *N Engl J Med* 1986; 315:1672–1674.

Renal adenocarcinoma

Few tumors have caused as much confusion histologically and histogenically as the malignant epithelial tumors of the kidney parenchyma. Regardless of whether they are called hypernephroma, renal cell carcinoma, or just renal cancer, they are all adenocarcinomas. Their diagnosis and management continue to present a complex problem to the practicing physician. Though a relatively rare neoplasm comprising only 3% of all tumors in the adult, it is the most common malignant tumor of the kidney. However, there is a relative scarcity of information concerning this disease in childhood. Since it comprises less than 7% of all primary intrarenal tumors in persons under 21 years of age, few urologists have had experience in dealing with this entity. In this issue of the *Journal*, Kaufmann and Becker[1] provide an overview of the diagnosis, evaluation, and management of this disease in children. The authors present a review of the literature and highlight the similarities and differences between a renal cell carcinoma in the child and in the adult.

They describe a seven-and-a-half-year-old boy with painless gross hematuria and a tender, palpable abdominal mass. An intravenous urogram demonstrated a tumor, and arterial studies demonstrated an avascular mass. Nephrectomy was performed, and the diagnosis was a clear cell renal cell carcinoma.

The presentation of tumors in adults and children is similar. One point made in the report by Kaufmann and Becker[1] is that in children, a renal mass is more easily palpable and, therefore, is usually diagnosed at an earlier age and with less likelihood of associated metastatic disease. Though rarely seen, renal cell carcinoma must be considered in a child with an abdominal or flank mass.

Symptoms of renal cell carcinoma in adults and children frequently consist of hematuria, flank pain, or symptoms related to metastasis. Key aspects of the history often include the presence of red urine. Microscopic or gross hematuria in the presence or absence of a urinary tract infection is an indication for urologic referral in order to rule out a tumor of the urinary system. Physical examination is extremely important, since a flank or abdominal mass may be present.

The diagnosis demands an orderly approach. Traditionally, the diagnostic workup would include intravenous urography. The finding of any suspicious lesion would then lead to renal aortography, which would highlight the great vascularity of the tumor and any other anatomic abnormalities. Arteriography formerly was the mainstay in diagnosis. However, with the arrival of newer technology, ultrasound and computed tomography (CT) are now liberally used in the evaluation of nonspecific abdominal pain and other conditions. Therefore, more unsuspected tumors are identified at earlier stages, even before there are symptoms of hematuria or flank pain. Also, arteriography is no longer considered mandatory, and a conclusive diagnosis is frequently made on the basis of CT and sonography. Though the morbidity associated with renal angiography is minimal in skilled hands, distal emboli, hemorrhage, and impairment of renal function have been reported. Numerous authors have compared CT to angiography and have concluded that tomography is superior both in accuracy and in cost effectiveness. Of major importance is that a CT scan was used to diagnose renal vein involvement, vena caval extension, tumor outside the kidney, and lymph node metastasis in more than 80% of the cases.[2] Therefore it is believed by many that as a single diagnostic study, CT is the most cost effective and accurate method of evaluating a renal mass.

The treatment of patients with advanced renal cell carcinoma continues to be a problem. No effective systemic therapy is currently available and, therefore, the indications for an operation must be extended as far as is reasonable and prudent. Extension of disease into the renal vein and inferior vena cava does not necessarily carry a poor prognosis. A review of 19 cases at the Massachusetts General Hospital showed that survival was influenced more by the tumor's extending beyond the renal capsule than by vascular invasion.[3] The three- and five-year survival rates for patients with renal vein and inferior vena caval extension and tumor beyond the renal capsule were 14% and 0%, respectively, compared to 82% and 67% for patients with tumors confined to the renal capsule with renal vein and inferior vena caval involvement, respectively. These results support the need for aggressive surgical management. When the stage of disease was determined retrospectively for a large group of patients, it was shown that the timing of the surgical procedure performed had little effect on the outcome.[4] Survival of patients depended on the local extent of the primary tumor determined at the time of surgical exploration, being most favorable in patients with small and large tumors confined to the renal capsule.

Renal cell carcinoma can occur at any age but is rare in childhood. A review of the literature has not revealed any biological differences between childhood and adult disease. The signs and symptoms are highly variable, but the classic findings include hematuria, flank pain, and a mass. CT and ultrasonography represent major advances in the diagnosis and treatment of patients with localized disease. However, treatment of patients with advanced disease continues to be a problem. The excellent results of combination chemotherapy and surgery in the treatment of Wilms and testicular tumors has yet to be approached, and the radioresistance of this disease has been confirmed. In recent years, there has been an increased interest in interferon as an antitumor agent, with some responses noted in patients with metastatic disease. However, interferon has not yet offered significant benefit. Surgery, with the objective of excision of all tumor with adequate surgical margins, remains the only method of treatment of primary renal cell carcinoma.

BARRY N. NOCKS, MD
Assistant Professor of Surgery
Harvard Medical School

Director of Urology
Spaulding Rehabilitation Hospital

Associate Urologist
Massachusetts General Hospital
Boston, MA 02114

1. Kaufmann T, Becker NH: Renal adenocarcinoma in the pediatric age group. *NY State J Med* 1987; 87:664–665.
2. Jaschke W, van Kaick G, Peter S, et al: Accuracy of computed tomography in staging of kidney tumors. *Acta Radiol* 1982; 23:593.
3. Heney NM, Nocks BN: The influence of perinephric fat involvement on survival in patients with renal cell carcinoma extending into the inferior vena cava. *J Urol* 1982; 128:18–20.
4. Bassil B, Dosoretz DE, Prout GR Jr: Validation of the tumor, nodes and metastasis classification of renal cell carcinoma. *J Urol* 1985; 134:450–454.

Acoustic neuromas: Yesterday, today, and tomorrow

The surgical management of acoustic neuromas was introduced in the 1890s.[1] The early experiences involved heroic procedures with high mortality rates and little control over residual neurologic deficits. In the early 1900s, Harvey Cushing[2] advocated a bilateral suboccipital craniotomy and intracapsular subtotal removal of these tumors. This resulted in a marked decrease in operative mortality and in the severity of postoperative neurologic deficits. Unfortunately, no one was cured by use of this approach, but because these are slow-growing tumors, the patients were less likely to die as a direct result of the tumor or the treatment. Walter Dandy[3] recognized that these tumors were related to cranial nerve VIII and correlated the clinical symptoms of hearing loss, tinnitus, and imbalance with the lesion. He reintroduced total removal of the tumor as the appropriate treatment. Improvements in anesthesia, the availability of blood transfusion, and an awareness of smaller tumors allowed Dandy to decrease the operative mortality rate in his series and to achieve a cure for some patients.[3]

In the decades between World War I and World War II, great strides were made in diagnostic capability. Caloric testing and audiometry were introduced and provided objective evidence of nerve VIII abnormality. Roentgenographic techniques were devised for detecting destruction of the petrous bone, which was indirect evidence of a mass lesion. Cautery, as well as blood replacement and improved anesthetic agents, made surgery easier. During the 1950s, these tumors were approached through the posterior cranial fossa, and total removal was the goal. However, hearing loss, facial paralysis, and corneal anesthesia were common, and the operative mortality rate was high.[4]

In the late 1950s, William House[5] introduced a new approach—translabyrinthine—and a new technique—microsurgery. The early results with this approach were uneven. Often, two procedures were required, and incomplete removal and cerebrospinal fluid fistulas were frequent. Initially, this approach was suspect in the neurosurgical community. Neurosurgeons were not familiar with the temporal bone anatomy, and most of them had little or no experience with microsurgical techniques. As experience with this procedure and these techniques increased, it became apparent that total tumor removal, with facial nerve preservation and a low mortality rate, was feasible. Absolute numbers are difficult to find, but it is probable that half of all acoustic neuroma removals now are done by the translabyrinthine approach.

During the 1960s and 1970s diagnostic techniques again improved. Traditional roentgenography depended on indirect evidence of tumor, namely bone erosion. Pneumoencephalography and contrast myelography, using air and contrast, respectively, were uncomfortable, expensive, of some risk to the patient, and not completely reliable. Computed tomography (CT) was introduced in 1973, and the early scans were useful for identifying tumors that extended at least 2 cm into the posterior cranial fossa. It was then observed that CT with intravenous injection of contrast produced a positive image of the tumor, which confirmed its presence and revealed its location and size. Over the next decade, the technology rapidly improved, and nearly all tumors that extended into the posterior cranial fossa could be identified.

The very small tumor (intracanalicular) was still a problem because of the temporal bone density. Air cisternography with CT was introduced. This involves the injection of 5–10 mL of air into the lumbar subarachnoid space, which then is maneuvered into the cerebellopontine angle. The air provides marked contrast to the bone and allows visualization of tumors within the internal auditory canal. This is considered to be the most definitive test at the present time.

The role of magnetic resonance imaging (MRI) is still evolving. The medium and large tumors are easily seen. Positive identification of small tumors has been a problem. Use of head coils in the magnetic resonance procedure has produced impressive images of the temporal bone. It is possible to identify the individual nerves within the internal auditory canal with these coils. The obvious advantages are avoiding radiation and the injection of air. The disadvantages have been high cost, restricted availability, and lack of confidence in interpretation of the films. As experience increases and technology improves, MRI will likely play a larger role.

Audiometry has also advanced. The routine testing of air conduction, bone conduction, and speech remains the best screening device available. Any patient who shows asymmetry of hearing ability must be suspected of having a cerebellopontine angle lesion. In our experience, only 5% of patients with tumor have "normal" hearing and a significant number of these show some asymmetry. During the 1960s, Bekesy testing, the Short Increment Sensitivity Index, alternate binaural loudness balance testing, and tone decay were utilized to identify retrocochlear lesions. These have been replaced by acoustic reflex testing and brainstem evoked audiometry. Generally, these tests are more accurate but require that some hearing be present. At least 30% of patients with surgically proven acoustic neuromas have no hearing.

Currently, total removal of acoustic neuroma can be expected in 95–98% of the cases. The operative mortality is in the range of 1% to 5%. Anatomic preservation of the facial nerve is in the 90% range,[6-9] although there is still significant impairment in its function in many cases. Hearing preservation is relatively infrequent. Cerebrospinal fluid fistula remains the most common complication but this is fairly easy to manage. The report by Mattucci et al[10] in this issue of the *Journal* highlights many of the diagnostic and therapeutic issues associated with these tumors.

As we look to the future, further improvements appear to be possible. There will be a greater emphasis on intraoperative monitoring of the facial nerve.[11] It is anticipated that fewer nerves will be lost and more facial function will be preserved. There is also some desire now for preservation of hearing. For this to happen, cochlear monitoring will need to be refined. It also will be necessary to reassess the surgical approach. Hearing preservation is not possible with the translabyrinthine approach but is possible when one uses the posterior fossa and middle fossa approaches. Other aspects that need investigation are more reliable methods of preventing cerebrospinal fluid leakage, the etiology of these tumors, and the role of genetic screening.

The medical community can be proud of the improvements in management of acoustic neuroma. These tumors are being recognized at a much earlier point in their development and are being removed with very low morbidity and mortality. A high degree of cooperation has developed among otologists, neurosurgeons, neuroradiologists, neurologists, and ophthalmologists, all to the benefit of the patients.

STEPHEN G. HARNER, MD
Consultant
Department of Otorhinolaryngology
Mayo Clinic and Mayo Foundation

Associate Professor of Otolaryngology
Mayo Medical School
Rochester, MN 55905

1. Ballance CA: *Some Points in the Surgery of the Brain and Its Membranes.* London, Macmillan and Co, 1907, p 276.
2. Cushing H: *Tumors of the Nervus Acusticus and the Syndrome of the Cerebellopontile Angle.* Philadelphia, WB Saunders Co, 1917.
3. Dandy WE: An operation for the total removal of cerebellopontile (acoustic) tumors. *Surg Gynecol Obstet* 1925; 41:129–148.
4. Olivecrona H: Acoustic tumors. *J Neurosurg* 1967; 26:6–13.
5. House WF: Transtemporal bone microsurgical removal of acoustic neuromas. Report of cases. *Arch Otolaryngol* 1964; 80:617–667.
6. House WF, Nelson JR: Long-term cochleo-vestibular effects of acoustic tumor surgery, in House WF, Luetje CM (eds): *Acoustic Tumors: Management.* Baltimore, University Park Press, 1979, vol 2, pp 207–234.
7. House WF, Hitselberger WE: Fatalities in acoustic tumor surgery, in House WF, Luetje CM (eds): *Acoustic Tumors: Management.* Baltimore, University Park Press, 1979, vol 2, pp 235–264.
8. Harner SG, Ebersold MJ: Management of acoustic neuromas, 1978–1983. *J Neurosurg* 1985; 63:175–179.
9. Glasscock ME 3d, Kveton JF, Levine SC, et al: A systematic approach to the surgical management of acoustic neuroma. *Laryngoscope* 1986; 96:1088–1094.
10. Mattucci KF, Glass WM, Setzen M, et al: Childhood acoustic neuroma. *NY State J Med* 1987; 87:665–666.
11. Harner SG, Daube JR, Ebersold MJ, et al: Improved preservation of facial nerve function with use of electrical monitoring during removal of acoustic neuromas. *Mayo Clin Proc* 1987; 62:92–102.

Henry Irwin Fineberg, MD

Dr Henry I. Fineberg, who served as the Executive Vice-President of the Medical Society of the State of New York for a 20-year period from 1961 to 1981, died on Sunday, October 4, 1987, at Huntington (L.I.) Hospital. He was 83 years old.

Dr Fineberg was born on January 23, 1904, in Cleveland, Ohio, and attended Columbia College, from which he received a BA degree in 1924. He studied medicine at Yale University School of Medicine, receiving his MD degree in 1927. He practiced pediatrics, with a special interest in communicable diseases, in Brooklyn until 1939, when he became physician-in-charge of the Kingston Avenue Hospital for Communicable Diseases in Brooklyn, NY.

In 1941 Dr Fineberg became the Medical Superintendent of the Queens General Hospital and a year later the Supervising Medical Superintendent of the Queens Hospital Center, which at the time included Queens General Hospital, the Triboro Hospital for Tuberculosis, and the Queensboro Hospital for Communicable Diseases. He held the latter position until 1957.

In 1957 Mayor Robert F. Wagner appointed Dr Fineberg Deputy Commissioner of Hospitals in charge of medical affairs. In this capacity he supervised the medical boards and the medical activities of the 20 hospitals then in the New York City municipal hospital system. Four years later, in 1961, Dr Fineberg became the Executive Vice-President of the Medical Society of the State of New York (MSSNY), a position he held until his retirement in 1981.

Dr Fineberg's association with the MSSNY and the Medical Society of the County of Queens (MSCQ) spanned a number of years prior to his assuming the MSSNY's executive vice-presidency. He served as a trustee of the MSCQ from 1946 to 1949 and 1952 to 1973 and as a delegate to the MSSNY from 1949 to 1954. From 1957 to 1973 he was Chairman of the Board of Trustees of the MSCQ, in 1950 was President-Elect, and in 1951 was President.

Dr Fineberg was an active member of the MSSNY for many years, serving on the Council from 1954 to 1960. A member of the Executive Committee, 1955–1959, he became President-Elect, 1958–1959, and President, 1959–1960. He then served as a trustee for the period of one year, from 1960 to 1961. He was a delegate to the American Medical Association (AMA) from 1959 to 1978 and was an advisor to the MSSNY Auxiliary from 1961 to 1975. He served as chairman of numerous society committees, including legislation, public relations, medical policies, and others.

Dr Fineberg also served on the boards of a number of governmental and voluntary organizations, including the New York State Hospital Review and Planning Council, the Committee on Medical Grievances of the New York State Education Department, and the Governor's Advisory Council on Narcotic Addiction. He was a member of Mayor John V. Lindsay's Task Force on Hospitals and a member of the Faculty Council of New York Medical College from 1946 to 1957. He was also a member of the Advisory Committee on Medical Care of the New York State Department of Health from 1966 to 1968. He was a member of many social, civic, and fraternal organizations as well, including the Elks, Masons, Shriners, and the Kiwanis Club of Fresh Meadows.

Between 1952 and 1966 he was a medical columnist for the *Long Island Press*, writing a medical advice column called "The Doctor's Corner."

As Executive Vice-President Dr Fineberg oversaw the MSSNY's move from its Manhattan offices at 750 Third Avenue to Lake Success on Long Island in 1971. A highly respected medical administrator, he was a man of broad experience and unique skills as well as a compassionate and caring physician.

In 1980 the MSSNY established the Henry I. Fineberg, MD, Award for Distinguished Service which is annually presented by the Board of Trustees of the society.

Dr Fineberg is survived by his wife of 51 years, Grace; a daughter, Karen Fineberg of Dix Hills; and a son, Coleman Fineberg, of East Northport, who is the Executive Director of the Suffolk County Medical Society. Dr Fineberg is also survived by three grandchildren.

Pascal James Imperato, MD
Editor

". . . Henry I. Fineberg, MD . . . served our Society and the medical care interests of the people of New York State with distinction for many years. He is especially remembered for his many contributions to this Society as a Councilor, President and Executive Vice-President, and for his work in the field of communicable diseases which led in 1957 to his appointment as Medical Deputy Commissioner of Hospitals for the City of New York. Dr Fineberg will be sorely missed."

Samuel M. Gelfand, MD
President
Medical Society of the State of New York

Cancer screening and prevention

Knowledge, attitudes, and practices of New York City physicians

DONALD H. GEMSON, MD, MPH; JACK ELINSON, PhD

ABSTRACT. The degree to which practicing physicians are familiar with and adhere to guidelines for cancer screening and prevention is uncertain. A survey of 120 randomly selected primary care physicians in New York City (response rate = 90%) questioned physicians with respect to their knowledge of and compliance with American Cancer Society (ACS) guidelines for the cancer-related checkup. Results revealed that a significant proportion of physicians did not follow certain ACS guidelines, particularly with respect to mammography (20%), sigmoidoscopy (41%), and stool guaiac test (48%). Although almost three quarters of physicians cited the American Cancer Society as a source of information for preventive medicine, relatively few were able to recall selected ACS guidelines for cancer screening and prevention. Results of this survey suggest a need for increased dissemination and promotion of guidelines for cancer screening and prevention in primary care.

(NY State J Med 1987; 87:643–645)

Recommendations for cancer screening and prevention in primary care have come from a variety of sources within the past decade. The American Cancer Society,[1] the Canadian Task Force Report on the Periodic Health Examination,[2] Frame and Carlson,[3] and Breslow and Somers[4] have offered recommendations, subsequently cited by the American College of Physicians[5] and the American Medical Association[6] as appropriate guides to primary care physicians. The United States Preventive Services Task Force has recently begun to issue recommendations for health promotion and disease prevention in primary care as well.[7]

While guidelines for cancer screening and prevention are readily available, the degree to which practicing physicians are familiar with and adhere to the guidelines cited above is uncertain. A survey of Canadian physicians, conducted shortly after publication of the Canadian Task Force Report on the Periodic Health Examination, found that the majority of physicians were not following Task Force recommendations for breast cancer screening or checking the stool for occult blood.[8] A survey of physicians in the United States, conducted by the American Cancer Society, found that ACS guidelines were followed infrequently for sigmoidoscopy (18% of physicians surveyed) and for mammography (11% of physicians surveyed).[9]

Failure to comply with guidelines for cancer screening and prevention may be due to a variety of reasons. Lack of awareness or knowledge of the guidelines on the part of physicians, patient resistance, economic factors, physicians' disagreement with the guidelines, or, perhaps, an attitudinal resistance to the value of preventive medicine in general or to cancer screening in particular may contribute in varying degrees to a failure to perform recommended tests and procedures.

With the increasing interest in disease prevention in the US health care system, manifested in part by the array of published guidelines intended to aid physicians in the practice of preventive medicine, it is of interest to know to what extent primary care physicians have decided to incorporate cancer prevention guidelines into their practices and to document the factors that contribute to their decisions. In order to determine the attitudes, knowledge, and practices of physicians in New York City with respect to cancer screening and prevention, we analyzed data from a survey of 120 randomly selected primary care physicians with respect to their adherence to cancer screening guidelines issued by the ACS.[1]

SUBJECTS AND METHODS

The details of the sampling and interview process have been described elsewhere.[10] In brief, 307 physicians with office listings in New York City were chosen in a systematic probability sample from among the physicians listed in the 1982–1983 Directory of the Medical Society of the State of New York. To be eligible, physicians were required to spend at least 50% of their clinical practice time in either internal medicine, family practice, or general practice. Of the 307 physicians initially selected, 134 were found to be eligible, and 120 completed telephoned interviews (response rate = 90%) that lasted an average of 25 minutes. Participants were informed that the New York City Division of the American Cancer Society was the primary sponsor of the survey. The survey was completed in October 1984.

Questions relating to cancer screening and prevention were asked so as to conform to ACS guidelines. For purposes of this analysis, physician recommendations for screening and prevention were evaluated in regard to the three most common causes of cancer mortality in the United States: lung cancer, colon cancer, and breast cancer.[11] In each case, physicians were asked whether

---

From the Columbia University School of Public Health, Division of Sociomedical Sciences, New York, NY.

Address correspondence to Dr Gemson, Columbia University School of Public Health, Division of Sociomedical Sciences, 600 West 168th St, New York, NY 10032.

they agreed with the ACS guideline for that particular type of cancer (eg, "Do you recommend periodic mammography for female patients over age 40?") without identifying the guideline. Participants were informed that all questions referred to asymptomatic patients with no known chronic diseases. Respondents who disagreed with an ACS guideline were read a series of possible reasons for not recommending that test or procedure, and were asked whether they agreed or disagreed with each one.

RESULTS

Of the 120 physicians who responded, 79% practiced internal medicine, 13% were in family practice, and 8% identified themselves as general practitioners. Most of the physicians (67%) were in solo practice, with the second most frequent practice type being hospital-based salaried (13%). The mean year of graduation from medical school was 1955, with a range from 1930 to 1978. Approximately half the physicians (51%) said they were board-certified, and 55% were foreign medical graduates (FMGs). (Although the percentage of participating physicians who were FMGs (55%) exceeded the percentage of FMGs in the initial sample of 307 physicians (44%), we suspect that the difference reflects a higher proportion of US medical graduates in subspecialty practices who did not meet the eligibility criteria for "primary care," and were therefore not included in the survey.) The 14 eligible physicians who refused to participate did not significantly differ from participating physicians in age, sex, or whether they were graduates of foreign or US medical schools.

Lung Cancer. The 1980 ACS report, "Guidelines for the cancer-related checkup: recommendations and rationale,"[1] cited several studies that failed to show a significant benefit for annual screening by chest film or sputum cytology, and advised against the use of periodic chest film or sputum cytology screening for early detection of lung cancer in all patients (both smokers and nonsmokers). Nevertheless, 49% of surveyed physicians recommended chest films and 5% recommended sputum cytology as screening tools for early detection of lung cancer in all asymptomatic patients. The proportion of physicians recommending screening tests for patients who smoke was considerably higher: 90% advised chest films and 19% recommended sputum cytology for these patients.

Colon Cancer. The 1980 ACS report[1] recommended an annual stool guaiac slide test for all patients over age 50, and sigmoidoscopy every 3-5 years after two negative examinations one year apart for patients over age 50 for early detection of colon cancer. Just under half (48%) of physicians said they periodically supplied hemoccult or guaiac tests to asymptomatic patients over age 50. Physicians who did not supply hemoccult or guaiac tests indicated the following reasons: risk of false positives (49%), patient resistance (30%), no medical indication (29%), and inaccuracy of the test (29%). When asked whether there were any additional reasons, 34% of physicians who did not supply hemoccult or guaiac tests said they did not do so because they checked for occult blood during the rectal exam.

With respect to periodic sigmoidoscopy, 41% of physicians recommended the procedure for asymptomatic patients over age 50. Physicians who did not recommend sigmoidoscopy indicated the following reasons for not doing so: not medically indicated (70%), patient resistance (65%), cost of the procedure (52%), risk of the procedure (28%).

Breast Cancer. The 1980 ACS report contained a three-fold recommendation for early detection of breast cancer: monthly breast self-examination (BSE); physical examination of the breast by a physician every three years for women aged 20-40 and annually for women over 40; and mammography once between age 35 and 40 as a baseline, every one to two years for women aged 40-49, and annually thereafter.[1] The recommendation for women in their 40s had been revised in 1983.[12]

Almost nine out of ten physicians (88%) said they routinely advised asymptomatic women to perform BSE. Of those, 55% (or 48% of all physicians) said they advised women to perform BSE monthly, with other recommended intervals including every six months (9%), every week (7%), daily (7%), "whenever they can" (7%), every two weeks (6%), and every two months (6%). The 13 physicians who did not recommend BSE indicated the following reasons: it is an unnecessary worry for patients (75%), lack of patient compliance (55%), risk of false positives (42%). The great majority of physicans (94%) did recommend periodic physical examination of the breast by a physician for female patients over age 40.

Only one out of five physicians (20%) recommended periodic mammography for asymptomatic female patients over age 40. The physicians who did not recommend mammography indicated the following reasons: not medically indicated (71%), risk of radiation (57%), cost of the test (50%), unnecessary worry for patients (48%), low yield (47%), patient resistance (45%), and risk of false positives (34%).

Familiarity with ACS Guidelines. Although 71% of physicians cited the ACS as a source of knowledge for their decisions for preventive medicine, less than half of the physicians (47%) said they were familiar with the ACS guidelines for the cancer-related checkup. Of these, 9% said they were "very familiar," 54% said they were "somewhat familiar," and 37% said they were "slightly familiar" with the ACS guidelines. The 57 physicians who expressed at least slight familiarity with the ACS guidelines were asked whether they could recall several specific guidelines. The percentage of physicians who correctly identified certain ACS guidelines and the percentage of physicians who followed the guidelines in clinical practice are shown in Table I.

When asked to recall the ACS guideline for chest film screening, 55% said they could not recall it; 11% said screening was recommended yearly, 9% said every two years, 9% said every one or two years only for smokers, 8% said less frequently than every two years, and 11% cited the actual guideline—that chest film screening was not recommended.

When asked to recall the ACS guideline for checking the stool for occult blood, 50% said they could not recall it; 36% said the test was recommended yearly, 9% said yearly after age 40, 4% said more frequently than yearly, and 2% cited the actual guideline—yearly after age 50. Fifty-four percent of physicians said they could not recall the guideline for sigmoidoscopy; 14% said it was recommended yearly, 14% said yearly after age 40, 5% said every 2-3 years after age 50, 4% said yearly after age 50, 4% said every three years after age 40, 4% said it was not recommended, 2% said every two years, and 4% cited the actual guideline—every 3-5 years after two negative examinations one year apart after age 50.

When asked to recall the ACS guideline for BSE, 45% of physicians said they could not recall it; 38% cited the actual ACS guideline—monthly, 5% said whenever the patient can, 4% said whenever the patient showers, 4% said weekly, 4% said every 2-3 months, and 2% said every six months. When asked to recall the ACS guideline for mammography, 51% said they could not; 12% said it was recommended yearly after age 40, 12% said yearly after age 50, 7% said every 2-3 years, 7% said yearly, 4% said it was not recommended, 4% said it was only recommended if there was a family history of breast cancer, and 5% cited the actual guideline—baseline at age 35, every 1-2 years for women in their 40s, and yearly after age 50.

Physician Attitudes Toward Prevention. Almost nine out of ten physicians (88%) agreed with the statement that "physicians should probably practice more preventive medicine than they presently do," and a similar number (87%) agreed with the statement that "medical schools should put more emphasis on preventive medicine in their curricula." Approximately two thirds of physicians (66%) said they would attend a continuing medical education course specifically devoted to preventive medicine. Almost four out of five physicians (79%) supported the need for a task force to "clarify recommendations" for preventive medicine. It would appear that the majority of physicians surveyed were supportive of the value of preventive medicine in primary care.

DISCUSSION

The potential value of prevention in reducing cancer mortality has received renewed attention recently with the observation by Bailar and Smith[13] that the predominantly treatment-based approach to cancer has resulted in little change in cancer mortality rates in the past 35 years. They

TABLE I. Percentage of Physicians Who Followed and Recalled Selected American Cancer Society (ACS) Guidelines

ACS Guideline	Physicians Following ACS Guideline (N = 120)	Physicians Correctly Recalling ACS Guideline (N = 57)*
Chest film screening	10% (4.6-15.4)†	11% (2.9-19.1)
Stool guaiac test	48% (39.1-56.9)	2% (0-5.6)
Sigmoidoscopy	41% (32.2-49.8)	4% (0-9.1)
Monthly breast self-examination	48% (39.1-56.9)	38% (25.4-50.6)
Mammography	20% (12.8-27.2)	5% (0-10.7)

* Only asked of physicians who expressed at least "slight" familiarity with ACS guidelines.
† 95% confidence interval.

concluded that prevention may offer greater promise than treatment for reduction of cancer mortality.

Data from this survey reveal that, in regard to the three most common causes of cancer mortality in the United States, a substantial proportion of physicians interviewed do not comply with ACS guidelines for the cancer-related checkup. In particular, only 20% of physicians recommended screening mammography, 41% screening sigmoidoscopy, and 48% stool guaiac test. In addition, 49% recommended chest films for all patients, and 90% recommended chest films for patients who smoke as screening methods for the early detection of lung cancer, although the ACS specifically advised against this test.

One must consider a variety of factors when attempting to determine the basis for physician decisions regarding preventive tests or procedures. Each test represents a potential burden in terms of time, cost, risk of false positives (and possible subsequent unnecessary work-up), patient anxiety and/or discomfort, and patient safety. If the potential yield of the test does not outweigh these burdens (in the physician's judgment), it is unlikely that he or she will recommend the test. The practices of colleagues and the extent of patient demand for a particular test also have an impact on the decision to recommend a test or procedure.

The extent to which physicians are aware of and knowledgeable about guidelines for prevention is also of obvious import. While almost three out of four physicians cited the ACS as one of their sources of information for the evaluation of asymptomatic patients, relatively few appeared to be knowledgeable of the content of the guidelines. In many instances, failure of physicians to comply with ACS guidelines may represent less a measured judgment of risks and benefits than a lack of awareness of the guidelines' contents.

Nationally recognized guidelines for cancer prevention are undoubtedly useful to primary care physicians. Approximately four out of five physicians supported the need for a task force to "clarify recommendations" for preventive medicine. Scientific consensus on the indication for preventive tests and procedures may not be sufficient to ensure their adoption by primary care physicians, however. If guidelines are not clearly communicated and promoted to the target audience, if they do not address specific concerns of practitioners and patients (eg, lack of health insurance reimbursement for preventive tests or procedures, risk of radiation from screening mammography), or if they are not presented in a format conducive to practical implementation, their value may be considerably diminished.

This study suggests that ACS guidelines have not, in many instances, been adequately diffused into the community of primary care physicians in New York City. Achieving more widespread compliance may require greater effort in disseminating the guidelines' content, educating physicians as to their value, and continued promotion of their use.

Acknowledgments. The authors thank the New York City Division of the American Cancer Society and Don Charbonneau, former Director of Professional Education at the Division, for their assistance. Mrs Raie Bauman and Ms Lesley Field, RN, used their expert interviewing skills, Athilia Siegmann, MPhil, provided technical assistance, and Peter Messeri, PhD, offered advice on statistical analysis. We also thank the 120 New York City physicians who graciously volunteered their time to participate in the study.

REFERENCES

1. American Cancer Society: Guidelines for the cancer-related checkup. *Cancer* 1980; 30:194-240.
2. Canadian Task Force on the Periodic Health Examination: The periodic health examination. *Can Med Assoc J* 1979; 121:1193-1254.
3. Frame PS, Carlson SJ: A critical review of periodic health screening using specific screening criteria. *J Fam Pract* 1975; 2:29-36, 123-129, 189-194, 283-289.
4. Breslow L, Somers AR: The lifetime health-monitoring program. A practical approach to preventive medicine. *N Engl J Med* 1977; 296:601-608.
5. American College of Physicians, Medical Practice Committee: Periodic health examination: A guide for designing individualized preventive health care in the asymptomatic patient. *Ann Intern Med* 1981; 95:729-732.
6. Council on Scientific Affairs: Medical evaluations of healthy persons. *JAMA* 1983; 249:1626-1633.
7. Lawrence RS, Mickalide AD: Preventive services in clinical practice: Designing the periodic health examination. *JAMA* 1987; 257:2205-2207 .
8. Battista RN: Adult cancer prevention in primary care: Patterns of practice in Québec. *Am J Public Health* 1983; 73:1036-1039.
9. American Cancer Society: Survey of physicians' attitudes and practices in early cancer detection. *Cancer* 1985; 35:197-213.
10. Gemson DH, Elinson J: Prevention in primary care: Variability in physician practice patterns in New York City. *Am J Prev Med* 1986; 2:226-234.
11. American Cancer Society: Cancer statistics, 1986. *Cancer* 1986; 36:5-37.
12. American Cancer Society: Mammography guidelines 1983: Background statement and update of cancer-related checkup guidelines for breast cancer detection in asymptomatic women age 40 to 49. *Cancer* 1983; 33:255.
13. Bailar JC, Smith EM: Progress against cancer? *N Engl J Med* 1986; 314:1226-1232.

Deprenyl versus placebo in Parkinson disease: A double-blind study

ABRAHAM N. LIEBERMAN, MD; GOVINDAN GOPINATHAN, MD; ANDREAS NEOPHYTIDES, MD; SON HOO FOO, MD

ABSTRACT. In order to evaluate deprenyl's efficacy and safety, the drug was studied in 33 patients with Parkinson disease (PD), in a randomized double-blind parallel manner. All patients were no longer responding satisfactorily to levodopa (combined with carbidopa in Sinemet™). Seventeen patients received deprenyl (10 mg/day) in addition to levodopa, and 16 patients received placebo in addition to levodopa. Patients were evaluated on a modified Columbia University Scale. Patients kept 24-hour diaries to help in assessing daily fluctuations in performance. An attempt was made to decrease the dose of levodopa in all patients.

Age, duration of PD, duration of Sinemet™ therapy, and presence of "wearing off" phenomena were comparable in both groups. All patients completed the eight-week study. Compliance was assessed by measuring inhibition of monoamine oxidase (MAO) B in platelets. Patients on deprenyl experienced a significant ($p \leq 0.05$) 22% decrease in symptoms, a significant ($p < 0.05$) 17.4% decrease in signs, and a significant ($p \leq 0.025$) 21% decrease in levodopa use. There were no significant changes in the number of hours "on." Adverse effects were minimal and were similar to those associated with levodopa. We conclude that deprenyl is an effective and safe antiparkinson drug.

(NY State J Med 1987; 87:646–649)

There is great interest in using deprenyl in the treatment of Parkinson disease. Deprenyl is a selective inhibitor of monoamine oxidase B.[1,2] There are two kinds of MAO: type A, outside the brain; and type B, inside the brain and in platelets.[3] The MAO inhibitors available in the United States are nonselective and inhibit both MAO A and MAO B. As a result of inhibition of MAO A, tyramine, the dietary amine, gains access to the circulation. Because an average meal of some foods (especially cheeses) has enough tyramine to markedly elevate blood pressure, ingestion of such foods by patients receiving nonselective MAO inhibitors may lead to a hypertensive crisis.[4] However, inhibition of MAO B does not result in hypertensive effects, and specific dietary restrictions are not necessary.

Deprenyl has been shown to be a useful adjuvant to levodopa treatment. Originally, in an open study, deprenyl given with levodopa was shown to potentiate the antiparkinson effect of levodopa.[5] In subsequent studies, both open and double-blind, deprenyl given with levodopa was shown to either potentiate the antiparkinson effect of levodopa, or decrease the severity of the diurnal fluctuations in performance (the "wearing off" effect), or both.[6–9] Although deprenyl, when added to levodopa, has only a moderate antiparkinson effect, it is easy to use and is relatively free of adverse effects. Given the paucity of drugs that are available to treat patients with Parkinson disease, especially patients who are no longer responding satisfactorily to levodopa, there has always been interest in gaining Food and Drug Administration (FDA) approval for deprenyl.

Recently there has been heightened interest in this, centering on the possibility that deprenyl, used by itself or combined with levodopa, may delay or prevent the progression of PD. In animal studies it has been shown that deprenyl blocks the oxidation of 4-methyl-4-phenyl 1,2,3,6 tetrahydropyridine (MPTP) to its toxic metabolite 1-methyl-4-phenyl-pyridine (MPP+) and prevents the development of MPTP-induced parkinsonism.[10] If the cause of idiopathic PD is related to the formation of an MPP+-like substance, deprenyl may delay the progression of PD.[11–14] Indeed, in one retrospective study, Parkinson disease patients on deprenyl lived longer than PD patients not on deprenyl.[15,16]

Presently, deprenyl is available only on an experimental basis in the United States. Prior to being approved for use as an adjuvant to levodopa or as a means of slowing the progression of PD, deprenyl will have to demonstrate its antiparkinson efficacy in a double-blind trial.

PATIENTS AND METHODS

This study was a double-blind, randomized, parallel, placebo-controlled outpatient study of eight weeks' duration. During the first two weeks, eligible patients remained on their baseline antiparkinson drugs. The patients were evaluated by the same neurologist on two separate occasions while they were in an "on" period. During the baseline period, all patients underwent an electrocardiogram, an electroencephalogram, a blood count, and tests of renal and liver function, as well as a chest radiograph. Patients who met the entrance criteria received medication kits containing either deprenyl or placebo. Patients were evaluated using a Symptom and Sign Disability Scale that is a modification of the Columbia University Scale. The maximum weighted score for the Symptom Disability Scale was 100. The maximum weighted score for the Sign Disability Scale was 336. All patients

---

From the New York University School of Medicine, New York, and the New York Infirmary-Beckman Downtown Medical Center, New York.

Address correspondence to Dr Lieberman, 650 First Ave, New York, NY 10016.

were instructed in evaluating themselves. Prior to each visit, patients kept a 24-hour diary card on which they noted every hour whether they were in an "on" or an "off" period. Each card was checked for accuracy and consistency.

On completion of the study, the "blinded" investigator made a global determination as to whether or not a patient's condition had improved. Because of the difficulty in evaluating the responses to any treatment in PD patients who exhibit diurnal fluctuations in performance, a conscious attempt was made in all patients to lower the dose of levodopa (which was combined with carbidopa in Sinemet™). Before this study, previous attempts to lower the dose of levodopa by at least 20% in these patients resulted in increasing parkinsonian symptoms and signs. The ability to significantly lower the dose of levodopa, without compromising efficacy, is one of the more objective pieces of evidence in assessing a drug's effect.

To be eligible for the study the patients' stage of PD had to be 2, 3, or 4, as assessed during their "on" period using the Hoehn and Yahr scale. In addition, patients had to be undergoing treatment with Sinemet. They had to be having a less than satisfactory response to treatment. They also had to be on a stable dose of Sinemet for at least the previous three months.

Patients on anticholinergic drugs were not excluded from the study. However, the following patients were excluded: patients who were on amantadine, dopamine agonists, tricyclic antidepressants or trazodone; patients with rapidly cycling, unpredictable "on/off" phenomena; and patients with severe or unstable heart disease. (Patients in whom there was a question of heart disease were evaluated by a cardiologist.)

Patients meeting all of the entry criteria were assigned after two weeks to either deprenyl (5 mg twice a day) or placebo treatment, according to a computer-generated randomization schedule. This information was not available to the investigators. All patients signed an informed consent form. During the six weeks of the study, after the two-week baseline period, an attempt was made to lower each patient's dose of Sinemet. For purposes of evaluation, the patient's lowest Sign Disability Score (and associated Symptom Disability Score) in the pretreatment period was compared to the lowest Sign and Symptom Disability Score in the last two weeks of the study when the dose of Sinemet had been stabilized. The lowest Sign Disability Score was used because this represented the patient's score when in an "on" period. Thus a pretreatment "on" period could be compared to a posttreatment "on" period.

Patients were questioned about adverse effects such as dyskinesias, dystonias, changes in higher intellectual functioning, and depression. These adverse effects were evaluated on a weighted scale of 106. A comparison of the patients' parkinsonian symptoms, signs, hours "on," adverse effects, levodopa dose, and the observer's global rating was made in the first group before and after treatment with deprenyl and in the second group before and after treatment with placebo. In this way, each patient served as his or her own control. In addition, a univariate analysis of covariance was made between the two groups of patients' symptoms, signs, hours "on," adverse effects, and levodopa dose before treatment, and the two groups of patients' symptoms, signs, hours "on," adverse effects, and levodopa dose after treatment. Compliance was checked at each visit by counting the unused drug (or placebo). Compliance was also checked by measuring the level of platelet MAO B activity. (A unit of activity is a nanomole of tryptamine deaminated/hour/mg of platelet.)

RESULTS

Seventeen patients were randomly assigned to the deprenyl group and 16 to the placebo group. All patients completed the eight-week trial. Both groups of patients were comparable in age, sex, duration of PD, Sinemet dose, duration of levodopa treatment, disease severity, and prior treatment with bromocriptine or other dopamine agonists. Although the patients given deprenyl were younger, this difference was not significant (Table I).

The 17 patients who were randomly assigned to the deprenyl group experienced a significant ($p \leq 0.05$) 22% decrease in their parkinsonian symptoms; a significant ($p \leq 0.05$) 17.4% decrease in their parkinsonian signs; and a significant ($p \leq 0.025$) 21% decrease in their Sinemet dose. The 17 patients did not as a group experience an improvement in the number of hours that they were "on." Adverse effects were not increased over baseline levels. Overall, the conditions of 12 of the 17 patients (71%) were judged to have improved. Although there was no increase in the number of hours "on," patients reported that their dose of levodopa lasted longer, that the transitions between their "on" and "off" periods were less abrupt, that the "on" periods were better, and that the "off" periods were less severe. Although these subjective responses could not be quantified, they were useful in arriving at the global assessment.

The dose of deprenyl was 10 mg/day (Table II). The activity of platelet MAO B as determined on the patients' last visit was 0.13 units ± 0.02 standard error of the mean (SEM) (normal range, 1.0–8.0 units). The 16 patients on placebo did not have a significant decrease in symptoms, signs, Sinemet dose, hours "on," or adverse effects. Overall, the conditions of two of the 16 patients (12.5%) were judged by the observer to have improved.

When the univariate analysis of covariance was made between the baseline scores and Sinemet doses of the patients on deprenyl and the patients on placebo, none of the p values for symptoms, signs, hours "on," Sinemet dose, or adverse effects were significant. When the univariate analysis of covariance was made between the final scores and the Sinemet dose of the patients on deprenyl with the patients on placebo, only the Sinemet dose was significantly ($p \leq 0.01$) less in the deprenyl group than in the placebo group. The parkinsonian sign score was improved in the deprenyl group and approached but did not achieve significance ($p \leq 0.09$).

Overall, 12 of 17 patients showed improvement on deprenyl, whereas only two of 16 patients showed improvement on placebo. It is conceivable that the improvement on placebo was secondary to increased compliance or the placebo effect, but this also should have been a factor in the deprenyl group and does not explain the difference between the two groups. The improvement that occurred in the two patients on placebo reinforces the necessity for such double-blind studies. The mean activity of platelet MAO B for the patients who were on placebo was 1.2 units ± 0.13 SEM. The mean activity of platelet MAO B for this group was significantly more ($p \leq 0.005$) than the mean activity of platelet MAO B in the patients who were treated with deprenyl (0.13 units ± 0.02 SEM). These data suggest that the patients who were in the deprenyl group actually took the drug.

There were no differences between the deprenyl or placebo groups in adverse effects. On completion of the study, three of the 17 de-

TABLE I. Characteristics of Patients in Deprenyl and Placebo Groups

Treatment Group (N)		Age (yr)	PD Duration (yr)	Levodopa Duration (yr)	Stage	Sex	Bromocriptine Prior Treatment	Other Agonists	Wearing Off
Deprenyl	Mean	60.6	7.9	7.2	2.2	5 F	10 patients	4 patients	16 patients
(17)	Range	(42–69)	(1–19)	(1–16)	(1–3)	12 M			
Placebo	Mean	65.6	7.1	6.8	2.4	6 F	10 patients	3 patients	14 patients
(16)	Range	(49–74)	(2–14)	(1–14)	(1–4)	10 M			

TABLE II. Analysis of Covariance of Deprenyl Group Versus Placebo Group

		Deprenyl	Sinemet*	Symptom Score[†]	Sign Score[‡]	Hours "On"	Adverse Score[§]
Before	Mean	0	897	30.2	112.8	9.0	18.1
	(±SEM)[‖]		(±144)	(±4.5)	(±11.9)	(±1.7)	(±4.2)
After	Mean	10 mg	706	23.5	93.2	11.1	15.4
	(±SEM)		(±105)	(±3.3)	(±11.6)	(±1.5)	(±3.1)
	Change		−21%	−22%	−17.4%	+23%	−15%
			$p \leq 0.025$	$p \leq 0.05$	$p \leq 0.05$	NS[¶]	NS

		Placebo	Sinemet	Symptom Score	Sign Score	Hours "On"	Adverse Score
Before	Mean		906	30.7	118.9	8.5	11.9
	(±SEM)		(±117)	(±3.9)	(±10.5)	(±1.6)	(±3.8)
After	Mean		864	26.3	111.8	9.3	11.8
	(±SEM)		(±101)	(± 4.2)	(± 14.5)	(±18)	(± 3.5)
	Change		−5%	−14%	−7.1%		
			NS	NS	NS	NS	NS

* Mg levodopa.
[†] Maximum symptom score = 100.
[‡] Maximum sign score = 336.
[§] Maximum adverse score = 106.
[‖] SEM = standard error of the mean.
[¶] NS = not significant.

prenyl-treated patients discontinued the drug. Two patients discontinued the drug because they had not achieved any benefit from deprenyl. Only one patient discontinued the drug because of an adverse effect. This patient had a mild dementia, and during the course of treatment with deprenyl became more confused. On discontinuation of deprenyl, the patient returned to his baseline state. Among the 16 patients who were on placebo, 15 elected to go on deprenyl. The only patient who did not go on deprenyl was a patient who had a mild dementia and who had experienced increased confusion during the study.

DISCUSSION

The motor deficit in PD is related to a deficiency in striatal dopamine that results from degeneration of the dopamine-producing neurons in the substantia nigra. As PD advances, the degeneration of the nigral neurons may proceed to the point where the remaining nigral neurons cannot synthesize sufficient dopamine to stimulate the striatal dopamine receptors. This progressive nigral degeneration may be responsible for the increasing motor disability. The remaining nigral neurons may not only synthesize insufficient dopamine, but the synthesized dopamine may be unable to be stored within the terminals of the remaining nigral neurons. The dopamine that is synthesized may be delivered in an inappropriately synchronized manner to the striatal dopa receptors, or some of the striatal dopamine receptors may be subsensitive to the effects of dopamine.

One or more of the above mechanisms may be responsible in part for the increased parkinsonian symptoms and signs and the fluctuating response to levodopa. Deprenyl may be effective in many of these patients, because it increases striatal dopamine levels.[1,2,16,17] Deprenyl may increase striatal dopamine by inhibiting the MAO B that is present in the mitochondria of the migral neurons.[1,2,16,17] This inhibition would result in an increase in the intraneural pool of dopamine. While increases in striatal dopamine may result in improvement in parkinsonian symptoms and signs, it is less clear how such increases may result in fewer diurnal fluctuations. Indeed, in this study we were unable to document a reduction in diurnal fluctuations, although this has been documented in the past.[6,8] Perhaps the increased striatal dopamine is able to exert a longer as well as a better antiparkinson effect.

The addition of deprenyl permits a reduction in the dose of levodopa. However, adding deprenyl is not merely the equivalent of adding more levodopa. Deprenyl and a lower dose of levodopa may be more beneficial than just more levodopa alone, because deprenyl and levodopa given together may result in an increase in intracerebral dopamine (similar to that achieved with the addition of more levodopa) but with less intracerebral 3-0-methyldopa. While increased formation of 3-0-methyldopa, the major metabolite of levodopa, may reduce the absorption of levodopa from the gastrointestinal tract and reduce levodopa's entry into the brain,[18] it is also conceivable that in the brain 3-0-methyldopa may have a specific anti-dopamine effect. In addition, deprenyl may block the re-uptake of extraneuronal dopamine.[2] This can result in a greater concentration of extraneuronal dopamine than can be achieved by adding more levodopa. Also, deprenyl may release intraneuronal dopamine.[19] As intraneuronal dopamine stores are increased because of inhibition of MAO B and as extraneuronal dopamine re-uptake is blocked by deprenyl, the increased release of dopamine may result in an even greater antiparkinson effect. Finally, because deprenyl blocks the oxidation of dopamine to hydrogen peroxide, a free radical, there may be fewer reactions involving nucleophilic attacks on cellular components by such electrophilic byproducts of dopamine. The formation of such free radicals might itself result in worsening parkinsonian symptoms and signs. Such worsening would be prevented by deprenyl but increased by additional levodopa.

The highest activities of MAO A and B are found in the striatum (caudate and putamen), globus pallidus, substantia nigra, hypothalamus, and mammilary bodies. Brains obtained at autopsy from deprenyl-treated PD patients show that whereas MAO B is fully inhibited by therapeutic doses of deprenyl (10 mg/day), substantial MAO A is still present.[16] This may be interpreted to mean that an even greater antiparkinsonian effect could be achieved if an MAO A inhibitor were added. In addition, if the cause of idiopathic PD is related to the formation of an MPP+-like substance, and if deprenyl, by inhibiting the formation of this MPP+-like substance, slows the progression of the disease, then a compound that inhibits both MAO A and B may be even more effective. However, any added benefit from using a compound that inhibits both MAO A and B would have to be balanced by the increased risks of adverse effects such as hypertensive crises. Although autopsy data reveal that MAO B is fully inhibited by therapeutic doses of deprenyl (10 mg/day), our own unpublished data obtained by using C-11-labelled deprenyl with positron emission tomography (PET) indicate that with current therapeutic doses of deprenyl, inhibition of brain MAO B is incomplete. Although a greater antiparkinsonian effect could possibly be achieved by using a compound that inhibits MAO A and B, it is also conceivable that a better antiparkinsonian effect with relatively few adverse effects may be achieved by using a higher dose of deprenyl (20 mg/day).

Acknowledgments. This project was sponsored in part by the Leon Lowenstein Foundation, the American Parkinson Disease Association, and Somerset Pharmaceuticals. The authors thank Ms Linda Chin, RN, and Ms Kathleen Faridazar for their help in organizing and conducting the study and Drs Peter Lefkow and Peter Pasternack.

REFERENCES

1. Knoll J: The possible mechanisms of action of (-)deprenyl in Parkinson's disease. *J Neural Transm* 1978; 43:117-198.
2. Riederer P, Youdim MB, Rausch WD, et al: On the mode of action of L-deprenyl in the human central nervous system. *J Neural Transm* 1978; 43:217-226.
3. Youdim MB: The active centers of monoamine oxidase types "A" and "B": Binding with (14C)-clorgyline and (14C)-deprenyl. *J Neural Transm* 1978; 43:199-208.
4. Sandler M, Glover V, Ashford A, et al: Absence of "cheese effect" during deprenyl therapy: Some recent studies. *J Neural Transm* 1978; 43:209-215.
5. Birkmayer W, Riederer P, Ambroze L, et al: Implications of combined treatment with "Madopar" and L-deprenyl in Parkinson's disease. A long-term study. *Lancet* 1977; 1:439-443.
6. Schachter M, Marsden CD, Parkes JD, et al: Deprenyl in the management of response fluctuations in patients with Parkinson's disease on levodopa. *J Neurol Neurosurg Psychiatry* 1980; 43:1016-1021.
7. Lieberman AN, Gopinathan G, Neophytides A: Deprenyl in the treatment of Parkinson's disease: A specific type B monoamine oxidase inhibitor. *NY State J Med* 1984; 84:13-16.
8. Rinne UK: Deprenyl (selegiline) in the treatment of Parkinson's disease. *Acta Neurol Scand* 1983; 95(suppl):107-111.
9. Yahr MD, Mendoza MR, Moros D: Treatment of Parkinson's disease in early and late phases. Use of pharmacological agents with special reference to deprenyl (selegiline). *Acta Neurol Scand* 1983; 95(suppl):95-102.
10. Heikkila RE, Manzino L, Cabbat FA: Protection against the dopaminergic neurotoxicity of 1-methyl-4-phenyl-1,2,5,6 tetrahydropyridine by monoamine oxidase inhibitors. *Nature* 1984; 311:467-469.
11. Langston JR, Ballard P, Tetrud JW: I. Chronic parkinsonism in humans due to a product of meperidine-analog synthesis. *Science* 1983; 219:979-980.
12. Burns RS, Lewitt PA, Ebert MH, et al: The clinical syndrome of striatal dopamine deficiency. Parkinsonism induced by 1-methyl-4-phenyl-1,2,3,6 tetrahydropyridine (MPTP). *N Engl J Med* 1985; 312:1418-1421.
13. Javitch JA, D'Amato RJ, Strittmatter SM, et al: Parkinsonism inducing neurotoxin, N-methyl-4-phenyl-1,2,3,6-tetrahydropyridine: Characterization and localization of receptor binding sites in rat and human brain. *Proc Natl Acad Sci USA* 1984; 81:4591-4595.
14. Javitch JA, D'Amato RJ, Strittmatter SM, et al: Parkinsonism-inducing neurotoxin, N-methyl-4-phenyl-1,2,3,6-tetrahydropyridine: Uptake of the metabolite, N-methyl-4-phenylpyridine by dopamine neurons explains selective toxicity. *Proc Natl Acad Sci USA* 1985; 82:2173-2177.
15. Birkmayer W, Knoll J, Riederer P: Increased life expectancy resulting from addition of L-deprenyl to Madopar treatment in Parkinson's disease: A long-term study. *J Neural Transm* 1985; 64:113-127.
16. Riederer P, Jellinger K: Neurochemical insights into monoamine oxidase inhibitors with special reference to deprenyl (selegiline). *Acta Neurol Scand* 1983; 95(suppl):43-55.
17. O'Carroll AM, Fowler CJ, Phillips JP, et al: The deamination of dopamine by human brain monoamine oxidase. Specificity for the two enzyme forms in seven brain regions. *Nauyn Schmiedebergs Arch Pharmacol* 1983; 322:198-202.
18. Reches A, Fahn S: 3-0-methyldopa blocks dopa metabolism in rat corpus striatum. *Ann Neurol* 1982; 12:267-271.
19. Karoum F, Chuang LW, Eisler T: Metabolism of (-)deprenyl to amphetamine and methamphetamine may be responsible for deprenyl's therapeutic benefit: A biochemical assessment. *Neurology* 1982; 32:503-509.

REVIEW ARTICLE

Lacunes: The pervasive strokes

Emilio D. Soria, md; Edward J. Fine, md; Margaret W. Paroski, md

Lacunes are small cerebral infarcts located in the midline basal areas of the cerebrum and brain stem, including the basal ganglia, internal capsule, thalamus, and pons. They result from the occlusion of a particular subset of arteries, the penetrating branches that arise from the large cerebral arteries[1] (Table I). They constitute the most common anatomopathologic stroke. Asymptomatic lacunes are frequently seen as incidental findings at autopsy, where they outnumber all other varieties of cerebrovascular lesions combined. In a sample of 1,042 brains examined by C. M. Fisher,[2] one or more lacunes were present in 11% of the cases (Fig 1). Even when considering the symptomatic cases only, one finds that lacunes comprise 19% of all strokes.[3]

What actually constitutes the dimensions of a lacune varies from author to author, each setting an arbitrary upper limit at different levels. It is preferable to leave the term "small" unqualified; lacunes range in size along a spectrum, and some of the larger ones may reach a diameter of 2 cm or more. At times, much larger infarcts may take place in the deep midline areas of the brain. Some authors have referred to these lesions as giant lacunes. These sizable infarcts are most likely due to occlusion of the parent arteries of the circle of Willis rather than to penetrating artery disease and, therefore, they should not be classified as true lacunes (Fig 2).

Vascular Pathology of Lacunes

The processes that lead to occlusion of penetrating vessels can be intrinsic to the arterial wall or can be secondary to disorders that originate elsewhere, which indirectly lead to obstruction of the lumen[4] (Fig 3).

Primary Occlusion of Penetrating Branches. The specific entity that causes lacunes is the hypertensive angiopathy of small arteries and arterioles characterized by microatheromatosis and lipohyalinosis (Fig 4).

In the conventional arteriosclerosis unrelated to hypertension, the plaques are confined to the large cerebral vessels, mostly in areas of bifurcation of extracranial arteries and in the stem of the large circumferential arteries. In chronic hypertension, however, multiple minute atheromatous foci are found in small arteries. These vessels show marked tortuosity and, with specific stains, they reveal amorphous lipid deposits in the intima. These microatheromatous plaques on the walls of the penetrating branches are a prevailing cause of lacunes by the occlusive effect in the lumina.[5]

TABLE I. Anatomical Characteristics of Penetrating Branches

Small caliber (40 to 500 microns in diameter)
Derivation directly from a much larger parent vessel, without mid-size intermediate arteries
Lack of gradual reduction of pressure, making them particularly susceptible to hypertensive damage
Origin at right angles from parent vessel, perforating the brain parenchyma in a straight course
End arteries: meager or no collateral circulation
Relatively uncomplicated configuration: scanty branching
Origin from the circle of Willis, basilar artery, and proximal segments of major cerebral arteries
Clustered in tufts irrigating deep, midline, basal areas of the cerebrum and brain stem

In lipohyalinosis, the changes are predominantly seen in very small arteries and arterioles (50–200 microns in di-

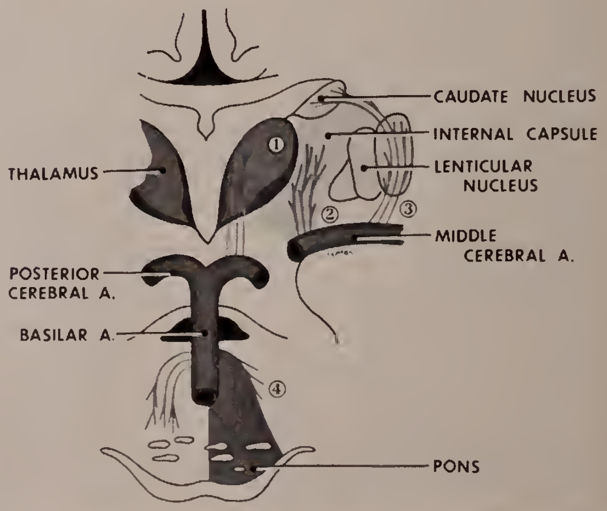


FIGURE 1. Lacunes are predominantly located in the territories of four penetrating groups: 1) thalamoperforating arteries; 2) medial lenticulostriate arteries; 3) lateral lenticulostriate arteries; 4) pontine paramedian arteries.

From the Department of Neurology, Veterans Administration Medical Center, Buffalo, NY.

Address correspondence to Dr Soria, Assistant Professor of Neurology, Veterans Administration Medical Center, 3495 Bailey Ave, Buffalo, NY 14215.

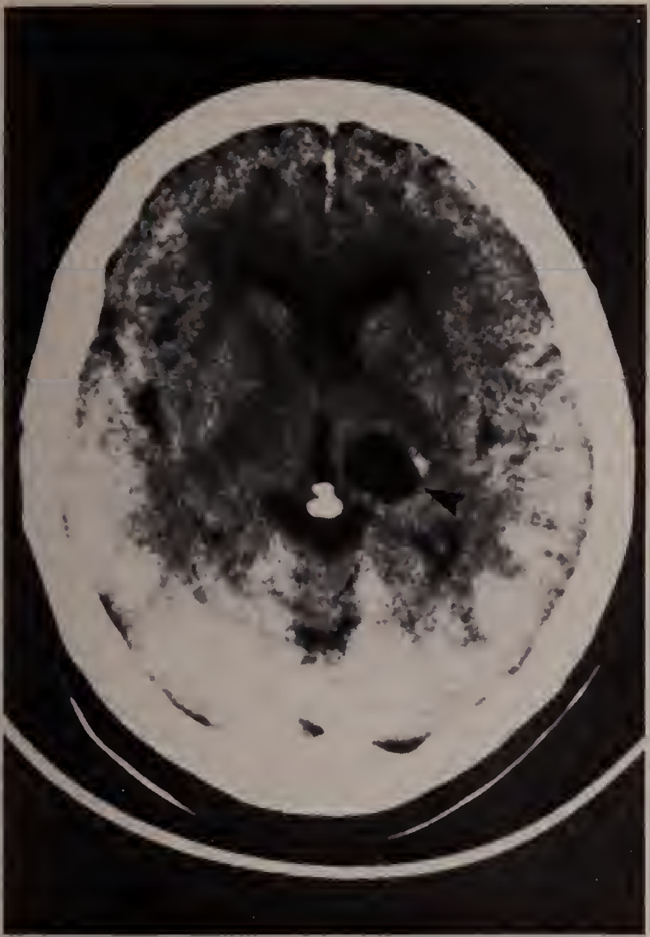


FIGURE 2. The term "giant lacune" was coined for the lesions that exceeded the size of conventional lacunes. A giant lacune is located in the pulvinar nucleus of the thalamus (courtesy of Dr G. J. Alker, Buffalo, NY).

ameter). Hyaline change occurs in the subendothelial region and in the media of the vessels, with eosinophilic homogeneous deposits in the connective tissue. The vessel becomes thickened and fibrotic and prone to thrombotic occlusion.[6]

Fibrinoid necrosis, a closely related entity, is seen in patients with extremely high blood pressure in hypertensive crisis. Its microscopic appearance is similar to lipohyalinosis, but in fibrinoid necrosis, a large deposition of fibrin takes place in the arterial wall. These deposits react with stains specific for fibrin, such as phosphotungstic acid-hematoxylin (PTAH).[7] Microatheroma, lipohyalinosis, and fibrinoid necrosis are probably the result of the same process, ranging in severity along a continuum.

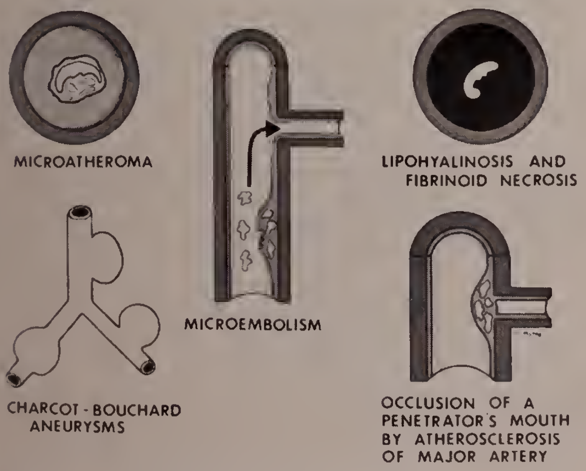

MICROATHEROMA
LIPOHYALINOSIS AND FIBRINOID NECROSIS
MICROEMBOLISM
CHARCOT - BOUCHARD ANEURYSMS
OCCLUSION OF A PENETRATOR'S MOUTH BY ATHEROSCLEROSIS OF MAJOR ARTERY

FIGURE 3. Vascular pathology underlying lacunes.

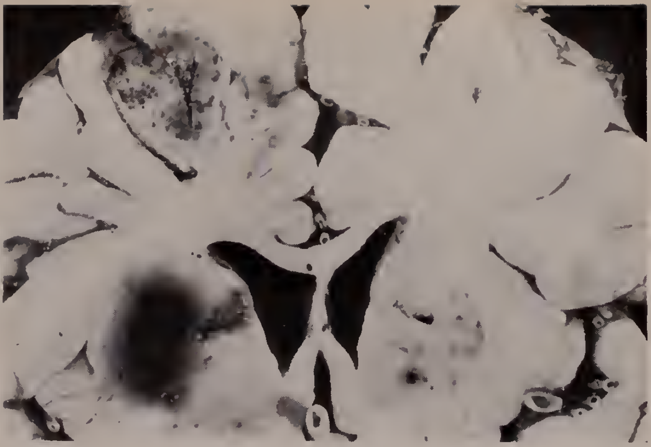


FIGURE 4. The broad spectrum of hypertensive cerebrovascular complications is illustrated in this brain. Several capsular and ganglionic lacunes are associated with a putaminal hemorrhage. A large superficial infarct, with some hemorrhagic components, is present as well (courtesy of Dr W. A. Olzewski, Buffalo, NY).

Miliary aneurysms were first described by Charcot and Bouchard[8] in 1868. They are commonly found in chronic hypertension and usually affect those arteries also disfigured by microatheroma and lipohyalinosis. Their exact role in the development of lacunes is not well understood.

Secondary Occlusion of Penetrating Branches. If the only abnormality affecting these arteries were the hypertensive microangiopathies previously described, there would be no difficulty in assigning lacunes to a single disease process: hypertension. However, the matter is complicated by extrinsic problems that secondarily involve the penetrators. In some cases, a luminal lesion in the major parental vessel may by chance occlude the mouth of the penetrating branch. An infarct will result in this region, despite the otherwise clean walls of the penetrating artery.[9]

Several cases of lacunar infarct, conscientiously studied at postmortem with serial cuts, failed to show any arterial abnormality. The inferred conclusion was that microembolization (artery-to-artery or heart-to-artery), with disintegration of the embolus, was the plausible cause of the stroke.[10]

CLINICAL PRESENTATION OF LACUNES

The development of lacunar strokes differs from that of the large ischemic cerebrovascular accidents. The onset is typically insidious, reaching the point of maximal deficit rather leisurely. In a large proportion of cases, the stroke evolves for one or two days. The plateau period is short, and thereafter the lacunar strokes tend to improve more rapidly and with less permanent residual effects than the large infarcts that show a similar severity of initial deficits.

Lacunar infarcts can present as a transient neurologic deficit with complete resolution. Such warning spells are relatively common in patients with the arteropathy underlying lacunes.

Another form of presentation, actually of nonpresentation, is the asymptomatic lacune or the lacune with such mild symptomatology as to be neglected by the patient. These silent lacunes, by all accounts the commonest, are discovered later, at necropsy, as an unpredicted finding.[2]

Three clinical attributes are worthy of consideration: the selective involvement of only one neurologic function; the characteristic sparing of certain functions; and the lack of signs of diffuse cerebral involvement.

Lacunar infarcts typically produce selective involvement of a single cerebral function, sparing other modalities. For example, lacunar infarcts can produce motor paralysis without a parallel sensory involvement or hemisensory syndromes without motor weakness. This is not the case with the large ischemic surface strokes. It would be extremely unlikely that a large superficial cerebrovascular accident would run extensively along the course of the cortical-subcortical motor areas, leaving the adjacent sensory areas unharmed, and vice versa.

The second attribute under consideration is equally important. As essential for the diagnosis as the positive findings are the absent ones: Certain symptoms are so infrequent in lacunar infarcts as to make the diagnosis quite suspect if present. At the supratentorial level, lacunes are not commonplace in the cortical gray matter, the visual radiations, and centrum semiovale. In the brain stem, they favor the basis pontis and avoid the midbrain and medulla. It follows that symptoms attributable to damage of any of these areas argue against a lacune. In general, lacunes will not produce involvement of any of the so-called higher cortical functions of either the dominant or the nondominant hemisphere. The presence of aphasias, agnosias, and apraxias would almost always rule out a lacunar infarct. Seizures, being cortical events, are also distinctly absent. The sparing of visual radiations eliminates lacunes as a cause of hemianopic visual deficits. Among brain stem symptoms, diplopia and other signs of disconjugate ocular motility are occasionally seen in lacunes, but ordinarily are not present in the absence of a definite hemiplegia.

The third criterion of diagnosis, the lack of diffuse or generalized cerebral involvement, is dependent on the diminutive size of most lacunes. Lacunes do not disturb the brain as a whole and, accordingly, nonfocal neurologic symptoms such as headache and mental confusion are only rarely encountered, and often can be attributed to an intercurrent, unrelated event.

For the codification of specific lacunar syndromes, we are indebted to the work of C. M. Fisher,[2,5] who, in the 1960s, painstakingly catalogued most of the common forms and labeled them with a descriptive nomenclature. Since then, the list of clinical syndromes attributable to lacunes has increased. Many of these symptoms are mere variations, slightly divergent from a few basic patterns: pure motor stroke and its variants, pure sensory stroke, hemichorea-hemiballismus, ataxic hemiparesis, dysarthria-clumsy hand, homolateral ataxia with crural paresis, and a few others[11,12] (Table II).

Pure Motor Stroke. As its name indicates, an unmixed motor weakness of a hemibody occurs as the sole deficit, without associated sensory, visual, or intellectual impairment.[13] Pure motor stroke (PMS) can result from lacunes at various levels along the pyramidal tract, but the usual locations are either the posterior limb of the internal capsule or the basis pontis. Despite the topographic variability, the clinical pictures are practically identical, but, in general, the more caudal the location of the lesion, the less the involvement of the face. As the lesion proceeds caudally, the clinical picture will gradually merge with the syndrome of pure motor stroke sparing the face, which is a common type of brain stem lacune.

A gaze paresis coupled with PMS has an excellent localizing value based on the gaze preference; the eyes look away from the hemiparetic limbs with a capsular lesion and towards the hemiparesis in the pontine counterpart.

TABLE II. Lacunar Syndromes in Relation to Location

Territory	Penetrating Arteries	Syndromes
Basal ganglia	Lateral lenticulostriate arteries	Often asymptomatic Hemichorea-hemiballismus*
Internal capsule	Medial and lateral lenticulostriate arteries	Pure motor stroke† Dysarthria-clumsy hand‡ Homolateral ataxia and crural paresis*
Thalamus	Thalamoperforating arteries	Sensorimotor stroke‡ Pure sensory stroke‡ Thalamic pain§ Thalamic dementia*
Midbrain	Mesencephalic paramedian arteries	Top of the basilar syndrome§ Weber syndrome* Claude syndrome*
Pons	Pontine paramedian arteries	Pure motor stroke† Pure motor stroke sparing the face‡ Dysarthria-clumsy hand‡ Ataxic hemiparesis† Homolateral ataxia and crural paresis‡ Pure dysarthria*

* Very rare.
† Very common.
‡ Common.
§ Rare.

Many patients with PMS will present with sensory complaints (numbness, lack of feeling, coldness, etc) which are not substantiated by the sensory examination. These patients may actually be referring, with ambiguous layman terminology, to the clumsiness and weakness that they are experiencing.

Dysarthria-Clumsy Hand Syndrome. This syndrome is actually a variant of PMS. Here the brunt of the injury involves the corticobulbar pathway, with relative sparing of the corticospinal subdivision of the pyramidal tract.[14] Facial weakness, slurred speech, and dysphagia are the conspicuous pseudobulbar symptoms. But the sparing of the corticospinal fibers is only relative: mild distal involvement of the upper extremity's power is apparent, noticeable as modest clumsiness and weakness of the hand. A telltale hint of the slight, general compromise of the pyramidal tract is the presence of brisk tendon reflexes and a Babinski sign on the side of the clumsy hand. Occasional cases of dysarthria in its purest form, without accompanying deficits, can be found.

Ataxic Hemiparesis. The basis pontis exhibits an intricate lattice work. There, the frontopontine fibers reach the pontine nuclei, which send axons across the midline to the cerebellum via the middle cerebellar peduncle. This afferent cerebellary circuitry is crisscrossed by the unraveled descending pyramidal fibers. In view of this close interconnection, one would expect that in many cases of pontine PMS these afferent cerebellar fibers would be implicated to some extent, and a picture of ataxic hemiparesis would emerge.[15]

The dispersion into bundles of the corticospinal tract in the pons provides the grounds for circumscribed involvement of fibers that control restricted muscle groups. When the lower extremity (often just the foot and ankle) is selectively weakened, the syndrome of homolateral ataxia and crural paresis[16] will emerge as a common pontine stroke. The limited extent of the motor weakness in these cases allows for the full expression of the ataxic component.

Pure Sensory Stroke. One of the leading lacunar syndromes, pure sensory stroke (PSS) is characterized by unmixed sensory disturbances in one hemibody, without accompanying motor symptomatology.[17] The most common site of infarction that produces PSS is the ventral posterior nucleus of the thalamus, the relay station for sensory perception.[10] A lesion affecting this nucleus usually produces a dense loss of all forms of general somatic sensation in the contralateral face, trunk, and limbs. A strict midline split, like a plumb line, is considered a reliable sign to separate a thalamic syndrome from the cortical-subcortical counterpart.

An athetoid posturing of the hand ("thalamic hand") can at times be observed, and a few unfortunate patients may experience severe burning, dysesthetic pain of the most distressing and disabling type ("thalamic pain") in the area of the sensory loss.

With other localizations of thalamic lacunes, a dementing process characterized by abulia, short attention span, and memory loss is a possible rare event, akin to the thalamic dementias observed in parkinsonian patients treated with small stereotactic lesions in the thalamus.

Finally, some thalamic infarcts are large enough to impinge on the neighboring internal capsule. A mixed sensory-motor syndrome may emerge, similar to common surface strokes on cursory examination. Closer scrutiny would show a revealing lack of cortical findings.

Hemichorea-Hemiballismus. In some cases of large lacunes overlapping the internal capsule and the striatum, certain extrapyramidal features such as tremor or rigidity can be detected superimposed on the hemiplegia, but a bona fide extrapyramidal movement disorder is quite rare. However, the occasional documentation of instances of hemichorea-hemiballismus due to basal ganglia infarction justifies the inclusion of this syndrome in the list of lacunar strokes[18] (Fig 5).

Mesencephalic Lacunar Syndromes. As previously mentioned, a mesencephalic location of lacunes is decidedly uncommon. However, infrequently, one may find an occasional midbrain lacunar syndrome with lesions in the territory of the superior and inferior mesencephalic paramedian arteries: Weber syndrome (PMS with crossed third nerve palsy), Claude syndrome (cerebellar ataxia with crossed third nerve palsy), and different forms of the Parinaud syndrome (selective paralysis of downward gaze).

Infarcts in the territory overlapping the midbrain and the thalamus will cause a Parinaud syndrome combined with a thalamic dementia. This syndrome—sometimes

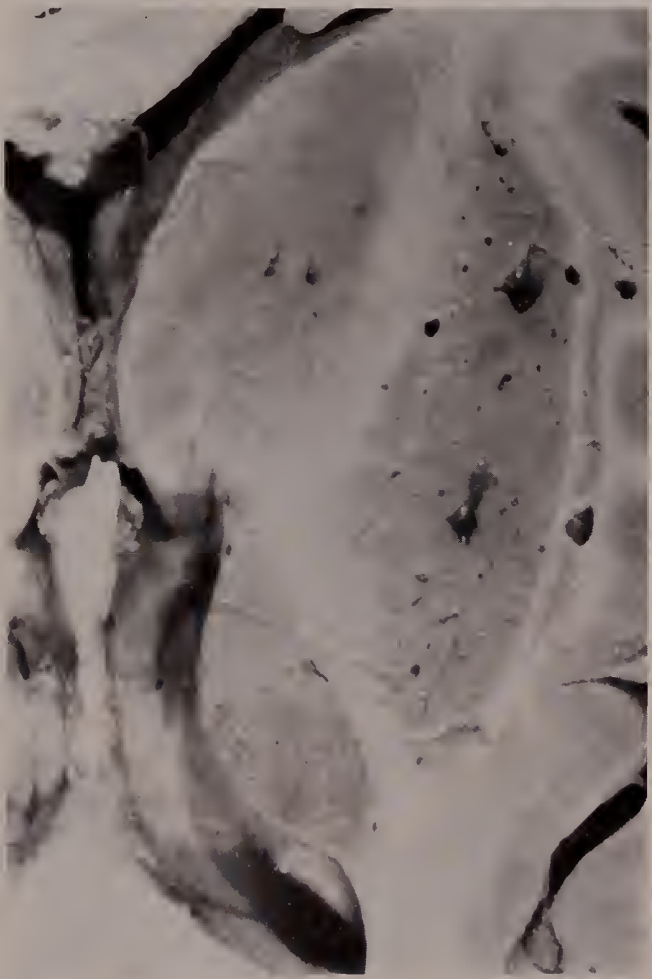


FIGURE 5. The putamen is the most common location of multiple lacunes. In this case some of the lacunes are present in the adjacent claustrum (courtesy of Dr W. A. Olzewski, Buffalo, NY).

colloquially referred to as "top o' the basilar embolism"—is one of the few lacunar syndromes in which an embolic cause seems firmly established. These infarcts are the result of the occlusion of the thalamo-subthalamic paramedian arteries that arise from the proximal segment of each posterior cerebral artery. The infarct typically has a butterfly shape. The tendency for emboli to enter these small vessels is probably due to their anatomic position, directly in line with basilar flow. This is another small vessel syndrome, peculiar in the fact that one of the midline arteries gives rise to bilateral branches.

Multilacunar Syndromes. Multiplicity of lacunes, a common clinical problem, can confound the picture by causing an additive effect. For instance, a quadriplegia or a locked-in syndrome could result from bilateral lesions in the pyramidal tracts (Fig 6).

In Fisher's opinion "l'etat lacunaire" described by Marie[8] (small step gait, dementia, pseudobulbar symptoms, incontinence) is rarely, if ever, encountered, and may even have been caused by a normal pressure hydrocephalus. Nonetheless, considering the possibility of a disparate array of lacunes scattered in time and place, the clinician may confront some bewildering mixture of findings and, consequently, is advised to expect the unexpected.

CLINICAL MANAGEMENT OF LACUNES

Computed tomographic (CT) scan remains the mainstay of the radiologic diagnosis.[19] But even the high resolution CT scanners miss a great number of lacunes, particularly the very small ones and the ones located in the pons (Fig 7). The ability of magnetic resonance imaging (MRI) to disclose these missed lacunes is still to be explored, but this technique will presumably increase the yield.

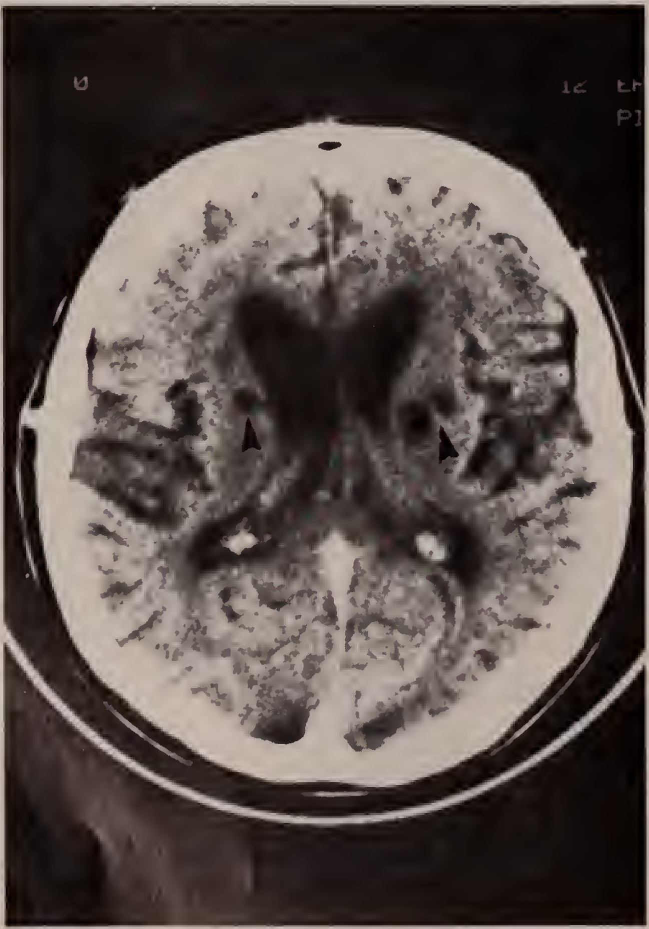


FIGURE 6. Bilateral lacunes in the internal capsule can produce a spastic quadriparesis (courtesy of Dr G. J. Alker, Buffalo, NY).

In the acute stage of strokes, a frequent clinical problem is the patient with an equivocal clinical picture and a normal CT scan. The quandary between a large stroke scanned too early versus a lacune too small to be seen has to be faced. The repetition of the CT scan in the ensuing days frequently settles the issue. The risk of submitting patients to repeated loads of contrast material is to be considered, especially in hypertensive patients with renal disease.

Lacunes are caused by arterial lesions that cannot be treated surgically; therefore, there is no point in exposing the patients with clear-cut lacunar syndromes to the danger of arteriography or other invasive studies.[1]

The treatment of lacunar infarcts remains deceptively simplistic. A few common sense extrapolations, unproven by empirical research, are the only guidelines for therapy. Treatment of arterial hypertension is a reasonable approach, in the hope of reducing the risk of further lacunes. There is some oblique evidence that this simple preventive method is effective. The incidence of lacunes found at autopsy seems to be decreasing as compared with earlier pathologic studies, which may reflect the impact of the better antihypertensive therapies in modern times. C. M. Fisher compared the results of his pathologic study of lacunes during the years 1950 to 1954 with the brains examined in the years 1975 and 1976, and found a reduction in the number of multiple lacunes. He had the impression that the florid lacunar states seen in the past are becoming almost a rarity.

Forceful treatment of hypertension in the acute stage of the stroke is to be shunned, since it may increase the ischemia in the affected area. It is probably better to wait until the deficits are stabilized before initiating the treatment of hypertension.[20]

Preventive treatment with platelet inhibitors has not been subjected to careful statistical scrutiny, but for lack of any satisfactory alternative, some physicians give this therapy the benefit of doubt.

Many lacunar strokes evolve over time and, consequently, a mode of therapy that would halt the progression of deficits in incomplete strokes would be of fundamental importance. Warnings against anticoagulation[11] with heparin or warfarin have been repeatedly given because of the risk of bleeding through the necrotic wall of the affected penetrating artery, particularly in hypertensive patients. How risky—and by the same token, how efficacious—anticoagulation therapy actually is has never been ascertained.

REFERENCES

1. Caplan LR: Lacunar infarction: A neglected concept. *Geriatrics* 1976; 31:71-75.
2. Fisher CM: Lacunes: Small, deep cerebral infarcts. *Neurology* (*Minneap*) 1965; 15:774-784.
3. Mohr JP, Caplan LR, Melski JW, et al: The Harvard Cooperative Stroke Registry: A prospective registry. *Neurology* (*Minneap*) 1978; 28:754-762.
4. Mohr JP: Lacunes. *Stroke* 1982; 13:3-11.
5. Fisher CM: The arterial lesions underlying lacunes. *Acta Neuropathol* (*Berl*) 1968; 12:1-15.

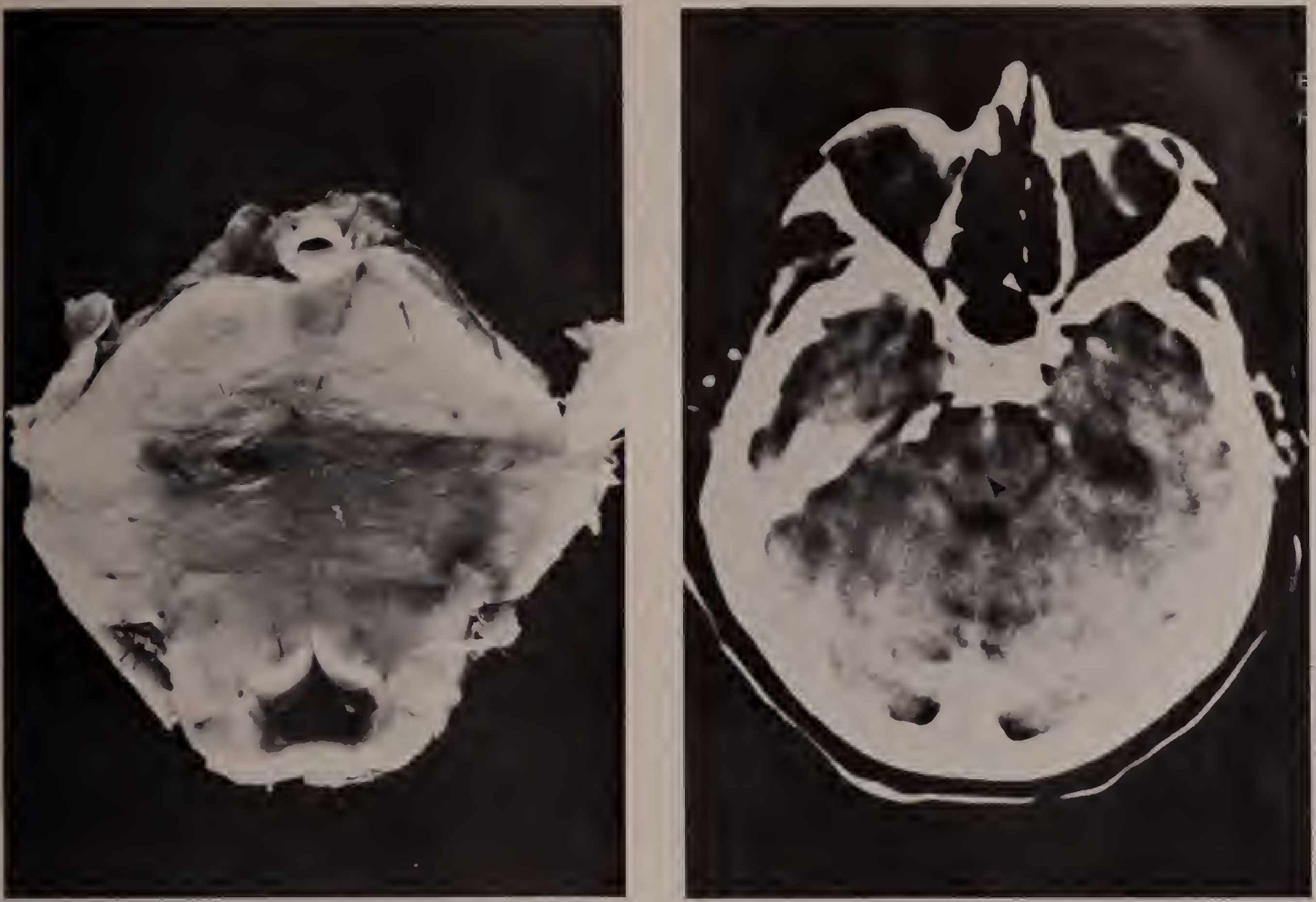


FIGURE 7. Pontine lacunes are often difficult to demonstrate with CT scan.

6. Fisher CM: Capsular infarcts: The underlying vascular lesions. *Arch Neurol* 1979; 36:65-73.

7. Chester EM, Agamanolis DP, Banker BQ, et al: Hypertensive encephalopathy: A clinicopathologic study of 20 cases. *Neurology* (*Minneap*) 1978; 28:928-939.

8. Marie P: Des foyers lacunaires de désintegration et des différents autres états cavitaires du cerveau. *Rev Med* 1901; 21:281-298.

9. Fisher CM, Caplan LR: Basilar artery branch occlusion: A cause of pontine infarction. *Neurology* (*Minneap*) 1971; 21:900-905.

10. Fisher CM: Thalamic pure sensory stroke: A pathologic study. *Neurology* (*Minneap*) 1978; 28:1141-1144.

11. Fisher CM: Lacunar strokes and infarcts: A review. *Neurology* (*NY*) 1982; 32:871-876.

12. Mohr JP: Lacunes. *Neurology Clinics* 1983; 1:201-221.

13. Fisher CM, Curry HB: Pure motor hemiplegia of vascular origin. *Arch Neurol* 1965; 13:30-44.

14. Fisher CM: A lacunar stroke: The dysarthria-clumsy hand syndrome. *Neurology* (*Minneap*) 1967; 17:614-617.

15. Fisher CM: Ataxic hemiparesis. A pathologic study. *Arch Neurol* 1978; 35:126-128.

16. Fisher CM, Cole M: Homolateral ataxia and crural paresis: A vascular syndrome. *J Neurol Neurosurg Psychiat* 1965; 28:48-55.

17. Fisher CM: Pure sensory stroke involving face, arm, and leg. *Neurology* (*Minneap*) 1965; 15:76-80.

18. Martin JP: Hemichorea (hemiballismus) without lesions in the corpus Luysii. *Brain* 1957; 80:1-10.

19. Rascol A, Clanet M, Manelfe C, et al: Pure motor hemiplegia: CT study of 30 cases. *Stroke* 1982; 13:11-17.

20. Fisher CM: Bilateral occlusion of basilar artery branches. *J Neurol Neurosurg Psychiatry* 1977; 40:1182-1189.

The medical writings of Moses Maimonides

Fred Rosner, MD

Moses, son of Maimon (*Rambam* in Hebrew, *Abu Imram Musa Ibn Maimun* in Arabic, and *Maimonides* in Greek),[1] was born in Cordova, Spain, on March 30, 1135, corresponding to Passover eve of the Hebrew year 4895. His mother died in childbirth and consequently his father, a dayan (judge), raised him. Persecution by the Almochades (Almoravids), a fanatical Moslem group from North and West Africa, forced the Maimon family to flee Cordova in the year 1148. The family wandered through southern Spain and northern Africa for the next ten years and finally settled in Fez, Morocco.

Little is known of Maimonides' early life and education. There are no sources indicating that Maimonides had any formal medical education. In his *Medical Aphorisms*, he mentions "the elders before whom I have read"; this is the only allusion to some semiprivate study of medicine. A few times he mentions the son of Ibn Zuhr from whom he heard teachings of the latter's illustrious father (the great physician Abu Merwan Ibn Zuhr), whom Maimonides held in great esteem.

Maimonides must have been an avid reader since his medical writings show a profound knowledge of ancient Greek authors in Arabic translations and of Moslem medical works. Hippocrates, Galen, and Aristotle were his Greek medical inspirations and Rhazes of Persia, Al Farabi, and Ibn Zuhr, the Spanish-Arabic physician, are Moslem authors frequently quoted by Maimonides.

The Maimon family left Morocco in 1165, traveled to Palestine, landing in Acco, and from there to Egypt where they settled in Fostat (old Cairo). Maimonides turned to medicine as a livelihood only after the death of his father in 1166 and the death of his brother in a shipwreck shortly thereafter. Maimonides was left with his brother's wife and children to support and, after a year's illness following his father's death, entered into the practice of medicine. In 1174, at age 39, he was appointed Court Physician to Visier Al-Fadhil, Regent of Egypt during the absence of the Sultan, Saladin the Great, who was fighting in the Crusades in Palestine. It was at this time that Richard the Lion-Hearted, also fighting in the Crusades, is reported to have invited Maimonides to become his personal physician, an offer which Maimonides declined. His reputation as a physician grew in Egypt and neighboring countries and his fame as a theologian and a philosopher became widespread.

In 1193, Saladin died and his eldest son, Al Afdal Nur ad Din Ali, a playboy, succeeded him. As a result, Maimonides' medical duties became even heavier, as described in the famous letter he wrote to his friend, disciple, and translator, French rabbi Samuel Ibn Tibbon, in the year 1199:

> . . . I live in Fostat and the Sultan resides in Cairo; these two places are two Sabbath limits [marked off areas around a town within which it is permitted to move on the Sabbath; approximately one and a half miles] distant from each other. My duties to the Sultan are very heavy. I am obliged to visit him every day, early in the morning, and when he or any of his children or concubines are indisposed, I cannot leave Cairo but must stay during most of the day in the palace. It also frequently happens that one or two of the officers fall sick and I must attend to their healing. Hence, as a rule, every day, early in the morning, I go to Cairo and, even if nothing unusual happens there, I do not return to Fostat until the afternoon. Then I am famished but I find the antechambers filled with people, both Jews and Gentiles, nobles and common people, Judges and policemen, friends and enemies,—a mixed multitude who await the time of my return.
>
> I dismount from my animal, wash my hands, go forth to my patients, and entreat them to bear with me while I partake of some light refreshment, the only meal I eat in twenty-four hours. Then I go to attend to my patients and write prescriptions and directions for their ailments. Patients go in and out until nightfall, and sometimes, even as the Torah is my faith, until two hours and more into the night. I converse with them and prescribe for them even while lying down from sheer fatigue. When night falls, I am so exhausted that I can hardly speak.
>
> In consequence of this, no Israelite can converse with me or befriend me [on religious or community matters] except on the Sabbath. On that day, the whole congregation, or at least, the majority, comes to me after the morning service, when I instruct them as to their proceedings during the whole week. We study together a little until noon, when they depart. Some of them return and read with me after the afternoon services until evening prayer. In this manner, I spend the days. I have here related to you only a part of what you would see if you were to visit me . . .

Maimonides was also the spiritual leader of the Jewish community of Egypt. At age 33, in the year 1168, shortly after settling in Fostat, he completed his first major work, the *Commentary on the Mishnah*. In 1178, ten years later, his *magnum opus*, the *Mishneh Torah*, was finished. This monumental work is a 14-book compilation of all biblical and talmudic law and remains a classic to this day. In

---

Dr Rosner is Director, Department of Medicine, Queens Hospital Center Affiliation of the Long Island Jewish Medical Center, and Professor of Medicine, Health Sciences Center, State University of New York at Stony Brook.

Address correspondence to Dr Rosner, Queens Hospital Center, 82-68 164th St, Jamaica, NY 11432.

1190, Maimonides' great philosophical masterpiece, the *Guide for the Perplexed*, was completed.

Maimonides died on December 13, 1204 (*Tebeth* 20, 4965, in the Hebrew calendar), and was buried in Tiberias. Legend relates that Maimonides' body was placed upon a donkey and the animal set loose. The donkey wandered and finally stopped in Tiberias.

MAIMONIDES' WRITINGS

Maimonides was a prolific writer. His famous trilogy, the *Commentary on the Mishnah*, the *Mishneh Torah*, and the *Guide for the Perplexed*, has already been mentioned. Each of these works alone would have indelibly recorded Maimonides' name for posterity. However, in addition to these, he also wrote a *Book on Logic* (*Ma'amar Hahigayon*), a *Book of Commandments* (*Sefer Hamitzvoth*), an *Epistle to Yemen* (*Iggereth Hashmad*), a *Treatise on Resurrection* (*Ma'amar Techiyath Hamethim*), commentaries on several tractates of the Talmud, and over 600 Responsa. Several additional works including the so-called *Prayer of Maimonides*[1] are attributed to him but such claims are, in fact, spurious, the prayer having been written in 1783.

In addition to all the books just enumerated, Maimonides also wrote ten medical works.[2,3] This article offers a brief examination and analysis of these medical writings.

Extracts from Galen. The first of Maimonides' medical writings is called *Extracts from Galen* or *The Art of Cure*. Galen's medical writings consist of over 100 books, and two volumes were required just to catalog and index them all. Maimonides therefore extracted what he considered the most important of Galen's pronouncements and compiled them verbatim in a small work which was intended primarily for the use of students of Greek medicine. This work, like all of Maimonides' medical books, was originally written in Arabic. At least two manuscripts exist today, one in Hebrew and one in Arabic letters. This work has heretofore never been published in any language, but brief excerpts therefrom appeared in both English and Hebrew in a Hebrew periodical.[4] The complete English and Hebrew translations by Uri Barzel will be published soon.

Commentary on the Aphorisms of Hippocrates. The second of Maimonides' medical writings is the *Commentary on the Aphorisms of Hippocrates*. The famous aphorisms of Hippocrates were translated from the Greek into Arabic by Hunain Ibn Ischak in the ninth century. Maimonides wrote his commentary on this translation. Two incomplete Arabic manuscripts exist. A good medieval translation into Hebrew was made by Moses ben Samuel Ibn Tibbon. In this work, Maimonides occasionally criticizes both Hippocrates and Galen where the views of either of these Greeks differ from his own. For example, in chapter five, Hippocrates is quoted as having said, "a boy is born from the right ovary, a girl from the left," to which Maimonides remarks: "A man should be either prophet or genius to know this." The introduction to this work was edited in the original Arabic with one German and two Hebrew translations by Steinschneider[5] in 1894. The entire work was published by Hasida[6] in 1935 and again in a definitive edition by Muntner[7] in 1961. Bar Sela and Hoff[8] published Maimonides' interpretation of the first aphorism of Hippocrates. This is the famous aphorism which has been called the motto or credo of the art of medicine:

> Life is short, and the art long, the occasion fleeting, experience fallacious and judgment difficult. The physician must not only be prepared to do what is right himself, but must also make the patient, the attendant and the externals cooperate.

I published Maimonides' *Introduction* to this work[9] as well as the entire work[10] in English.

Medical Aphorisms of Moses. The third of Maimonides' medical works, the most voluminous of all, is the *Medical Aphorisms of Moses* (*Pirke Moshe*). This book is comprised of 1,500 aphorisms based mainly on Greek medical writings. There are 25 chapters, each dealing with a different area of medicine, including anatomy, physiology, pathology, symptomatology, diagnosis, etiology of disease, therapeutics, fevers, blood-letting or phlebotomy, laxatives and emetics, surgery, gynecology, hygiene, exercise, bathing, diet, drugs, and medical curiosities. A complete Arabic original manuscript exists in the Gotha Library in East Germany. A Hebrew translation was made in the 13th century and published in Lemberg, Poland, in 1834 and again in Vilna in 1888.[11] The definitive Hebrew edition is that of Muntner[12] dated 1959. Maimonides' aphorisms[13] were also translated into Latin in the 13th century and appeared as an incunabulum in Bologna in 1489 and again in Venice in 1497, followed by several printed Latin editions.[14] Only small fragments of this work appeared in a Western language[15-18] until the complete English version by myself and Muntner was published in two volumes[19,20] and reprinted.[21]

A few excerpts from this most important work will give the reader the flavor of Maimonidean medical thinking. Maimonides speaks of cerebrovascular disease:

> . . . one can prognosticate regarding a stroke, called apoplexy. If the attack is severe, he will certainly die but if it is minor, then cure is possible, though difficult . . . the worst situation that can occur following a stroke is the complete irreversible suppression of respiration. . . .

Maimonides explains that diabetes mellitus was seldom seen in "cold" Europe whereas it was frequently encountered in "warm" Africa. He also reports this disease to be associated with the imbibition of suave water of the Nile (Maimonides lived in Fostat, or old Cairo). The English translation of this most important aphorism, No. 69, from the eighth chapter, is as follows:

> I, too, have not seen it in the West [Spain, where Maimonides was born, and/or Morocco, where he fled from the persecution of the Almochades] nor did any one of my teachers under whom I studied mention that they had seen it [diabetes]. However, here in Egypt, in the course of approximately ten years, I have seen more than twenty people who suffered from this illness. This brings one to the conclusion that this illness occurs mostly in warm countries. Perhaps the waters of the Nile, because of their suaveness, may play a role in this.

A very accurate description of obstructive emphysema is provided during a lengthy discussion of respiratory disease: ". . . reason [for respiratory embarrassment] is narrowing of the organs of respiration, then the breast is seen to greatly expand. This expansion produces rapid and cut off [respirations] . . . ."

Clubbing of the fingers associated with pulmonary disease, already described by Hippocrates, is beautifully depicted: "With an illness affecting the lungs called *'hasal'*; namely, phthisis, there develops rounding of the nail as a rainbow." The signs and symptoms of pneumonia are remarkably accurately described: "The basic symptoms which occur in pneumonia and which are never lacking are as follows: acute fever, sticking [pleuritic] pain in the side, short rapid breaths, serrated pulse and cough, mostly [associated] with sputum . . . ." Hepatitis is just as beautifully described:

> The signs of liver inflammation are eight in number as follows: high fever, thirst, complete anorexia, a tongue which is initially red and then turns black, biliary vomitus, initially yellow egg yolk in color which later turns dark green, pain on the right side which ascends up to the clavicle. . . Occasionally a mild cough may occur and a sensation of heaviness which is first felt on the right side and then spreads widely. . . .[22]

Treatise on Hemorrhoids. The fourth of Maimonides' medical writings is his *Treatise on Hemorrhoids*. This work was written for a nobleman, as Maimonides explains in the introduction—probably a member of the sultan's family. There are seven chapters dealing with normal digestion, foods harmful to patients with hemorrhoids, beneficial foods, and general and local therapeutic measures such as sitz baths, oils, and fumigations. Maimonides disapproves of bloodletting or surgery for hemorrhoids except in very severe cases. Maimonides' whole approach to the problem seems to bespeak a modern medical trend. The *Treatise on Hemorrhoids* was first published by Kroner[23] in 1911 in Arabic, Hebrew, and German. A general description of the work appeared in English in 1927 by Bragman.[24] The definitive Hebrew edition is that of Muntner[25] dated 1965, and Muntner and I[26] published an English translation of the entire work. An improved, more fully annotated English translation was recently published.[27]

In the introduction to this work, Maimonides describes the reason for writing it:

> There was a youth [descended] from knowledgeable, intelligent and comprehending forebears, from a prominent and renowned family, distinguished and charitable and of great means, in whom the affliction of hemorrhoids occurred at the mouth of the rectum, that interested me in his problem and placed the task [of healing them] upon me. These irritated him on some occasions and he treated them in the customary therapeutic manner until the pain subsided and the prolapsed rectum [literally, excesses that protruded] became reduced and returned to the interior of the body so that his [bodily] functions returned to normal. Because this [illness] recurred many times, he considered having them extirpated in order to uproot this malady from its source so that it not return again. I informed him of the danger inherent in this, in that it is not clear if these hemorrhoids [literally, additions] are of the variety which should be excised or not, since there are people in whom they have once been [surgically] extirpated and in whom other hemorrhoids develop. This is because the causes which gave rise to the original ones remained and, therefore, new ones develop.

Here Maimonides provides an insight into the etiology of disease in general in that he regards operative excision of hemorrhoids with skepticism, because surgery does not remove the underlying causes which produced the hemorrhoids in the first place.

Treatise on Sexual Intercourse. The fifth work is Maimonides' *Treatise on Sexual Intercourse*, written for the nephew of Saladin, the Sultan al Muzaffar Umar Ibn Nur Ad-Din. The sultan heavily indulged in sexual activities and asked Maimonides, his physician, to aid him in increasing his sexual potency. The work mainly consists of recipes for foods and drugs which are either aphrodisiac or antiaphrodisiac in their actions. Maimonides advises moderation in sexual intercourse and describes the physiology of sexual temperaments. There are two versions to this book, a short, authentic and a longer, spurious version. Both were first edited and published by Kroner[28] in 1906 in Hebrew and German. Ten years later, Kroner[29] published the true short version from the original Arabic manuscript in Granada. An Italian edition appeared in 1960,[30] and English[31] and Spanish[32] translations were published in 1961. The definitive Hebrew edition of both authentic[33] and spurious[34] versions of Maimonides' books on sex is that of Muntner, dated 1965. A new English translation of the true work was published by me[35] and reprinted.[27]

Treatise on Asthma. The sixth medical book of Moses Maimonides is his *Treatise on Asthma*. The patient for whom this book was written suffered from violent headaches which prevented him from wearing a turban. The patient's symptoms began with a common cold, especially in the rainy season, forcing him to gasp for air until phlegm was expelled. The patient asked whether a change of climate might be beneficial. Maimonides, in 13 chapters, explains the rules of diet and climate in general and those rules specifically suited for asthmatics. He outlines the recipes for food and drugs and describes the various climates of the Middle East. He states that the dry Egyptian climate is efficacious for sufferers from this disease and warns against the use of very powerful remedies. The first critical edition of this work appeared in Hebrew in 1940, edited by Muntner.[36] Additional manuscripts became available after World War II and a corrected, improved, and revised second Hebrew edition appeared in 1963.[37] Only 300 copies of this edition were printed and thus a third edition was published by Muntner[38] in 1965. An English version of Maimonides' book on asthma was published in 1963[39] and a French translation in 1965.[40] I have commented extensively on this work elsewhere.[41,42]

The last chapter of this work deals with concise admonitions and aphorisms which Maimonides considered "useful to any man desirous of preserving his health and administering to the sick." The chapter begins as follows: The first thing to consider. . . is the provision of fresh air, clean water and a healthy diet." Fresh air is described in some detail:

> . . . City air is stagnant, turbid and thick, the natural result of its big buildings, narrow streets, the refuse of its inhabitants. . . one should at least choose for a residence a wide-open site. . . living quarters are best located on an upper floor. . . and ample sunshine. . . Toilets should be located as far as possible from living rooms. The air should be kept dry at all times by sweet scents, fumigation and drying agents. The concern for clean air is the foremost rule in preserving the health of one's body and soul.

Treatise on Poisons and Their Antidotes. The seventh medical work of Maimonides is his *Treatise on Poisons and Their Antidotes*. It is one of the most interesting and popular works because it is very scientific and modern in its approach and was, therefore, used as a textbook of toxicology throughout the middle ages. The book was written at the request of Maimonides' noble protector, the Grand Vizier and Supreme Judge Al Fadhil, who, in 1199, asked Maimonides to write a treatise on poisons for the layman by which to be guided before the arrival of a physician. In the introduction, Maimonides praises Al Fadhil and his feats in war and peace. He mentions Al Fadhil's orders to import from distant lands ingredients lacking in Egypt but necessary for the preparation of two antidotes against poisonings, the "great theriac" and the "electuary of Mithridates."

The first section of the book deals with snake and dog bites and with scorpion, bee, wasp, and spider stings. The first chapter concerns the conduct of the victim in general. Thus Maimonides states as follows:

> When someone is bitten, immediate care should be taken to tie the spot above the wound as fast as possible to prevent the poison from spreading throughout the body; in the meantime, another person should make cuts with a black lancet directly above the wound, suck vigorously with his mouth and spit out. Before doing that, it is advisable to disinfect the mouth with olive oil, or with spirit in oil . . . Care should be taken that the sucking person has no wound in his mouth, or rotten teeth . . . should there be no man available to do the sucking, cupping-glasses should be applied, with or without fire; the heated ones have a much better effect because they combine the advantages of sucking and cauterizing at the same time . . . Then apply the great theriac . . . Apply to the wound some medicine which should draw the poison out of the body.

In his book on poisons, Maimonides also describes the long incubation period for rabies (up to 40 days). Numerous Arabic, Hebrew, and Latin manuscripts are extant.[43] A German translation was published in 1873 by Steinschneider.[44] A French translation by Rabbinowicz appeared in 1865 and was reprinted in 1935.[45] An English translation of Steinschneider's German version is that of Bragman[46] in 1926. The definitive Hebrew edition of Muntner[47] appeared in 1942 and Muntner's English version was published in 1966.[48] I commented on this work[49] and published a fully annotated new English translation with commentary and bibliography.[27]

Regimen of Health. The eighth book is the *Regimen of Health* (*Regimen Sanitatis*) which Maimonides wrote in 1198 during the first year of the reign of Sultan Al Malik Al Afdal, eldest son of Saladin the Great. The sultan was a frivolous and pleasure-seeking man of 30, subject to fits of melancholy or depression due to his excessive indulgences in wine and sex, and his warlike adventures against his own relatives and in the Crusades. He complained to his physician of constipation, dejection, bad thoughts, and indigestion. Maimonides answered his royal patient in four chapters. The first chapter is a brief abstract on diet taken mostly from Hippocrates and Galen. The second chapter deals with advice on hygiene, diet, and drugs in the absence of a physician. The third, extremely important chapter contains Maimonides' concept of "a healthy mind in a healthy body," one of the earliest descriptions of psychosomatic medicine. He indicates that the physical well being of a person is dependent on his mental well being and vice versa. The final chapter summarizes his prescriptions relating to climate, domicile, occupation, bathing, sex, wine drinking, diet, and respiratory infections.

The whole treatise on the regimen of health is short and concise but to the point. This is the reason for its great success and popularity throughout the years. It is extant in numerous manuscripts. A Hebrew translation from the original Arabic was made by Moses ben Samuel Ibn Tibbon in 1244 and this version was reprinted several times in the 19th century (Prague 1838, Jerusalem 1885, Warsaw 1886). Two Latin translations were made in the 13th century. Several 15th century incunabulae and 16th century editions of these Latin versions exist. One of the first Hebrew editions is that of Bloch[50] in 1838. An annotated German translation by Winternitz[51] was published in 1843; a French translation by Carcousse[52] in 1887 in Algiers; and Russian[53] and Spanish[54] translations in 1930 and 1961, respectively. The Arabic text with German and Hebrew translations was published by Kroner[55] in 1925, although he had already published the all-important chapter three dealing with psychosomatic medicine 11 years earlier in 1914.[56]

English translations of chapter three have been published by Bragman,[57] Savitz,[58] and Butterworth[59] and of the first two chapters by Skoss.[60] The definitive Hebrew edition is that of Muntner,[61] dated 1957, although the Maimonidean bibliographer Dienstag[62] cites several additional Hebrew editions. Two English translations of the entire work were published: one in 1958 by Gordon[63] and one in 1964 by Bar Sela, Hoff, and Faris.[64] Another German translation by Muntner[65] appeared in 1966. These numerous editions in many languages attest to the importance and popularity of Maimonides' *Regimen of Health*.

Discourse on the Explanation of Fits. The ninth medical writing of Maimonides is the *Discourse on the Explanation of Fits*. This work has been called Maimonides' swan song as it was thought to be the last of his medical works, having been written in the year 1200, four years before his death. It was also written for the Sultan Al Malik Al Afdal and is sometimes considered to represent chapter five of the *Regimen of Health*. The sultan persisted in his over-indulgences and wrote to Maimonides, who was himself ill, asking advice about his health. Maimonides confirms most of the prescriptions of the sultan's other physicians regarding wine, laxatives, bathing, exercise, and the like and, near the end, prescribes a very detailed hour-by-hour regimen for the daily life of the sultan. The original Arabic version was edited and published with Hebrew and German translations by Kroner[66] in 1928. English editions by Bar Sela, Hoff, and Faris[64] in 1964 and by myself and Muntner in 1969,[26] another German version by Muntner[65] in 1966, and another Hebrew edition by Muntner[67] in 1969 are available. The most recent and best edition is that by Leibowitz and Marcus[18] entitled *Moses Maimonides on the Causes of Symptoms*, in which the text is presented in four languages (Arabic, Hebrew, Latin, and English) and is accompanied by a running commentary, explanatory essays, and a comprehensive catalog of drugs.

Glossary of Drug Names. The final authentic medical book of Maimonides is the *Glossary of Drug Names*. This work was discovered by Max Meyerhof, an ophthalmologist in Egypt, in the Aya Sofia library in Istanbul, Turkey, as Arabic manuscript No. 3711.[69,70] Dr Meyerhof edited the original Arabic and provided a French translation with a detailed commentary which he published in 1940 in Cairo.[71] A Hebrew edition by Muntner[67] appeared in 1969 and my English translation was published in 1979.[72] The work is essentially a pharmacopoeia and consists of 405 short paragraphs containing names of drugs in Arabic, Greek, Syrian, Persian, Berber, and Spanish.

SUMMARY

Maimonides' medical writings are varied, comprising extracts from Greek medicine, a series of monographs on health in general and several diseases in particular, and a recently discovered pharmacopoeia demonstrating Maimonides' extensive knowledge of Arabic medical literature and his familiarity with several languages. Some people feel that Maimonides' medical writings are not as original as his theological and philosophical writings. However, his medical works demonstrate the same lucidity, conciseness, and formidable powers of systematization and organization so characteristic of all his writings. The *Book on Poisons*, the *Regimen of Health*, and the *Medical Aphorisms of Maimonides* became classics in their fields during medieval times.

REFERENCES

1. Rosner F: The Physician's Prayer attributed to Moses Maimonides. *Bull Hist Med* 1967; 41:440-454.
2. Rosner F: Maimonides the physician: A bibliography. *Bull Hist Med* 1969; 43:221-235.
3. Rosner F: Maimonides the Physician: A bibliography. *Clio Medica* 1980; 15:75-79.
4. Barzel U: The art of cure: A non-published medical book by Maimonides. *Harofe Haivri* 1955; 2:82-83 (Hebr), 177-165 (Eng).
5. Steinschneider M: Die Vorrede des Maimonides zu seinem Commentar über die Aphorismen des Hippokrates. *Ztschr d deutsch Morganland Gesellsch* 1894; 48:213-234.
6. Hasida (Bocian) MZ: *Perush lepirkê Abukrat shel Ha-Rambam. Hassegullah* (Jerusalem) 1934-1935, nos. 1-30 (stencil) (Hebr).
7. Muntner S: *Mosheh ben Maimon. Commentary on the Aphorisms of Hippocrates. Perush lepirkei Abukrat.* Jerusalem, Mossad Harav Kook, 1961.
8. Bar Sela A, Hoff HE: Maimonides' interpretation of the first aphorism of Hippocrates. *Bull Hist Med* 1968; 37:347-354.
9. Rosner F: The introduction of Maimonides to his "Commentary on the aphorisms of Hippocrates." *Clio Medica* 1976; 11:59-64.
10. Rosner F: *Moses Maimonides' Commentary on the Aphorisms of Hippocrates.* Haifa, Maimonides Research Institute, 1987.
11. Magid Z (ed): *Medical Aphorisms of Moses* (*Pirke Moshe*). Vilna, L. Matz, 1888 (1st ed Lemberg, 1834) (Hebr).
12. Muntner S: *Moshe ben Maimon.* (*Medical*) *Aphorisms of Moses in Twenty-Five Treatises.* (*Pirke Moshe Birefuah*). Jerusalem, Mossad Harav Kook, 1959 (Hebr, Eng summary).
13. Leibowitz JO: Maimonides' aphorisms. *Koroth* 1955; 1:213-219 (Hebr); I-III (Eng summary).
14. Leibowitz JO: The Latin translations of Maimonides' aphorisms. *Koroth* 1973; 6:273-281 (Hebr); XCIII-XCIV (Eng summary).
15. Steinberg W, Muntner S: Maimonides' views on gynecology and obstetrics. English translation of chapter sixteen of his treatise, "Pirke Moshe" (Medical Aphorisms). *Am J Obst Gynec* 1965; 91:443-448.
16. Rosner F, Muntner S: Moses Maimonides' aphorisms regarding analysis of urine. *Ann Intern Med* 1969; 71:217-220.
17. Rosner F, Muntner S: The surgical aphorisms of Moses Maimonides. *Am J Surg* 1970; 119:718-725.
18. Rosner F: Moses Maimonides and diseases of the chest. *Chest* 1971; 60:68-72.
19. Rosner F, Muntner S: *Studies in Judaica. The Medical Aphorisms of Moses Maimonides.* New York, Yeshiva University Press, 1970, vol 1.
20. Rosner F, Muntner S: *Studies in Judaica. The Medical Aphorisms of Moses Maimonides.* New York, Yeshiva University Press, 1971, vol 2.
21. Rosner F, Muntner S: *Studies in Judaica. The Medical Aphorisms of Moses Maimonides* (2 vols). New York, Bloch Publishing Co for Yeshiva University Press, 1973.
22. Rosner F: The medical aphorisms of Moses Maimonides, in Leibowitz JO (ed): *Memorial Volume in Honor of S. Muntner.* Jerusalem, Israel Institute of the History of Medicine, 1983, pp 6-30.
23. Kroner H: Die Haemorrhoiden in der Medicin XII and XIII. Jahrhunderts. *Janus* 1911; 16:441-456, 644-718.
24. Bragman LJ: Maimonides' treatise on hemorrhoids. *NY State J Med* 1927; 27:598-601.
25. Muntner S: *Moshe ben Maimon. On Hemorrhoids* (*Birefuoth Hatechorim*). Jerusalem, Mossad Harav Kook, 1965 (Hebr).
26. Rosner F, Muntner S: *The Medical Writings of Moses Maimonides. Treatise on Hemorrhoids and Maimonides' Answers to Queries.* Philadelphia, JB Lippincott Co, 1969.
27. Rosner F: *Moses Maimonides' Treatises on Poisons, Hemorrhoids, and Coitus.* Haifa, Maimonides Research Institute, 1984.
28. Kroner H: *Ein Betrag zur Geschichte der Medizin des XII Jahrhunderts an der Hand Zweier Medizinischer Abhandlungen des Maimonides auf Grund von 6 unedierten Handschriften.* Oberdorf-Bopfingen, Germany, Itzowski, 1906.
29. Kroner H: Eine Medizinische Maimonides Handschrift aus Granada. Ein Beitrag zue Stilistik des Maimonides und Charakteristik der Hebraischen Uebersetzungsliteratur. *Janus* 1916; 21:203-247.
30. DeMartini U: *Maimonides. Segreto dei segreti.* Rome, Instituto di storia della Medicina dell'Universita di Roma, 1960.
31. Gorlin M: *Maimonides "On Sexual Intercourse"* (*Fi'l-Jima*). Brooklyn, NY, Rambash Publishing, 1961.
32. Chelminski E: Notes introductorias al "Guia sobre el contacto sexual" de Maimonides. *Anales de ars Medici* (Mexico) 1961; 5:240-248.
33. Muntner S: *Moshe ben Maimon on the Increase of Physical Vigour* (*M'amar al chizuk koach hagavra*). Jerusalem, Mossad Harav Kook, 1965, pp 35-65 (Hebr).
34. Muntner S: Pseudo-Maimonides on sexual life, in *Sexual Life, Collection of Medieval Treatises* (*Ma'amar al razei hachajim haminiyim*). Jerusalem, Geniza, 1965 (Hebr).
35. Rosner F: *Sex Ethics in the Writings of Moses Maimonides.* New York, Bloch Publishing Co, 1974.
36. Muntner S: *Moshe ben Maimon* (*Maimonides*). *Sefer Hakatzereth* (*The Book on Asthma*). Jerusalem, Rubin Mass, 1940 (Hebr).
37. Muntner S: *Rabbi Moses ben Maimon. Sefer hakatzereth, or Sefer hamisadim* (*The Book on Asthma*). Jerusalem, Geniza, 1963.
38. Muntner S: *Moshe ben Maimon on Asthma* (*Sefer Hakatzereth*). Jerusalem, Mossad Harav Kook, 1965, pp 67-119 (Hebr).
39. Muntner S: *The Medical Writings of Moses Maimonides. Treatise on Asthma.* Philadelphia, JB Lippincott Co, 1963.
40. Muntner S, Simon I: Le Traité de L'Asthme de Maimonide (1135-1304). Traduit Pour la Premiere Fois en Français d'Après le Texte Hébreu. *Rêv d'Hist Méd Héb* 1963; 16:171-186, 1964; 17:5-13, 83-97, 127-139, 187-196, 1965; 18:5-15.
41. Rosner F: Moses Maimonides' treatise on asthma. *Thorax* 1981; 36:245-251.
42. Rosner F: Moses Maimonides' treatise on asthma. *J Asthma* 1984; 21:119-129.
43. Rosner F: Moses Maimonides' treatise on poisons. *JAMA* 1968; 205:914-916.
44. Steinschneider M: Gifte und ihre Heilung. *Virchows Arch F Path Anat* 1873; 57:62-120.
45. Rabbinowicz IM: *Traité des Poisons.* Paris, Lipschutz, 1935 (1st ed, 1865).
46. Bragman LJ: Maimonides' treatise on poisons. *Med J and Rec* 1926; 124:103-107.
47. Muntner S: *Moshe ben Maimon* (*Maimonides*), *Samei hamaveth veharafuoth kenegdam* (*Poisons and Their Antidotes, or "The Treatise to the Honored One"*). Jerusalem, Rubin Mass, 1942 (Hebr).
48. Muntner S: *The Medical Writings of Moses Maimonides. Vol 2: Treatise on Poisons and Their Antidotes.* Philadelphia, JB Lippincott Co, 1966.
49. Rosner F: Moses Maimonides' treatise on poisons. *NY State J Med* 1980; 80:1627-1630.
50. Bloch S: *Michtav Harav Rabenu Moshe ben Maimon Be'ad HaSultan. Kerem Chemed* 1838; 3:31-39.
51. Winternitz D: *Das Diatetische Sendschreiben des Maimonides* (*Rambam*) *an den Sultan Saladin.* Vienna, 1843.
52. Carcousse M: *Hygiène Israélite, Principes de Santé Physique et Morale de l'Arabe par Arab Mouchi ben Mimoun.* Algiers, 1887.
53. Shmukler IK: *Pismo Moiseh Maimonida K Egipefskomu Sultanu.* Gugienicheskie Sovetia Perevod S Drevneevreiskogo Doctora I.K. Shmuklera (Kiev). Otdelnii Ottisk Iz, "Vrach Dela" #14-16. Charkov. "Nauchnaja Misl." Uchr. NKZ UKSSR (1930).
54. Chelminsky E: "La Preservacion de la Juventud" de Maimonides. Version Castellana. *Anales de Ars Medici* (Mexico) 1961; 5:303-344.
55. Kroner H: *Fi tadbir as sihhat.* Gesundheitsanleitung des Maimonides fur den Sultan al-Malik al-Afdhal. *Janus* 1923; 27:101-116, 286-330, 1924; 28:61-74, 143-152, 199-217, 408-419, 455-472, 1925; 29:235-258.
56. Kroner H: *Die Seelenhygiene des Maimonides. Auszug aus dem 3. Kapital des diatetischen Sendschreibens des Maimonides an den Sultan al Malik Alafdahl* (ca. 1198). Frankfurt A. M., J. Kauffmann, 1914 (Germ, Hebr, and Arab).
57. Bragman LJ: Maimonides on physical hygiene. *Ann Med Hist* 1925; 7:140-143.
58. Savitz H: Maimonides' hygiene of the soul. *Ann Med Hist* 1932; 4:80-86.
59. Butterworth CE: On the management of health, in Weiss RL, Butterworth CE (eds): *Ethical Writings of Maimonides.* New York, New York University Press, 1975, pp 105-111.
60. Skoss SL: The treatises of Maimonides on health care, in *Portrait of a Jewish Scholar; Essays and Addresses.* New York, Bloch Publishing Co, 1957, pp

99-116.

61. Muntner S: *Moshe ben Maimon. Hanhagath Habriyuth. Regimen sanitatis. Letters on the hygiene of the body and of the soul.* Jerusalem, Mossad Harav Kook, 1957 (Hebr).

62. Dienstag JI: Translators and editors of Maimonides' medical works; a bio-bibliographical survey, in Leibowitz JO (ed): *Memorial Volume in Honor of S. Muntner.* Jerusalem, Israel Institute of the History of Medicine, 1983, pp 95-135.

63. Gordon HL: *Moses ben Maimon, The Preservation of Youth. Essays on Health (Fi Tadbir As-Sihha).* New York, Philosophical Library Inc, 1958.

64. Bar Sela A, Hoff HE, Faris E: *Moses Maimonides' Two Treatises on the Regimen of Health.* Philadelphia, American Philosophical Society (trans n.s.), 1964, vol 54, part 4.

65. Muntner S: *Regimen Sanitatis oder Dietetik fur die Seele und den Korper mit Anhang der Medizinischen Responsen und Ethik des Maimonides.* Basel, Switzerland, S. Karger, 1966.

66. Kroner H: Der Medizinische Schwanengesang des Maimonides. *Janus* 1928; 32:12-116.

67. Muntner S: *Moshe ben Maimon. Biyur Shaymoth Harefuoth (Lexicography of Drugs, and Medical Responses).* Jerusalem, Mossad Harav Kook, 1969.

68. Leibowitz JO, Marcus S: *Moses Maimonides on the Causes of Symptoms.* Berkeley, University of California Press, 1974.

69. Meyerhof M: Sur Un Glossaire de Matière Médicale Arabe Composé par Maimonide. *Bull Inst Egypte* 1935; 17:223-235.

70. Meyerhof M: Sur Un Ouvrage Médicale Inconnu de Maimonide. *Mélanges Maspêro* 1935-1940; 3:1-7.

71. Meyerhof J: *Un Glossaire de Matière Médicale, Composé par Maimonide (Sarh Asma al'Uqqar).* Cairo, Memorial Institute Egypte, vol 41, 1940.

72. Rosner F: *Moses Maimonides' Glossary of Drug Names.* Philadelphia, American Philosophical Society, 1979.

FROM THE LIBRARY

MAIMONIDES' TREATISE ON HEMORRHOIDS

. . .Of the local remedies, oil of Indian nuts, castor oil, spikenard oil, and jasmine oil are effective. Bdellium and extract of lead are particularly penetrating. An effective clyster is made of two ounces of garlic water, one ounce of celery water, oil of nuts, and oil of turpentine or castor oil, each one ounce, radishes, one ounce, mixed together, boiled, and used every two or three months. A very good remedy is prepared by taking two ounces of one of the above mentioned oils, one ounce of pomade of dates, and one ounce of blue bdellium; these are heated over a gentle flame and then pounded in a lead mortar, with a quarter of a dram of saffron, in the sun for an entire day (the one who does the pounding sitting in the shade). The pain is readily eased if this concoction is smeared on the "piles." But one should not nullify these methods by sitting unwisely on cold marble, or by douching the anus with cold water, except in the summer.

Chapter six deals with methods of combating the more severe types of hemorrhoids. If the situation is very urgent, and the patient can stand it, bleeding is resorted to; but if the patient is too weak, cupping-glasses are placed between the hips, and in addition the internal and external remedies are used. Another valuable procedure is to sit in warm water in which leaves of althea or melilotus or linseed or camomile or cottonseed have been steeped. A good local application consists of starch mixed with sesame oil, fat of duck or hen, and a little saffron. Another formula contains one ounce of oil of apricot seed, in which are dissolved two drams of balsam, two drams of bdellium, oil of roses, egg yolk, fat of duck or hen, and saffron. Ibn Wafid boils one ounce of oil of roses, well perfumed, with three ounces of white wax. Arrazi kneads two drams of zinc oxide and one dram of Indian lycium into an ointment made of Ibn Wafid's mixture. Avicenna had good results with a preparation made of two drams each of serafinum, and bdellium, one dram of balsam, one half dram of opium, one and a half ounces of oil of apricot seed, dissolved together over the fire, to which one-half of a dram of powdered castoreum is added. Finally, Maimonides states that the fat of crocodiles mixed with oil of roses is said to be especially alleviative . . . .

This is the substance of Maimonides' treatise on hemorrhoids. The regulation of diet, the listing of foods indicated and contra-indicated, the local and systemic remedies, the radical and conservative measures—all these bespeak a decidedly modern trend. And the entire treatise verifies Maimonides' own contention that the aim of the doctor is to prevent illness more than to cure it. . . .

LOUIS J. BRAGMAN, MD
(*NY State J Med* 1927; 27:598-601)

Amebic liver abscess without obvious risk factors

Ashok Patel, md; Sudhakar A. Reddy, md; Francis U. Steinheber, md; B. Khurana, md

Amebic liver abscess is rarely encountered in the northeastern United States. Early recognition of this disease is important, because appropriate management can prevent serious consequences. We report an unusual case of a large amebic liver abscess occurring in a heterosexual man from Brooklyn, NY.

Case Report

A 35-year-old man from Brooklyn presented at Coney Island Hospital with severe pain in the right lower chest and in the right upper quadrant of the abdomen, radiating to the shoulder, as well as fever of four months' duration. He did not have a history of either cough or diarrhea. He had been treated by a private physician with antibiotics for a presumed pneumonia.

There were no previous reports of diarrhea, pneumonia, fever, liver disease, or biliary tract disease, nor had he engaged in homosexual behavior. He lived with his girlfriend, who was originally from Mexico. Neither had been to Mexico in the preceding eight years. He had not visited any place outside of New York City and its suburbs.

Physical examination revealed a febrile young man in severe distress. Breath sounds were decreased over the lower fields of the right lung. The liver was palpable 5 cm below the right costal margin in the mid-clavicular line, had a smooth surface with a sharp edge, and was exquisitely tender to palpation. Laboratory analysis revealed a white blood cell count of $24{,}300/mm^3$ (no eosinophils on differential), mild anemia with a hemoglobin of 10.7 g %, and thrombocytosis with a platelet count of $752{,}000/mm^3$.

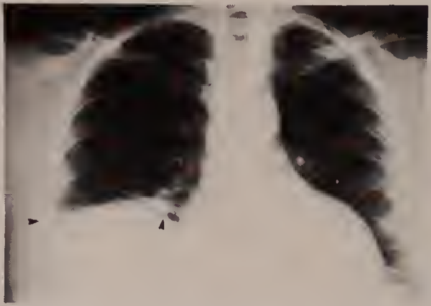


FIGURE 1. Chest roentgenogram shows elevated right hemidiaphragm with some pleural reaction.

The levels of electrolytes, blood glucose, blood urea nitrogen, and creatinine were all within reference limits. The alkaline phosphatase count was mildly elevated at 205 U/L (normal, up to 45 U/L). The electrocardiogram was normal. A chest roentgenogram revealed a normal cardiac silhouette with a markedly elevated right hemidiaphragm (Fig 1). The patient underwent ultrasonography of the abdomen, which revealed a large unilocular cystic lesion in the right lobe of the liver.

Needle aspiration and percutaneous catheter drainage of the liver abscess were performed under computed axial tomographic guidance (Figs 2, 3). The procedure yielded about 1,300 cc of pinkish, thick fluid. No organisms were seen on a routine Gram stain, and aerobic and anaerobic cultures were negative.

The patient's symptoms improved immediately following the procedure. He was initially treated with antibiotics (ampicillin, 2 g intravenously every six hours; nafcillin, 2 g intravenously every six hours; gentamycin, 80 mg intravenously every 12 hours; and metronidazole, 500 mg intravenously every six hours) pending cultures and because of the clinical suspicion of amebic liver abscess. Serologic tests for *Entamoeba histolytica* by hemagglutination inhibition assay were significantly positive. The patient was then treated with a course of metronidazole, 750 mg by mouth three times a day for ten days; and iodoquinol, 650 mg by mouth three times a day for 20 days.

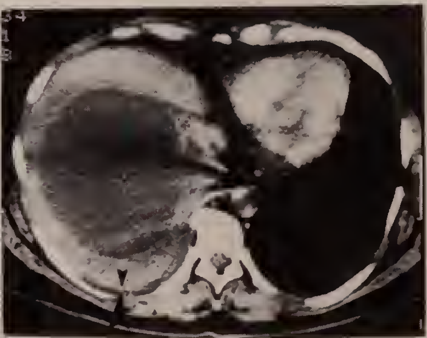


FIGURE 2. Computed axial tomographic scan of abdomen shows intrahepatic abscess measuring 12 cm in anteroposterior, 11 cm in transverse, and 10 cm in cephalocaudal dimension. A small right pleural effusion can also be seen.

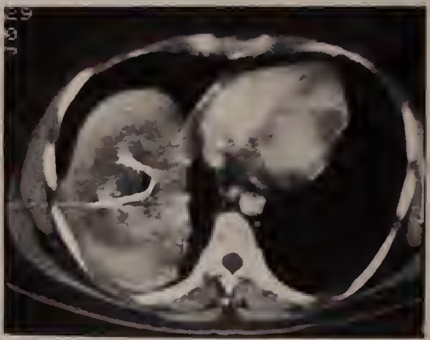


FIGURE 3. Computed axial tomographic scan of liver shows pig tail catheter in place after drainage of the abscess.

Follow-up examination of his girlfriend's stools revealed trophozoites of *Entamoeba histolytica*. On questioning, the girlfriend said that she had had intermittent bloody diarrhea. Additional questioning revealed that they had frequented Mexican restaurants.

Discussion

In a recent study from New York City, the incidence of amebic liver abscess in the hospital population was reported as 0.002%.[1] The majority of the patients were emigrants from areas in which amebiasis is endemic. Other categories of patients included Americans who had recently visited endemic areas, and male homosexuals with multiple sex partners.[2,3] In a study from Chicago, three of 35 patients with amebic liver abscess had none of the usual predis-

---

From the Departments of Medicine (Drs Patel and Reddy), Gastroenterology (Dr Steinheber), and Radiology (Dr Khurana), Coney Island Hospital, Brooklyn, NY.

Address correspondence to Dr Patel, Chief Resident, Department of Medicine, Coney Island Hospital, Brooklyn, NY 11235.

posing conditions.[4] The patient described here was a native Brooklynite, whose most likely sources of infection were his girlfriend, whose stools were positive for trophozoites of *Entamoeba histolytica*, and/or food handlers in any of the restaurants serving Mexican food that he had frequented.

As in most cases of amebic liver abscess, there was no history of intestinal amebiasis in this patient. The diagnosis was suggested by the presence of fever, pain in the right upper quadrant of the abdomen radiating to the right shoulder, hepatomegaly, and abnormalities of the right hemithorax on physical examination and on roentgenography. The fact that all the symptoms and signs had been there for a long period of time excluded the possibility of any malignant hepatic neoplasm. Lack of a cough made pneumonia unlikely.

Ultrasonography and computed axial tomography are not only effective tools for the diagnosis of liver abscess, but are also effective techniques for guided aspiration and percutaneous catheter drainage of the characteristic "anchovy sauce"-looking fluid.[5] The indirect hemagglutination inhibition assay has been reported to be accurate in up to 100% of cases.[4]

Entamoeba histolytica is a ubiquitous intestinal parasite of humans in the tropical climates. It most often causes diarrhea with blood and mucus because of colonic mucosal invasion. In some patients, the trophozoites are more invasive and migrate to the liver via the portal venous system and the lymphatic system. Rarely, hepatic involvement may occur by direct extension. An initial mild portal triaditis eventually leads to the formation of an abscess. Complications secondary to amebic liver abscess are infrequent, occurring in less than 15% of cases. They include rupture into the peritoneal cavity, perforation to the exterior, and rupture into the pleural cavity with formation of a bronchopleural fistula. Even more rare are cases of amebic brain abscess.[6]

The mainstay of therapy is metronidazole. However, since many cases of amebic liver abscess resistant to metronidazole alone have been reported, most physicians prefer to add iodoquinol to the regimen.[7] In most cases, medical therapy alone will suffice, but in selected cases, aspiration, percutaneous catheter drainage, and sometimes even surgical drainage may become necessary.

Amebiasis and amebic liver abscess are being reported with increasing incidence, especially in immigrant and male homosexual populations of New York City.[8] Less frequently, amebic liver abscesses have been found in native Americans who have visited endemic areas. It is logical to presume that more people in urban areas without the ordinary risk factors will be exposed to and contract amebiasis.[9] The diagnosis of amebic liver abscess must be kept in mind in the appropriate clinical setting.

REFERENCES

1. Greenstein AJ, Barth J, Dicker A, et al: Amoebic liver abscess: A study of 11 cases compared with a series of 38 patients with pyogenic liver abscess. *Am J Gastroenterol* 1985; 80:472-478.
2. Conter RL, Pitt HA, Tompkins RK, et al: Differentiation of pyogenic from amoebic liver abscess. *Surg Gynecol Obstet* 1986; 162:114-120.
3. Pomerantz BM, Marr JS, Goldman WD: Amoebiasis in New York City 1958-1978: Identification of the male homosexual high risk population. *Bull NY Acad Med* 1980; 56:232-244.
4. Abuabara SF, Barrett JA, Hau T, et al: Amoebic liver abscess. *Arch Surg* 1982; 117:239-244.
5. Adams EB, Macleod IN: Invasive amoebiasis. II. Amoebic liver abscess and its complications. *Medicine* 1977; 56:325-334.
6. Haaga JR, Weinstein AJ: CT-guided percutaneous aspiration and drainage of abscesses. *AJR* 1980; 135:1187-1194.
7. Thompson J, Forlenza S, Verma K: Amoebic liver abscess: A therapeutic approach. *Rev Infect Dis* 1985; 7:171-179.
8. Marr JS: Amebiasis in New York City: A changing pattern of transmission. *Bull NY Acad Med* 1981; 57:188-199.
9. Imperato PJ: Conclusions about amebiasis. *Bull NY Acad Med* 1981; 57:240-242.

Renal adenocarcinoma in the pediatric age group

THOMAS KAUFMANN, MD, NORWIN H. BECKER, MD

Renal cell carcinoma in infancy and childhood is an extremely rare entity. Catheart[1] in 1929 was the first to report renal adenocarcinoma in a 15-year-old child. Bell,[2] in a review of 373 renal tumors, failed to find a case in the first decade of life. Gross[3] did not encounter a single case in 30 years at the Boston Children's Hospital. Riches et al,[4] in a review of 1,746 cases of renal cell carcinoma, disclosed five cases in patients under the age of 20 (the youngest was 11 years old), with an incidence of 0.3% for all renal tumors. In reviewing the records at Montefiore Medical Center from 1972 to 1985, out of 140 cases of renal cell carcinoma, we found only one patient with hypernephroma during the first two decades of life.

This case report, together with a review of the literature, describes similarities and differences between pediatric and adult renal cell carcinomas.

CASE REPORT

A seven-and-a-half-year-old boy was admitted to the pediatric service of Montefiore Medical Center in 1981 with a one-week history of painless, gross hematuria. His medical history was unremarkable. On examination, he was found to be a well-developed, healthy-appearing child. His vital signs were within normal limits. A large, firm, mildly tender mass was palpable in his left abdomen. An excretory urogram (IVP) revealed a focally calcified left renal mass occupying the lower pole of the left kidney, with obstructive changes of the caliceal system of the upper pole. Nephrosonography disclosed the cystic nature of this mass. The differential diagnosis included Wilms tumor, adenocarcinoma, or cystic teratoma. An abdominal aortogram and bilateral renal arteriogram demonstrated that the mass was avascular. At this time, Wilms tumor was thought to be the most likely diagnosis.

An exploratory laparatomy, including a

From the Department of Pathology, Montefiore Medical Center and Albert Einstein College of Medicine, Bronx, NY.

Address correspondence to Dr Kaufmann, Resident, Department of Radiation Oncology, Beth Israel Medical Center, First Ave at 16th St, New York, NY 10003.

left nephroureterectomy and left aortic lymph node dissection, were performed, showing a large cystic mass involving most of the kidney.

Gross examination of the specimen revealed a 5.5 × 7 × 3.8-cm cystic mass which contained red-brown fluid located in the medial margin of the kidney (Fig 1). Multiple white-gray sessile nodules measuring, on average, 2 cm were attached to the inner surface of the cyst. The inner lining of the cyst was focally calcified, and the renal pelvis was compressed by the cystic tumor mass. Microscopic examination revealed a clear cell adenocarcinoma (Fig 2), Stage I, Grade 2, according to the grading system described by Fuhrman et al.[5] All five lymph nodes were negative for tumor.

The patient made an uneventful recovery, and no further treatment was required. A five-year follow-up showed no evidence of metastasis.

DISCUSSION

Renal cell carcinoma, up to the second decade of life, is very unusual. The youngest patient ever reported was a three-and-a-half-month-old baby.[6] The tumor has been found in many racial groups.[7] The 3:1 male predominance in adults[8] has not been found in children.

Bilateral renal tumors have been reported,[9] but, as in adults, it is not known if they are coexisting primary tumors or represent metastatic disease.[10]

Some children have had urogenital anomalies such as ectopic ureter[11] or horseshoe kidney.[12] Coexisting primary tumors such as neuroblastoma[13] and ependymoma[14] have also been reported. Development of renal adenocarcinoma years after successful treatment of Wilms tumor has been observed in children[15] and in adults.[16] Von Hippel-Lindau disease has been seen in adults, but not in children.[8,10] Genetic susceptibility has been suggested, and a familial case of renal cell carcinoma has been reported.[17] In another study, no consistent karyotypic anomalies were found.[18] It appears that correlation with developmental defects or chromosomal translocations is rare.

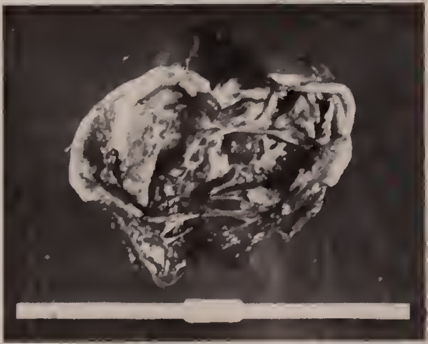


FIGURE 1. Resected kidney showing cystic and nodular tumor mass.

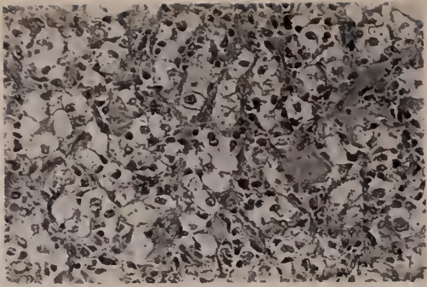


FIGURE 2. Photomicrograph illustrating sheets of cells with clear cytoplasm and enlarged, hyperchromatic nuclei with prominent nucleoli (hematoxylin-eosin stain; original magnification × 100).

The most common clinical signs are a palpable abdominal or flank mass and hematuria.[19] Hematuria should be stressed, as it occurs more often in hypernephroma than in Wilms tumor. Since a mass is more easily palpable in a child's abdomen or flank, renal adenocarcinoma in children is not necessarily associated with metastatic disease. However, most adults who have a palpable mass on presentation already have metastases.[8]

Booth[6] noted the frequency with which onset in children is precipitated by minor trauma, causing either hematuria, severe flank pain, or an acute abdomen. Spontaneous rupture occurs in both adults and children.[20,21]

Constitutional symptoms, such as loss of weight, anorexia, nausea, vomiting, and weakness, occur with equal frequency in both adults and children. No cases with endocrine effects or polycythemia have been recorded, but hepatic dysfunction and abnormalities in several coagulation factors have been described.[22] Hypertension has been noted in 6% of cases.[14] The sites of metastases are similar in both age groups, but involvement of the renal vein seems to occur much more frequently in adults.[23] The duration of symptoms is not necessarily correlated with survival, as survival has been reported among patients who were treated one to two years after the appearance of symptoms.[8,14]

Ultrasound is useful in differentiating between neoplasm, obstructive uropathy, or congenital anomaly, and angiography, as demonstrated by Shanberg et al,[24] may be helpful in making the definitive diagnosis. The typical renal cell carcinoma is a well-vascularized tumor which may exhibit hypervascularity, tumor vessels, and arteriovenous fistulas on angiography. However, it should be remembered that 6% of adult renal cell carcinomas may be avascular.[25] Computed tomography plays an important role in the staging and diagnosis of renal cell carcinoma.

The histopathologic features of pediatric renal cell carcinomas are essentially similar to those of the adult form.[26]

The primary mode of treatment for pediatric renal cell carcinoma is radical nephrectomy. In adults, more conservative surgical treatment, such as partial nephrectomy, is now being employed in low-stage and low-grade tumors.[27] However, there is no evidence in the literature to suggest that this surgical procedure could be employed successfully in the pediatric age group. Radiation therapy and chemotherapy can be added when a curative operation cannot be performed, but, as in adults, these measures have not been shown to improve survival rates significantly. Adoptive immunotherapy (interleukin-2) is still experimental.

It is difficult to compare survival rates in both age groups since the number of cases in children is limited; most patients were not followed long enough for a conclusive analysis. Nevertheless, a number of long survivals have been reported,[28] the longest being 24 years.[29]

There is no evidence to suggest that the biological behavior of these tumors is different in children than in adults. Limited extent of disease at diagnosis offers an optimistic outlook. According to Dehner et al,[30] the presence or absence of renal vascular invasion by tumor is, in children, the single most important anatomic feature in predicting the probable course of the disease. Therefore, one may conclude that tumor staging and grading are the most important factors in the prognosis of renal adenocarcinoma in the pediatric age group.

REFERENCES

1. Catheart E: An unusual case of renal tumor. *Mayo Clin Proc* 1929; 4:342–343.

2. Bell ET: Renal disease, in *Textbook of Pathology*. Philadelphia, Lea and Febiger, 1946, p 433.

3. Gross RE: *Surgery of Infancy and Childhood*. Philadelphia, WB Saunders Co, 1953, p 592.

4. Riches EW, Griffiths IH, Thackray AC: New growths of the kidney and ureter. *Brit J Urol* 1951; 23:297–356.

5. Fuhrman SA, Lasky LC, Limas C: Prognostic significance of morphologic parameters in renal cell carcinoma. *Am J Surg Pathol* 1982; 6:655–663.

6. Booth CM: Renal parenchymal carcinoma in children. *Br J Surg* 1986; 73:313–317.

7. Schellhammer PF, Smith MJ: Renal cell carcinoma in children. *South Med J* 1973; 66:1345–1350.

8. Bennington JL, Beckwith JB: Tumors of the kidney, renal pelvis and ureter, in *Atlas of Tumor Pathology*. Washington DC, Armed Forces Institute of Pathology, 1975, p 108.

9. Kobayashi A, Hoshino H, Ohbe Y, et al: Bilateral renal cell-carcinoma. *Arch Dis Child*

1970; 45:141-143.
10. Herschorn S, Hardy BE, Churchill BM: Renal cell carcinoma in children. *Can J Surg* 1979; 22:412-418.
11. Hogan JF Jr, Simons CE Jr: Renal cell adenocarcinoma in a five-year-old girl with a reduplicated collecting system and ectopic ureter opening into urethra. *J Urol* 1957; 78:212-215.
12. Fisher RG, Granmayeh M, Wallace S, et al: Renal adenocarcinoma in adolescence and childhood: Emphasis on angiographic findings. *J Urol* 1977; 118:83-86.
13. Cassady JR, Filler R, Jaffe N, et al: Carcinoma of the kidney in children. Results of an interdisciplinary approach to management. *Radiology* 1974; 112:691-693.
14. Castellanos RD, Aron BS, Evans AT: Renal adenocarcinoma in children: Incidence, therapy and prognosis. *J Urol* 1974; 111:534-537.
15. Breatnach F, Androulakakis PA: Renal papillary adenocarcinoma following treatment for Wilms' tumor. *Cancer* 1983; 52:520-523.
16. Banner MP, Bleshman MH, Novicki DE: Renal cell carcinoma in a patient successfully treated for Wilms's tumor. *Am J Roentgenol* 1977; 128:77-80.
17. Cohen AJ, Li FP, Berg S, et al: Hereditary renal-cell carcinoma associated with a chromosomal translocation. *N Engl J Med* 1979; 301:592-595.
18. Kantor AF, Blattner WA, Blot WJ, et al: Hereditary renal cell carcinoma and chromosomal defects [letter]. *N Engl J Med* 1982; 307:1403-1404.
19. Hicks CC, O'Brien DP 3d, Majmudar B, et al: Hypernephroma in children. *Urology* 1975; 6:598-602.
20. Ross HB: Retroperitoneal haemorrhage from a ruptured hypernephroma. *Brit J Surg* 1962; 50:266-267.
21. Futrell JW, Filston HC, Reid JD: Rupture of a renal cell carcinoma in a child: Five-year tumor-free survival and literature review. *Cancer* 1978; 41:1565-1570.
22. Ward JS, Meddleton RG: Renal-cell carcinoma in children. *Urology* 1973; 2:50-52.
23. Nygaard KK, Simon HB: Hypernephroma in children. Review and case report. *Arch Surg* 1974; 108:97-100.
24. Shanberg AM, Srouji M, Leberman PR: Hypernephroma in the pediatric age group. *J Urol* 1970; 104:189-192.
25. Watson RC, Fleming RJ, Evans JA: Arteriography in the diagnosis of renal carcinoma. Review of 100 cases. *Radiology* 1968; 91:888-897.
26. Nourse MH, Yurdin DH: Renal cell carcinoma in children. *J Urol* 1959; 82:21-25.
27. Novick AC: Role of conservative surgery for patients with bilateral kidney tumors, in TL Ratcliff, WJ Catalona (eds): *Urologic Oncology*. Boston, Martinus Nijhoff Publishers, 1984.
28. Marcus R, Watt J: Renal carcinoma in children. *Brit J Surg* 1966; 53:351-353.
29. Nicholls MF: The behaviour of tumours. *J Roy Coll Surg* 1960; 5:253-268.
30. Dehner LP, Leestma JE, Price EB Jr: Renal cell carcinoma in children: A clinicopathologic study of 15 cases and review of the literature. *J Pediat* 1970; 76:358-368.

See also pp 638-640.

Childhood acoustic neuroma

KENNETH F. MATTUCCI, MD; WALTER M. GLASS, MD; MICHAEL SETZEN, MD;
WILLIAM LEVIN, MD

Acoustic neuroma is rare in children less than 16 years of age. At this time, there have been 12 reported cases of acoustic neuroma in children (unrelated to neurofibromatosis) in the world's otologic and neurosurgical literature.[1-3] These acoustic neuromas are histologically and clinically identical to the acoustic neuromas of neurofibromatosis. Krause[4] described a seven-and-a-half-year-old child with a unilateral acoustic neuroma and recommended that very young children be followed closely for occurrence of acoustic neuroma on the contralateral side. The youngest child reported with acoustic neuroma was one year of age.[5] Cerebellopontine angle lesions other than acoustic neuromas are more common in the pediatric population; they consist of cysts, granulomas, aneurysms, glomus tumors, ependymomas, osteomas, meningiomas, and cholesteatomas.

CASE REPORT

A ten-year-old boy presented with a history of left parietal headaches for approximately one year and a six-month history of a slowly progressive left-sided hearing loss. The patient had had chicken pox three years before, but reported no hearing difficulty after infection. There was no known ototoxic drug exposure, no otologic or head trauma, and no family history of hearing loss or von Recklinghausen disease.

Examination revealed no neurologic deficits except for a profound left-sided sensorineural hearing loss (Fig 1). Two faint cafe-au-lait spots were noted, each less than 1 cm in diameter. A computed tomographic (CT) scan (Fig 2) disclosed a 4.5 × 3.5-cm enhancing lesion at the left cerebellopontine angle extending into the internal auditory canal, with widening of the canal. An area of decreased attenuation was noted within the lesion, most likely representing necrosis or hemorrhage. The fourth ventricle was displaced to the right. There was no evidence of supratentorial hydrocephalus. The anteroposterior diameter of the left internal auditory canal was 1.4 cm, and that of the right was 0.4 cm. These findings, coupled with the sensorineural hearing loss, strongly suggested an acoustic neuroma, although the diagnosis of a meningioma was also considered.

The patient underwent a suboccipital excision of this intracranial mass. Microscopic examination noted classical Antoni Type A cells and confirmed a neurilemmoma

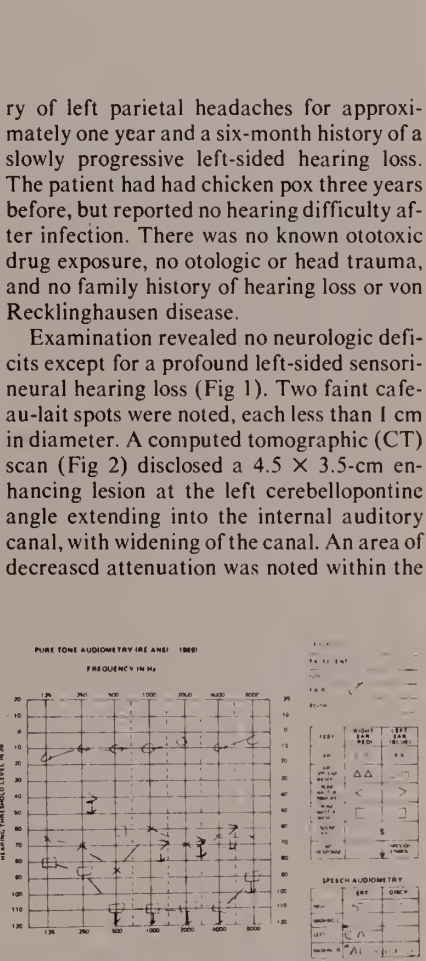


FIGURE 1. Audiogram demonstrating a profound left sensorineural hearing loss.

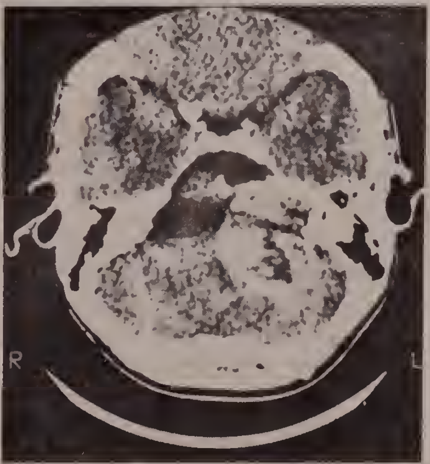


FIGURE 2. CT scan showing a 4.5 × 3.5-cm mass of the left cerebellopontine angle.

From the Department of Otolaryngology, North Shore University Hospital, Manhasset, NY (Drs Mattucci, Glass, and Setzen), and the Department of Otolaryngology, New York Eye and Ear Infirmary, New York, NY (Dr Levin).
Address correspondence to Dr Mattucci, 333 East Shore Rd, Manhasset, NY 11030.

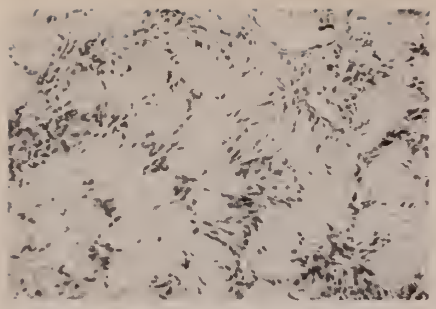


FIGURE 3. Photomicrograph depicting a neurilemmoma of the eighth cranial nerve.

(schwannoma) of the eighth cranial nerve (Fig 3).

After surgery, the child had some diplopia, balance difficulties, and a head tilt to the left with mild left-sided weakness. In addition, he had partial facial weakness on the left side that was peripheral in nature, and a profound sensorineural hearing loss. The facial paralysis and imbalance have improved during the six-month post-operative period in which the patient has been observed.

Discussion

The diagnostic skills of the otologist are challenged when a child presents with a unilateral sensorineural hearing loss. One must consider all of the genetically acquired syndromes, congenital anomalies of the temporal bone, as well as traumatic, ototoxic, infectious, and neoplastic causes. Our literature search identified only 12 cases of isolated acoustic neuroma in children, not related to neurofibromatosis. Given the rarity of this lesion, no one author has had experience with a substantial number of cases. The reports of acoustic neuromas in children are only case reports, or those cited in review series of intracranial tumors.

In 1985, Barrs et al[6] reviewed 305 patients with acoustic neuromas, and Harner and Ebersold[7] reviewed the management of 160 patients with acoustic neuromas. There were no patients reported in either series who were less than 14 years of age.

Although acoustic neuroma is very rare in children, it is the most common cerebellopontine angle tumor in the adult population. These benign neoplasms are most often found unilaterally and usually follow a fairly uniform rate of growth. Patients with von Recklinghausen disease frequently have bilateral schwannomas[8]; this condition is transmitted by autosomal dominant genes and may account for 1–4% of all acoustic neuromas. Hardy and Crowe[9] discovered six schwannomas in 250 temporal bones and estimated therefore that approximately 2% of the general population may suffer an acoustic neuroma.

Acoustic neuromas most often originate from the schwann cells of the vestibular division of the eighth cranial nerve and may be associated with sensorineural hearing loss and tinnitus. Further audiometric study may demonstrate abnormal discrimination, tone decay and/or reflex decay, and abnormal auditory brain stem response (ABR). Vestibular testing may display a canal paresis, and there may be widened internal auditory canals on radiographic examination. Surgically proven acoustic neuromas, however, have been documented in patients with no hearing loss and no sign of retrocochlear pathology or abnormality in vestibular testing.

The earliest clinical symptom of an acoustic neuroma in children, as in adults, is usually a progressive sensorineural hearing loss. However, as so often is the case with any type of unilateral hearing loss and especially that in children, the deficit may go unnoticed by the child or the parents. The suspicion of an intracranial lesion comes only after other more overt signs of space-occupying lesions become apparent, such as gait instability, nystagmus, and cranial nerve deficits. Headache is not uncommon in patients with acoustic neuroma, especially with neuromas of large size. The mechanism is unclear and the headaches often occur in the absence of increased intracranial pressure.

In patients presenting with a unilateral sensorineural hearing loss, tinnitus, and/or signs of possible retrocochlear disease, a careful and complete evaluation of the audio-vestibular pathways is mandatory. Traditionally, this included complete audiometric evaluations, tympanometry, vestibular function tests (electronystagmography), radiographic evaluation of the internal auditory canals (conventional and/or tomography), followed by posterior fossa myelography if indicated. More recently, clinicians have used ABR audiometry and enhanced CT. Enhanced CT can accurately identify more than 97% of tumors greater than 1.5 cm. ABR can be 98% accurate in the diagnosis of acoustic neuromas, although it is of little value in patients with hearing loss greater than 70 decibels. Depending on the clinical situation, the use of ABR and/or enhanced CT will identify nearly all acoustic tumors. Magnetic resonance imaging (MRI) with surface coils, although in the early phases of development, may actually prove to be the most accurate test available for small tumors, some of which may be missed on CT examination. MRI is of particular importance in the pediatric population, since there is no exposure to radiation, and no dye or air is injected.

Acoustic neuromas not associated with von Recklinghausen disease represent approximately 8% of all primary intracranial tumors in the adult population but are rare in children. They are one of several mass lesions more commonly encountered in the cerebellopontine angle in children. These schwannomas may more often be found in patients with neurofibromatosis and in, these instances, frequently occur bilaterally. It may, at times, be difficult to diagnose von Recklinghausen disease. Cafe-au-lait spots are commonly found in otherwise normal patients, but it is generally agreed that the presence of more than six spots, each greater than 1.5 cm in diameter, is virtually pathognomonic of neurofibromatosis (von Recklinghausen disease).

References

1. Craig WM, Dodge HW Jr, Ross PJ: Acoustic neuroma in children. Report of 2 cases. *J Neurosurg* 1954; 11:505–508.
2. Bjorkesten GAF: Unilateral acoustic tumours in children. *Acta Psychiat Neur Scand* 1957; 32:1–5.
3. Rushworth RG, Sorby WA, Smith SF: Acoustic neuroma in a child treated with the aid of pre-operative arterial embolization. *J Neurosurg* 1984; 61:396–398.
4. Krause CJ, McCable BF: Acoustic neuroma in a 7 year old girl. Report of a case. *Arch Otolaryngol* 1971; 94:359–363.
5. Fabian A, Croveri G, Torta R: Neurinoma della fossa posteriore in un bambino di un anno. *Acta Neurol (Napoli)* 1975; 30:218–222.
6. Barrs DM, Brackman DE, Olson JE, et al: Changing concepts of acoustic neuroma diagnosis. *Arch Otolaryngol* 1985; 111:17–21.
7. Harner SG, Ebersold MJ: Management of acoustic neuromas 1978–1983. *J Neurosurg* 1985; 63:175–179.
8. Anderson MS, Bentinck BR: Intracranial schwannoma in a child. *Cancer* 1972; 29:231–234.
9. Hardy M, Crowe S: Early asymptomatic acoustic tumor: Report of 6 cases. *Arch Surg* 1936; 32:292–301.

See also pp 640–641.

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Limb salvage with sarcomas

TO THE EDITOR: Steinherz et al[1] in their case report and Karakousis in his commentary[2] emphasize the importance of the correct diagnosis and treatment of epithelioid sarcomas. A number of authors have emphasized the importance of limb salvage with sarcomas.[3,4] However, some surgeons continue to opt for primary amputation even when limb salvage is possible. Primary amputation imposes physical, psychological, social, and occupational handicaps on patients. The five-year survival rate for patients with known metastasis on whom combined treatment is used is now 20–30%.[5] Limb salvage can be carried out for both primary soft tissue sarcomas and recurrent sarcomas by persons experienced in the field. I share and practice Dr Karakousis' philosophy. While ten-year results are pending we will continue to treat sarcomas with combination therapy using limb salvage.

MUHAMMAD ABU HENA, MD
632 New Scotland Ave
Albany, NY 12208

---

1. Steinherz LJ, Rosen G, Steinherz PG, et al: Isolated cardiac metastatic recurrence of epithelioid sarcoma after two and a half disease-free years. *NY State J Med* 1987; 87:231-233.
2. Karakousis CP: Epithelioid sarcoma. *NY State J Med* 1987; 87:197-198.
3. Karakousis CP, Emrich LJ, Rao U, et al: Feasibility of limb salvage and survival in soft tissue sarcomas. *Cancer* 1986; 57:484-491.
4. Eilber FR, Mirra JJ, Grant TT, et al: Is amputation necessary for sarcomas? A seven-year experience with limb salvage. *Ann Surg* 1980; 192:431-438.
5. Huang MN, Edgerton F, Takita H, et al: Lung resection for metastatic sarcoma. *Am J Surg* 1978; 135:804-806.

Koplik spots and echo 9 virus

TO THE EDITOR: I read the article on adult measles in the July issue of the *Journal.*[1] I am currently in the process of writing a paper on Koplik spots and noted that reference was made that echo 9 virus can produce lesions that mimic Koplik spots.

We have had several epidemics of echo 9 disease but have never seen Koplik spots with same. I would greatly appreciate any references which you can supply to me regarding Koplik spots in other than rubeola illnesses.

DAVID ANNUNZIATO, MD
Associate Chairman
Department of Pediatrics
Nassau County Medical Center

Associate Professor of
Clinical Pediatrics
State University of New York
Health Sciences Center at
Stony Brook

---

1. Flynn JT: Adult measles. *NY State J Med* 1987; 87:391-397.

In reply. Reference is made to the relationship of echo 9 virus to lesions resembling Koplik spots in *Principles and Practice of Infectious Diseases.*[1] In the paragraph entitled, "Rubelliform or Morbilliform Exanthems" on page 816, the sentence which confirms the occurrence of an enanthem resembling Koplik spots with echo 9 viral infection is not directly referenced. It is possible that this statement reflects the personal experience of the author of this section of the source-text.[1] I have not been able to find other confirmatory data in this matter.

The staff of the *Journal* is always pleased to be of service to the research interests and to the continuing medical education needs of its readership.

JOHN T. FLYNN, MD
Consulting Editor

---

1. Mandell GL, Douglas RG Jr, Bennett JE (eds): *Principles and Practice of Infectious Diseases,* ed 2. New York, John Wiley and Sons, 1985, pp 816, 823-824.

Measles rash

TO THE EDITOR: In the July issue of the *Journal* it is stated in the clinical conference on adult measles that "the rash of measles is said to have a superficial texture somewhat like that of sandpaper."[1]

The "sandpaper" or "goose-flesh"-like rash is characteristic of scarlet fever but not of measles.

HELMUT A. MIKK, MD
418 N Main St
Penn Yan, NY 14527

---

1. Flynn JT: Adult measles. *NY State J Med* 1987; 87:391-397.

In reply. Dr Rao and I stand corrected. The sandpaper quality of the skin is characteristic of scarlet fever, rather than measles. The lesions in rubeola (measles) are described as macular to maculopapular, discrete with marked coalescence about the face and thorax, progressing frequently to branny desquamation. The lesions of scarlet fever are described as punctate on an erythematous base, pin-head sized, imparting a sandpaper-like texture to the skin, progressing typically to desquamation.[1]

It is encouraging to realize that the *New York State Journal of Medicine* has interested and meticulous readers who take the time to respond to our efforts, and whose attention to the *Journal's* contents ultimately means better education for all.

JOHN T. FLYNN, MD
Consulting Editor

---

1. Fitzpatrick TB, Freedberg IM (eds): *Dermatology in General Medicine,* ed 3. New York, McGraw Hill, 1987, vol 1, pp 2290, 2293, 2110.

Strategic planning for medical societies

TO THE EDITOR:

"If you don't know where you're going,
How will you ever get there?"
—Richard A. Taylor, MD

Medical societies must, of necessity, plan for the future. It has become an even greater imperative given all of the changes taking place in medicine and medical care. A frequently used tool to accomplish this purpose is strategic planning. Informed members of the medical society should be familiar with the process.

A strategic plan, which is a type of road map, often begins with a mission statement that describes the purpose, hopes, and aspirations of an organization. It is a keynote, a guide for the plan.

The actual plan is usually developed at a retreat. There, the leaders of the organization—past, present, and future—work through the process with the help of a facilitator and resource personnel. They review documents, interviews, and other materials which have been prepared by the resource personnel. The group scans the external environment of the organization with its accompanying opportunities and threats and analyzes the internal milieu of the society with its strengths and weaknesses. In each case, they must evaluate the problems that confront them and develop the techniques they will employ to manage them. Beginning with brainstorming sessions and proceeding to problem-solving and decision-making sequences, the group seeks consensus in determining the direction and the steps they must take to reach an agreed-upon goal.

After the plan is exhaustively critiqued by the group, it is then submitted to the governing body of the organization which carefully studies it and makes recommendations for its implementation. Both the planning group and the governing body add to the plan their intuitive sense of what must be done if it is to succeed.

This intuitive sense of what is attainable is also stressed in the next phase of tactical planning, in which the strategic plan is shared with the operational arm of the organization, the committee structure. It is here that problems may arise because failure to get this level of "stakeholder" involved and committed to the plan may cause it to gather dust in the "Office of Irrelevant Documents." If the plan begins at a state level, it must resonate with the concerns of the county societies as well as the national body if it is to achieve broad acceptance. Other elements such as a marketing plan for membership evolve from the strategic planning process.

The actions of the strategic plan must be monitored and from this feedback steps are taken to modify the design. This is a continuous process which can be of considerable benefit to a medical society.

The leadership of a medical society is confronted with a multiplicity of pressures as it attempts to direct the activities. If the organization is to thrive and move forward, it should sculpt its desired future with a purpose and a focus on priorities. To do this, strategy, the "General's Art," is worth considering.

RANDALL D. BLOOMFIELD, MD
President
Medical Society of the County of Kings
1313 Bedford Ave
Brooklyn, NY 11216

FROM THE LIBRARY

MEDICAL LEADERSHIP

Much has been written on the subject of leadership. Instruction in leadership is given to varied classes of employees ranging from executives at the head of great corporations to boy scouts on the theory that the ability to lead may be developed. The *New York Times* of July 5 has an editorial on the elements of leadership, which says:

"A Chicago University professor has been applying exact scientific methods in trying to find out whether there is such a distinct quality in man as 'leadership.' The word and the thing have been thrown at us in countless ways. Even babes in arms have been minutely studied to see if they bore in their tiny bodies and dawning minds promise of becoming leaders of mankind. But now we are told after these painful researches that 'there ain't no sech a thing.' The Chicago professor has studied sixty men, ranging from the heads of gangs now in prison, to soldiers and business men who were offered as fine specimens of leadership. But they all baffled inquiry. Nothing could be found in them, any more than Mark Twain's man could find in the Jumping Frog, to show superiority over their fellows.

"The conclusion is that this mysterious gift of 'leadership' is largely a delusion. Fortunate environment and lucky opportunities count much more than latent ability. So runs the Chicago professor's logic, but there is one evident weakness in it. The man who succeeded must have had at least the native ability to seize the happy skirts of circumstance when they passed him.

"There is the little indefinable something in one individual that enables him to open his oyster, when it is presented to him, while another merely fumbles at it. This may not be wholly luck or exclusively leadership, but the blend of the two is necessary to success."

The Medical Society of the State of New York is searching for men who will be leaders in County Medical Societies. The editors suggest that leaders are those who do things according to the plan of the preacher in the Book of Ecclesiastes: "Whatsoever thy hand findeth to do, do it with thy might." Lincoln said that he had known many great men and all of them seemed like the rest of us, except in their practical knowledge of one or two subjects which made them leaders.

(*NY State J Med* 1929; 29:901)

Doctors in Science and Society. Essays of a Clinical Scientist

By Sir Christopher Booth. 318 pp, illustrated. Tavistock Square, London, British Medical Journal, 1987. $32.00 (hardcover)

The 18th century is frequently viewed as a dark age in medicine, characterized by quackery, apprentice-trained physicians, bleeding, purging, and endless theorizing and speculation. It has become increasingly evident to serious medical historians, however, that this century of the Enlightenment produced not only many notable physicians, but also many medical scientists who made a number of important discoveries. In part, medicine of the 18th century has suffered from the shadow of the subsequent century in which so many major advances were made. Not surprisingly, 19th century medicine is now viewed as terribly primitive compared to its successor of the 1980s. Judgments concerning the internal merits of an era based solely on comparisons of what came before or afterwards tend to fall far from the mark. Of course comparisons are very useful in charting technical and scientific progress and the development of medical thought. However, a truer picture of the accomplishments of an era emerges when such comparisons are linked to insightful analyses of the era itself and of the men and women who were a part of it.

Sir Christopher Booth, who is currently director of the Clinical Research Centre in Great Britain and who serves as president of the British Medical Association, has written a meticulously researched book that not only carries us back to the 18th century, but also brings us face to face with modern medical technology and the role of modern medical science in society. In so doing he provides readers with a valid historical account of Enlightenment medicine and with clear insights into where we stand today.

Based on research carried out over a period of many years, the essays begin with an examination of the contributions of the Age of Reason to medical discovery. On careful examination this was scarcely a fallow era, since scurvy was conquered through empiricism, digitalis was discovered, vaccination was introduced, and, as the author notes, "both the nature of oxygen and the anaesthetic properties of nitrous oxide [were]... described." In the first chapter, "Clinical Science in the Age of Reason," the author comprehensively yet succinctly lays out the social and medical profiles of the age. The humoral theory of disease causation still dominated medical practice. Yet there were stirrings generated by scientific minds seeking answers that theory alone could not provide. The next chapters present detailed biographies of three outstanding physicians: Samuel Garth, William Hillary, and John Fothergill. Distinct and different from one another in many ways, these three men exemplify their age. Garth, the physician-poet, set to verse a burning medicosocial issue of the day, the setting up of dispensaries for the poor by the Royal College of Physicians. There was much opposition to these dispensaries on the part of apothecaries, who saw them as an economic threat. Garth, a close friend of both John Dryden and Alexander Pope, put the dispensary issue to rest with his poem "The Dispensary," which was published in 1699. As Sir Christopher observes, "the importance of Garth's 'Dispensary,' however, lies not only in its undoubted effect on a prolonged and undignified professional quarrel, but also in its unique contribution to English literature." It provided readers then as it does now with insights into the social and economic dimensions of medical practice of the day. As such it is an important social document. Garth served as physician to the Duke of Marlborough (the one who held such great influence over Queen Anne) and not surprisingly shared his political fortunes, falling from favor at court towards the end of the queen's reign and returning to favor on the accession of George I.

Sir Christopher next examines the life and work of William Hillary, a physician who as he says "enjoyed no outstanding reputation in his lifetime." Hillary wrote one of the first treatises on tropical medicine, *Diseases of Barbados*, published in 1759. What sets Hillary apart from so many of his contemporaries was his advocacy of scientific method. This is evidenced in his writings and observations. While in Barbados, for example, he observed the rising and ebbing of the sea one day in November 1755, which he concluded was due to an earthquake at some distance. Two months later he received confirmation of the terrible earthquake in Lisbon, Portugal, that killed 30,000 people. A prolific author, Hillary's writings display a "clarity of thinking" and a "wholehearted enthusiasm of his belief in the value of a scientific approach to medicine." It was this approach as the author points out that represented the scientific renaissance that was beginning in Britain in the 18th century.

John Fothergill is described in the next chapter and of the three he is perhaps the best known to Americans. A modest man and a Quaker, he was, in the words of his biographer, John Coakley Lettsom, "... more desirous of doing good than of having it known." A keen botanist, he is best known for correlating the clinical features of angina pectoris with pathological findings in the heart at autopsy. Although William Heberden had previously documented the clinical picture, it was Fothergill who established the link between diseased coronary arteries and the clinical syndrome.

Fothergill also played an important role in trying to prevent the American War of Independence. As a Quaker he was by definition a member of the most nonconformist sect of the day. He included among his patients Lord Dartmouth, Secretary of State for the American colonies, and Sir Fletcher Norton, Speaker of the House of Commons. Sir Christopher presents much interesting history about the many efforts by individuals in Britain to redress the wrongs inflicted on the colonies such as the Stamp Act of 1765. Fothergill was in the forefront of these efforts, writing not only letters but also a pamphlet, *Considerations Relative to the American Colonies.* He also met with Benjamin Franklin in London in 1774 in a final attempt to hammer out the differences between Britain and her American colonies. These efforts failed, and by the time Franklin was on his way home the "shot heard round the world" had already been fired.

Two other physicians in addition to John Fothergill are discussed in the chapter, "Three Doctors and the American Revolution." They are Joseph Warren, who died a hero at the Battle of Bunker Hill, and Benjamin Rush, who signed the Declaration of Independence. As Sir Christopher notes, all three were idealists and all three played a role in the events of the American Revolution. Fothergill unsuccessfully tried to prevent it. Warren was "attractive, honest, and sincere, sought no personal glory and was able to give leadership and guidance to the men of Massachusetts at a crucial period of their history." Rush had the courage to sign the Declaration of Independence, gave dedicated service during the Philadelphia yellow fever epidemic of 1793, and lived to see the War of 1812.

At this point in Sir Christopher's book, even firm believers in the distortion that the 18th century produced little in the way of medical progress and few great physicians will have changed their minds. Sir Christopher next provides readers with two delightful chapters that vividly portray the lives of John Fothergill's sister Ann who was mistress of his household; the Willans of Marthwaite; and Dr John Dawson. These chapters are followed by one on the conquest of smallpox which carries the story up until the disease's eradication in 1977.

In chapter 10, the author gives a splendid overview of the development of medical journals in Britain. As he cogently observes, "The story of the publication of medical journals reflects the history of both medical practice and science."

The *Philosophical Transactions* of the Royal Society was the first journal in which physicians published. However, by the middle of the 18th century physicians had become dissatisfied with the general decline of the society's scientific activity. This in turn led the society to introduce peer review of submitted articles prior to acceptance for publication. William Heberden, a noted physician, served as a member of the review committee which generally improved the quality of the publication. It was in Edinburgh, however, that the first medical journal was published. Called *Medical Essays and Observations*, it was put out by the Edinburgh Medical School in 1731. Other journals followed, including the *Medical Transactions* of the Royal College of Physicians in 1767, and *The Lancet*, which began publication on October 5, 1823. Sir Christopher provides an interesting account of the development of these and other journals, including the *British Medical Journal*, which began in 1840 as the *Provincial Medical and Surgical Journal.*

Two chapters in *Doctors in Science and Society* deal with the history of the British Medical Research Council and the British Postgraduate Medical School at Hammersmith, respectively. A final chapter provides an overview of clinical research in Britain today, the author providing many useful and interesting details about funding, the loci of research, and the overall role of investigators in clinical service and teaching.

In order to appreciate where medical science is today, one must have an understanding of where it once was and know something of the roads it travelled. Sir Christopher provides readers with an insightful and fascinating guide to this exciting advance of medical science. He brings to this endeavor not only the skills of a meticulous medical historian and the experience of a distinguished clinical scientist, but also the ability to turn a phrase and coin a metaphor. As a result the book is authoritative, stimulating, beautifully written, and a pleasure to read.

PASCAL JAMES IMPERATO, MD
Editor

Reprinted from the September 1987 issue of Epidemiology Notes (*Vol. 2, No. 6*), *published by the Division of Epidemiology, New York State Department of Health, Albany, NY.*

Tuberculosis and AIDS: Guidelines for management of mycobacterial infections in people with HIV infections

The total number of acquired immunodeficiency syndrome (AIDS) patients in New York State meeting the federal Centers for Disease Control (CDC) surveillance case definition represents only a fraction of the number of people with human immunodeficiency virus (HIV) infection.

The number of reported cases of tuberculosis (TB) did not decline in New York State or the nation in 1985 as had been the trend in previous years. Instead, there was an increase in the incidence of tuberculosis in New York City and upstate New York, which are geographic areas of high and moderate incidence of AIDS. Increased tuberculosis morbidity may be partly explained by the presence of prior latent tuberculosis infection in HIV-infected individuals, which progressed to disease as their immune systems became compromised.

The cellular immune deficiency caused by HIV infection poses special problems for medical management of people with coexisting mycobacterial and HIV infections. The association between mycobacterial disease and HIV infection raises several important public health issues.

Diagnosis of disease

The most common mycobacterial species isolated from patients with AIDS is *Mycobacterium avium* complex (MAC). In groups with a high prevalence of tuberculosis infection, however, disease caused by *M tuberculosis* (MTB) may be more common. Prompt identification of HIV-infected persons who have mycobacterial disease, and determination of whether MAC or MTB is the cause, is critical to the choice of therapeutic regimen and the appropriateness of public health measures.

A tuberculin skin test (Mantoux) should be administered to those people suspected of having mycobacterial infection. A negative reaction should not be presumed to rule out the diagnosis of tuberculosis, however, because the immunosuppression associated with HIV infection may cause false negative results.

Diagnosis is made difficult by the fact that tuberculosis may have an unusual clinical picture in people with HIV infection. Extrapulmonary forms of tuberculosis, particularly lymphatic or disseminated disease, are seen more frequently in people with HIV infection.

Microscopic examination (acid-fast smears) and culture of sputum, urine, blood, lymph node, bone marrow, liver, or other tissue or body fluid as indicated by the clinical picture are essential to establish the diagnosis. When patients have pulmonary TB, it may not be distinguishable from other pulmonary infections on the basis of clinical and radiographic findings. Infiltrates may appear in any lung zone, and cavitation is uncommon. Hilar and/or mediastinal adenopathy is common. The radiographic pattern is more frequently consistent with primary tuberculosis than with reactivation tuberculosis.

Treatment of tuberculosis disease

Chemotherapy should be started whenever acid-fast bacilli are found in a specimen from a patient with HIV infection and there is clinical evidence of mycobacterial disease.

The drug regimen used while awaiting culture identification of the mycobacterial species must be effective against tuberculosis, because tuberculosis can be transmitted to others. An initial drug susceptibility test should always be performed. The treatment regimen should be revised if resistance is found to any drugs being used.

The recommended regimen is isoniazid (INH), 10–15 mg/kg/day up to 300 mg/day; rifampin, 10–15 mg/kg/day up to 600 mg/day; with either ethambutol, 25 mg/kg/day, or pyrazinamide, 20–30 mg/kg/day, given for the first two months. INH and rifampin should be given for a minimum of nine months (three months longer than the recently recommended six-month standard American Thoracic Society regimen) and at least six months after documented culture conversion to negative.

If INH or rifampin is not included in the regimen, treatment should be continued for at least 18 months and at least 12 months after culture conversion to negative. Even after treatment is completed, patients should be followed closely for relapse.

Evaluation of TB patients for HIV infection

Tuberculosis patients should be interviewed to identify risk factors for HIV infection. If any risk factor is identified, voluntary HIV antibody testing should be encouraged. HIV testing is recommended for all patients with severe or unusual manifestations of tuberculosis. HIV serologic status should be determined in order to accurately prescribe tuberculosis drug therapy and to adjust the length of therapy.

Tuberculosis contact investigation

Standard tuberculosis contact investigation procedures should be used for HIV-infected patients found to have tuberculosis.

Preventive therapy

Consistent with recommendations regarding immunocompromised individuals, preventive therapy with INH is recommended for an HIV-infected person of any age if the tuberculin skin test is positive and tuberculosis disease has been ruled out.

The preventive therapy regimen is a single daily dose of INH of 10–15 mg/kg up to 300 mg for adults and 10–14 mg/kg, not to exceed 300 mg, for children. Preventive therapy should be given for six to 12 months.

Evaluation of HIV-infected patients for TB and TB infection

The potential for false negative skin tests makes the identification of tuberculous infection in HIV-infected patients more difficult. The following recommendations should, therefore, be followed:

People with a positive HIV antibody test:

- All should be given a Mantoux method tuberculin skin test as part of their medical evaluation. The sooner the skin test is given in the course of HIV infection, the greater the likelihood of an accurate immune response to the test. False negatives are more likely to occur with deterioration of immune status.
- If the skin test reaction is significant, a chest radiograph should be obtained. If abnormalities are detected, additional diagnostic procedures (ie, sputum for smear and culture) for tuberculosis should be undertaken. If the person's clinical picture suggests extrapulmonary disease, appropriate diagnostic procedures must be undertaken.

People with AIDS or other HIV-related illness:

- All should receive both a Mantoux method tuberculin skin test and chest radiograph as part of the initial medical evaluation.

Treatment of MAC disease

The clinical significance and optimal therapy for MAC disease in HIV-infected patients is not well defined. MAC disease is usually resistant to the regimen recommended for treatment of TB. A regimen including INH, ethambutol, rifabutin, and clofazimine may be successful. The latter two drugs are available only to qualified investigators under investigational new drug protocols. Rifabutin is distributed by CDC (404-329-3670); clofazimine is available through Ciba-Geigy (201-277-5787). This regimen should be given when culture confirmation of MAC has been obtained.

If the patient was initially treated with one of the three drug regimens appropriate for tuberculosis, but is identified by culture to have MAC disease, the regimen may be continued if the patient is responding, but if not, the regimen should be switched to the INH, ethambutol, rifabutin, and clofazimine regimen.

Summary

Tuberculosis screening and diagnostic procedures are difficult to interpret and traditional treatment regimens are altered in people with HIV infection. The following statements outline important potential modifications to be considered in the clinical management of people with tuberculosis and HIV infection:

- To diminish the possibility of a false negative response, TB skin testing should be done as early as possible in any HIV-infected person. A negative skin test reaction should not rule out the possibility of TB infection.
- Chest x-rays are difficult to interpret because of possible other pulmonary infections, lack of cavitation, or infiltrates being demonstrated in all lung fields.
- Extrapulmonary disease is common in HIV-infected people and requires reliance on diagnostic procedures other than chest x-rays.
- Since acid-fast bacilli found on smear can be either MAC or MTB, tissue or fluid culture is extremely important. Only culture can determine the etiologic agent.
- Treatment regimen and preventive therapy recommendations are different for HIV-infected people.

Reporting requirements

Tuberculosis: The provider must follow the reporting requirements for a tuberculosis case or suspect (diagnosis pending) as mandated by public health law to the county/city health official where the provider is located and where the patient resides.

AIDS: The provider must follow the reporting requirements as mandated in Part 24.1, New York State Sanitary Code, by submission of a report to the New York State Department of Health or in New York City to the New York City Department of Health.

HIV counseling and testing

The New York State Department of Health HIV testing policy includes the following principles: The test must be voluntary; fully informed consent must be obtained; pre- and post-test counseling must be provided; and confidentiality of test results must be strictly maintained. More complete information can be obtained by review of New York State Department of Health Public Health Series Memorandum 86-40, "HTLV-III LAV (HIV) Testing in Health Care Facilities," and the *Physician's Guide to HIV Counseling and Testing* published by the New York State Department of Health in March 1987.

References

Centers for Disease Control: Classification system for human t-lymphotropic virus type III/lymphadenopathy-associated virus [HIV] infections. *MMWR* 1986; 35:334–339.

Centers for Disease Control: Diagnosis and management of mycobacterial infection and disease in persons with HTLV-III/lymphadenopathy-associated virus infection. *MMWR* 1986; 35:448–452.

Pitchenik AE, Rubinson HA: The radiographic appearance of tuberculosis in patients with the Acquired Immune Deficiency Syndrome (AIDS) and Pre-AIDS. *Am Rev Respir Dis* 1985; 131:393–396.

Changing patterns in cause specific mortality

For the past 25 years there had been no change in the ranking of the leading causes of death in New York State. The five leading causes had been diseases of the heart,

Difference in Death Rates for Selected Causes: New York State Residents, 1980 and 1985

Cause of Death	No. Deaths 1980	Rate per 100,000	No. Deaths 1985	Rate per 100,000	Difference in Rates	% Difference in Rates
Heart disease	74,090	422.0	70,636	400.0	−22.0	−5.2%
Cerebrovascular disease	12,108	69.0	10,155	57.5	−11.5	−16.7%
Motor vehicle accidents	2,760	15.7	2,041	11.6	−4.1	−26.1%
Other accidents	2,882	16.4	2,370	13.4	−3.0	−18.3%
Cirrhosis of liver	3,576	20.4	2,936	16.6	−3.8	−18.6%
Homicide	2,221	12.7	1,773	10.0	−2.7	−21.3%
Diabetes	3,367	19.2	3,218	18.2	−1.0	−5.2%
Suicide	1,665	9.5	1,544	8.7	−0.8	−8.4%
AIDS	*		1,717	9.7		
Chronic obstructive lung disease	4,216	24.0	5,190	29.4	+5.4	+22.5%
Infectious/parasitic disease	1,237	7.1	1,882	10.7	+3.6	+50.7%
Malignant neoplasms	37,489	213.5	37,997	215.2	+1.7	+0.8%
Nephritis and nephrosis	1,089	6.2	1,435	8.1	+1.9	+30.6%
Central nervous system disorders	737	4.2	1,053	6.0	+1.8	+42.9%
Pneumonia and influenza	5,876	33.5	6,111	34.6	+1.1	+3.3%
Mental disorders	1,321	7.5	1,519	8.6	+1.1	+14.7%

* Separate mortality counts for AIDS did not begin until 1983.

malignant neoplasms, cerebrovascular disease, pneumonia/influenza, and accidents. Data for 1985, however, indicate that the group of illnesses comprising chronic obstructive lung disease accounted for 15% more deaths (5,190) than accidents (4,411), and has replaced accidents as the fifth leading cause of death. In addition, recent data (since 1980) indicate that the five traditional leading causes of death are accounting for a decreasing percentage of the state's total deaths: 78.7% in 1980 compared to 76.0% in 1985. Mortality rates, however, have increased in several other causes or groups of related causes, and there has not been any major reduction in the overall level of mortality.

Many causes of death had significant changes in their rates between 1980 and 1985. Heart disease, the leading cause of death, had a rate of 422 deaths per 100,000 population in 1980 and decreased by 22 deaths per 100,000 population to 400 by 1985. Two other leading causes, cerebrovascular disease and accidents, also showed decreases in their rates. Both components of the total accident figure decreased, with motor vehicle accidents dropping by four deaths per 100,000 population and other (non-motor vehicle) accidents dropping by three deaths per 100,000. The remaining two leading causes, malignant neoplasms and pneumonia/influenza, showed slight increases in their rates.

An important fact that emerges when analyzing these recent patterns is the marked increase in rates observed for certain causes. Most notable is the increase of almost ten deaths per 100,000 population in the rate for acquired immunodeficiency syndrome (AIDS). Prior to 1983, when separate mortality counts for AIDS began, the small number of AIDS deaths were grouped with all deaths from immune system disorders. Chronic obstructive lung disease, as noted above, became the fifth leading cause of death in 1985. The mortality rate rose over five deaths per 100,000 population between 1980 and 1985 and now accounts for over 29 deaths per 100,000 population. The death rate due to infectious and parasitic diseases also rose considerably in that time period, with the 1980 rate of 7.1 deaths per 100,000 increasing to 10.7 deaths per 100,000 in 1985. Data for these and other selected causes appear in the accompanying table.

Available publications

Three new brochures, *How to Use Child Safety Seats—A Simple Guide*, *Child Safety Seats*, and *Motor Vehicle Trauma Is a Major Health Problem*, have been developed by the New York State Department of Health's Injury Control Program and the Bureau of Health Promotion.

These publications are available without charge and can be requested in limited quantities from: Publications, Corning Tower, Room 1084, New York State Department of Health, Albany, NY 12237.

AMERICA'S HEALTH CARE REVOLUTION. WHO LIVES? WHO DIES? WHO PAYS?

By Joseph A. Califano, Jr. 241 pp. New York, Random House, Inc, 1986. $17.95 (hardcover)

Many books are currently being written to teach the American public about the health care system in the United States. The varieties of approaches are as diverse as the backgrounds of the authors. Joseph Califano, Jr, has had a unique perspective on health care delivery in his roles as President Lyndon Johnson's Special Assistant for Domestic Affairs, later, as President Carter's Secretary of Health, Education, and Welfare, as a leader in Chrysler's struggle to reduce its health care costs, and as an attorney for both consumers as well as providers of health care. This book is Mr Califano's attempt to discuss the "revolutionary forces" affecting the health care system.

Reading it is an informative way to learn about how a knowledgeable health consumer thinks about our health care system. The style is a combination of eye-opening statistics along with anecdotal story-telling. This mix of data with emotional "case presentations" to illustrate the author's contentions makes a powerful argument for the need for change in the system. In all cases, the change is discussed as clearly beneficial to health care consumers in terms of decreased costs and more efficient and higher quality health care.

After introducing the coming revolution in the initial chapter, the book goes on to describe the different forces involved in the movement toward change. The chapter titled "The Chrysler Story" is a clear tale of how health costs have expanded and how the consumer is carrying the burden through the passing along of costs in the form of higher prices. Evidence and examples of overuse and abuse of health care benefits are offered and the story of dramatic savings is set out in a way to make the reader want to give Mr Califano a round of applause.

The next chapter is an abbreviated and simplified history of how health care has reached the current level of overutilization. The many players are all described and the reader begins to understand the complex interaction of health insurance, provider pressures, and consumer behavior which have created the "health industry colossus." In addition, some of the apparent paradoxes of the American health care system are described. Widely divergent treatment patterns existing in different communities are discussed, and the apparent lack of interest in the preventive areas of smoking cessation, alcohol abuse, and nutrition are noted by the author as displaying doctors' interest in treatment of disease rather than in its prevention or in health promotion.

All segments of the health care industry share the blame for the problems of our current system. Physicians (called "medicine men"), hospitals, (called "temples of the medicine men,") and other health care workers (called "profitable acolytes") each are given a chapter to discuss their vested interests in the current system. The result is described as a noncompetitive system with attempts to control costs turning into a "shell game" of many small steps which may hide costs or transfer costs, but do not really alter them. After this collage of information designed to make the reader sympathetic to his viewpoint, Califano does mention some concepts about payment incentives, the need for compassionate care, and the importance of health maintenance. After reading the initial three quarters of this book, the layperson as well as a large group of providers may recognize some of the basic concepts which will form the backdrop of the future health care delivery system.

The "solution" proposed by Califano is on the last three pages of the book. He suggests (1) that each employer should insure employees for a minimal level of health care and, (2) that the government should provide care for everyone else. Government should help to establish the consensus and the private sector should "lead the effort." This all sounds pretty simple for the outcome of a "revolution" which has required 225 pages to describe.

This book is a broad overview of health care in the US and runs from discussion of the financial incentives of technology to decisions about death and dying. Mr Califano has chosen a broad topic and attempts to cover all areas. This causes a superficiality along with an omission of important areas such as access to health care, the question of what constitutes good health care, and what level of spending the American public is willing to make. In addition, the simple solution of having private industry formulate health care for the employed and government having the responsibility for the care of all others (almost 100 million individuals) will not likely reduce total health care expenditures and will probably emphasize a two-tiered level of care.

If one is looking for a book that summarizes the issues facing health care delivery, *America's Health Care Revolution* is a good, initial text to read. If one is looking for a book that makes proposals for change and supports them in a factual manner within the context of the evolution of the health care delivery system, a more technically structured book would be better.

RICHARD SADOVSKY, MD
State University of New York
Health Science Center
at Brooklyn
Brooklyn, NY 11203

PERSONAL MONEY MANAGEMENT FOR PHYSICIANS

Fourth Edition. By Lawrence Farber. 286 pp, illustrated. Oradell, NJ, Medical Economics Books, 1986. $27.95 (hardcover)

This book is an excellent and comprehensive overview of the financial planning concepts that physicians must consider during their lifetimes. The information is applicable to the vast majority of physicians regardless of the type of practice, medical specialty, length of time in practice, or income level. It is very well organized and presented in concise, manageable chapters.

The author presents technical and complicated material in clear and understandable language. However, the treatment of the subject matter seems intentionally geared to the physician who is less financially sophisticated.

The reader is well advised to realize that no less than 53 aspects of financial decision making are discussed in a mere 273 pages. Therefore, any in-depth coverage of a particular subject is precluded. For this reason, the publisher's description of the content to "set you up to direct and get the most out of all your economic assets," is an overstatement.

This is particularly true with regard to the chapter on investment analysis of stocks, bonds, gold, and tax shelters in oil drilling and real estate. These are all highly complicated areas of wealth accumulation, and the information presented here should in no way be construed as preparing the physician to "direct and get the most out of your economic assets." However, the author clearly indicates that the physician should seek further advice, in addition to pointing out the many common pitfalls of the more popular vehicles for wealth accumulation. Farber also offers the opinion of an investment counselor in reprinting an interview with an individual on investing in the stock market. Again, it is all good information, but not designed to be sufficient preparation for doing one's own investing.

The lack of in-depth coverage notwithstanding, the accuracy of the information is generally quite reliable with two exceptions. First, the timing of the book's publication has made it available in the shadow of tax reform. Because the author treats most subjects superficially, any inaccuracies due to changes in the tax laws are not glaring. However, his statement that depreciation allowances from rental activities in real estate are deductible from all sources of taxable income is now untrue under tax reform.

Secondly, inaccuracies exist in the discussion of how to compute maximum contributions to a qualified retirement plan in chapter 45. However, the author must be given the benefit of the doubt in that his overall treatment of qualified retirement plans is excellent, and the minor inaccuracies are due to his task of explaining a highly technical planning area as simply as possible.

While chapter 14 covers disability income protection insurance very well, the chapter on life insurance could have been more current in light of the many recent changes in life insurance products. In addition to the insurance products mentioned, the physician in the market for life insurance should also investigate the new generation of interest-sensitive and investment-driven products. In the chapter on judging an insurance agent or broker, Farber does not mention the professional certification "Chartered Life Underwriter." This designation is a very important criterion in judging the qualifications of an agent or broker.

Farber encourages the physician to seek out the credentials "Certified Financial Planner" when choosing a financial advisor. However, he should have also included the professional designation "Chartered Financial Consultant" as conferred by The American College in Bryn Mawr, Pennsylvania.

Personal Money Management for Physicians provides a good checklist of those items that physicians should include in their overall financial planning, and gives the reader enough background in each area to be used as a springboard to further action.

CHERYL A. CAMPBELL
BRUCE A. LINGENFELTER, CLU
Pennsylvania Financial Group, Inc
State College, PA 16804

CLINICAL DOPPLER ECHOCARDIOGRAPHY

By Jose Missri, MD. 256 pp, illustrated. New York, Yorke Medical Books, 1986. $45.00 (hardcover)

In the preface the author notes that his book was written to address the need for a text that emphasized the clinical aspects of Doppler echocardiography. His book has achieved this goal, though the price of this success is oversimplification of some aspects of a complex topic. This is not a textbook for the trainee or the practitioner who needs to acquire knowledge of the physical principles on which Doppler echocardiography rests in order to perform or interpret such studies. It is more suited to the practitioner who will be ordering echocardiographic studies and who therefore will benefit from an introduction to the type of results to be obtained and their clinical significance. It details both the clinical questions that can be answered by Doppler echocardiography and the limitations of this diagnostic modality. The text is clearly written and the reference lists are comprehensive and to the point.

The important weakness of this textbook is its illustrations. Given the readership to which this book would be most useful, the illustrations should clearly demonstrate the point being depicted. Too many illustrations require some working knowledge of Doppler recording interpretation to tease out the point or lesion being demonstrated. Additionally, the lack of calibration of the flow velocity scale in most illustrations impedes quick assimilation of the information.

Despite these weaknesses, this textbook would be useful to anyone interested in a quick introduction to clinical Doppler echocardiography. Any reader whose interest is thereby whetted should then proceed to a more comprehensive textbook such as the second edition of Hatle and Angelson's *Doppler Ultrasound in Cardiology.*

DONALD C. WALLERSON, MD
4040 Baychester Ave
Bronx, NY 10466

POCKET REFERENCE FOR MEDICAL INTENSIVE CARE

Edited by Barbara Anne Phillips, MD. 412 pp, illustrated. Rockville, Md, Aspen Publishers, Inc, 1986. $23.50 (softcover)

This volume is intended as a quick reference manual for physicians working in the adult medical intensive care units (ICUs). The editor and many of the authors are pulmonary intensivists who should appreciate the needs of the practitioner in the ICU. Softcovered, 412 pages, and paperback-book-sized, the manual can conveniently be carried to the bedside.

The book is organized in 32 chapters covering the spectrum of topics in critical care medicine. Some of the chapters cover essentially technical areas such as mechanical ventilation, right heart catheterization, and blood and blood products, while others are system-oriented and cover areas such as respiratory failure, myocardial infarction, and electrolyte disorders. Discrete topics of interest in critical care such as diabetic ketoacidosis, drowning, status epilepticus, coma, and gastrointestinal bleeding are covered in separate chapters devoted to these areas. There is also an excellent section on death, dying, and grief which addresses the physician's problems as well as the family's. There are very few illustrative figures but ample tables and a few decision algorithms. Material is presented with reasonable clarity. The general therapeutic approach is appropriately conservative and personal biases of the authors are clearly identified.

The manual has an uneven style and depth. While the sections on heat stroke, hypothermia, acid-base problems, carbon monoxide poisoning, and overdose are thorough and excellent, the coverage of other topics such as the "post-code patient," arrhythmias, and mechanical ventilation is too superficial. Several sections, including those on analysis of right heart catheterization data, arrhythmias, and the approach to pulmonary embolus, use decision trees to illustrate the salient features which need to be considered in a rapid assessment of the critically ill patient. This is exactly the kind of approach that one expects in such a manual. Unfortunately, other areas where clinical decision algorithms are already widely used and would have been helpful, such as acid-base, renal failure, and fluid and electrolyte problems, are dealt with in a less accessible manner. Tables are liberally used but even more would have been helpful. References are provided and are generally adequate but are insufficient in some sections and would be more useful if they had been annotated. Indexing is also inadequate. Fever is listed only for carbon monoxide intoxication and heat stroke and ethical issues only for acute respiratory failure. A good index is an essential element to insure the utility of this book in its aim as a ready pocket reference.

A volume of this size which does not presume to be encyclopedic in scope but which can anticipate the urgent questions confronting the physician in the intensive care unit would be an invaluable addition to the medical literature. Unfortunately, this book suffers from a failure to recognize the niche it should be trying to fill. The ICU physician will consult the *Physicians' Desk Reference* for information on drugs, an EKG manual for help with interpretation of EKG morphology and rhythms, a medical text for the differential diagnosis of acute renal failure. A manual such as this should be a "user-friendly" aid to urgent decision making in differential diagnosis, laboratory data interpretation, and therapy, but though many of its sections are excellent, the *Pocket Reference for Medical Intensive Care* does not succeed in filling this need.

HERMAN ELLMAN, MD
Metropolitan Hospital Center
New York, NY 10029

A PRIMER OF WATER, ELECTROLYTE AND ACID-BASE SYNDROMES

Seventh Edition. By Emanuel Goldberger, MD. 414 pp, illustrated. Philadelphia, Lea & Febiger, 1986. $19.50 (paperback)

The popularity of this book is obvious. This is the seventh edition, and it has also been translated into six languages. It is easy to read and understand without a sophisticated knowledge of renal physiology. However, after having read the book, I do not have a clear idea as to its main purpose.

The title, perhaps, best expresses the sentiment of the author: an electrolyte disorder rarely presents itself as a single manifestation but rather as part of a complex syndrome. For reasons that are not entirely clear to me, the author classifies all the electrolyte and acid-base disorders into three categories: syndromes of water disturbances, syndromes of sodium disturbances, and syndromes of acid-base disturbances. There are no separate chapters for disorders of potassium, calcium, phosphate, and magnesium. Instead, they are discussed under the heading of "syndromes due to disturbances in acid-base balance." More specifically, hyperkalemia is discussed under the heading of metabolic acidosis syndromes, and hypokalemia under the heading of metabolic alkalosis syndromes. If the categorization of hyperkalemia is attributable to the fact that acute acidosis causes hyperkalemia, the author neglects to mention that chronic metabolic acidosis tends to cause hypokalemia, not hyperkalemia. Nor does he indicate that acute organic acidosis, in contrast to inorganic acidosis, rarely causes hyperkalemia. If there is a sufficient connection between acid-base disorders and magnesium metabolism to warrant discussion of the latter under syndromes of acid-base disturbances, the author does not elucidate this connection.

The disorders of sodium and water metabolism are appropriately discussed under separate headings, but the discussions on these topics are grossly inadequate. Diabetes insipidus is discussed only in the context of hypernatremia. However, most patients with diabetes insipidus do not suffer from hypernatremia but from polydipsia and polyuria. The book fails to emphasize the important physiological fact that hypernatremia does not occur with increased urinary loss of water unless water intake is impaired because of, for example, defective thirst. There should have been separate discussions on polydipsia and polyuria. In the discussion of the treatment of hyponatremia, it is unfortunate that the author does not even mention a danger associated with rapid treatment, namely central pontine myelinolysis.

The book is apparently written primarily for those who are nonspecialists in electrolyte and acid-base disorders, and the emphasis is on diagnosis and management rather than on physiology and pathophysiology. Yet, the author spends a great deal of time on the cumbersome Astrup concept of acid-base disturbances that most investigators in this area have abandoned. He states in his preface that he has tried to correlate electrolyte disorders with the clinical pictures of patients. Yet, the book often does not emphasize what is clinically important and sometimes omits it entirely. For example, it is not emphasized that hyporeninemic hypoaldosteronism is by far the most common cause of chronic hyperkalemia. The author's coverage of hyperkalemia does not include a discussion of perhaps the most commonly used method, namely the use of a diuretic in combination with increased salt intake. In addition, there are many outright errors such as the statement that a high BUN lowers serum sodium (p 88) and an anion gap of less than 9 meq/L suggests that hyponatremia is a lab error.

There are many good books on the market on the topic of fluid and electrolyte disorders, and this is not one of those that can be highly recommended.

MAN S. OH, MD
State University of New York
Health Science Center
at Brooklyn
Brooklyn, NY 11203

THE AMERICAN CANCER SOCIETY CANCER BOOK. PREVENTION, DETECTION, DIAGNOSIS, TREATMENT, REHABILITATION, CURE

Edited by Arthur I. Holleb, MD. 650 pp, illustrated. Garden City, NY, Doubleday & Co, 1986. $22.50 (hardcover)

This is an ambitious work whose goal is to provide the general reading public with an authoritative guide to the present state of cancer care. Its subtitle, "Prevention, Detection, Diagnosis, Treatment, Rehabilitation, Cure," clearly indicates that its message is one of progress and hope. Under the editorship of Dr Arthur I. Holleb, the American Cancer Society's Senior Vice-President for Medical Affairs, an impressive group of cancer specialists from leading centers around the country have contributed to a thorough discussion of the topic.

The book admirably fulfills its aim of patient education in several key areas.

There are thorough descriptions of specific cancers and their treatment that are easy to read and that provide a great deal of technical information. The chapter on breast cancer in particular is an excellent summary of the current thinking on pathogenesis, prevention, diagnosis, and treatment. Perhaps the most important aspect of the work, however, is its attempt to demystify common attitudes about cancer that tend to stigmatize the cancer patient. The book also tries to dispel the undue pessimism among physicians as well as patients that often accompanies the diagnosis of cancer. With this in mind, there is a complete discussion of psychological factors, pain management, and rehabilitation. Appropriate emphasis is placed on prevention and the benefits of screening.

Great care has been taken to make the book understandable to the average reader without a background in the medical sciences. The information presented is neither unduly simplified nor overly technical. It is a current survey of the important facts that patients should know if they are to collaborate effectively with their physicians in an intelligent, informed manner with regard to cancer. As a source of clear, accurate information to patients, the book is invaluable for physicians.

If the book has a shortcoming, it is that its emphasis on the "good news" in cancer care does not deal squarely with the issues of death and dying, of terminal care, that are all too often an integral part of managing patients with cancer. A description of hospice care is conspicuously absent. Obviously, there is certainly no need to project an attitude of hopelessness in the face of a disease that is so prevalent and widely feared. A discussion of the reality of living with cancer, however, should also confront the reality of dying of cancer, and how this can take place with compassion, dignity, and meaning.

This book is a welcome and important addition to the lay medical literature. It cannot be matched for comprehensiveness, or for the authoritativeness of its authors. It does not trivialize, sensationalize, or obscure its subject matter. While not a medical text for physicians, there are chapters on aspects of cancer care and specific cancers that may serve as a useful overview for physicians on topics outside their own areas of expertise. This book is a valuable resource for physicians who wish to provide patients and their families with a better understanding of cancer, making them informed and effective partners in the medical care of this complex disease.

MICHAEL STEHNEY, MD
Lutheran Medical Center
Brooklyn, NY 11202

THE REJUVENATION STRATEGY. A MEDICALLY APPROVED FITNESS PROGRAM TO REVERSE THE EFFECTS OF AGING

By Rene Cailliet, MD, and Leonard Gross. 287 pp, illustrated. Garden City, NY, Doubleday & Co, Inc, 1987. $16.95 (hardcover)

The ambitious goal of this book is to present a medically approved fitness program to reverse the effects of aging. The authors maintain that the two major problems of aging are flexibility and posture and that "one simple postural adjustment—'standing tall'—can make you look years younger and restore vital function and mobility to your body. No arduous workouts are required." This simplistic approach reminds the reviewer of the book by the junior author, *Total Fitness in 30 Minutes a Week*, a great creative but misleading title published in 1976; it is impossible to achieve total fitness in 30 minutes a week.

This book's usefulness is based not on its "hype" approach to rejuvenation, but rather on its sound advice on some common musculoskeletal problems associated with aging. Loss of flexibility is one of the major disabling conditions of aging people, which is aggravated by the adaptive muscle and tendon shortening caused by sitting with knees and hips flexed, and by a sedentary lifestyle. Certainly, good posture and attention to flexibility contribute to good body functioning and should be a primary objective for all patients and their doctors.

For some reason, the section, "Rejuvenation Stretching Routine—Daily Warm-Up" (pages 180–249), repeats all the illustrations and instructions presented earlier.

Part Three, "Maintaining a Useful Life," briefly warns that reversing the effects of aging is not just a matter of rejuvenating prematurely aged tissue with a series of exercises, but must encompass four other important components of a youthful life: relaxation, nutrition, health, and mental attitude. Youthfulness is defined as an "absence of pain, discomfort and disability from either mental or physical causes."

Dr Cailliet, the senior author, is a well-credentialed authority in physical medicine and rehabilitation and the author of excellent medical books. Good drawings, a hallmark of his books, illustrate and explain well the problems of poor posture and flexibility, and describe many exercises to improve the aches and pains, poor posture, and loss of flexibility of aging people. Daily stretching is the best way to maintain maximum flexibility, and paying attention to proper posture will improve the body's appearance and function. The exercises and advice addressed to improving posture and flexibility are medically sound and well worth the time of the reader and the price of the book.

RAYMOND HARRIS, MD
Albany Medical College
Albany, NY 12208

Saving the US health care system

"The medical profession in this country is responsible, at least in part, for its plight regarding the recent promulgation of a plethora of highly questionable and suspect rules, regulations, and requirements that govern today's medical care costs," begins Roland T. Smoot, MD, in a guest editorial for the *Journal of the National Medical Association* (1987; 79:697–699). He accuses the medical profession of lacking fiscal responsibility, and contends that this attitude of indifference toward skyrocketing health care costs has resulted in the exclusion of medical professionals from efforts to reintroduce fiscal efficiency into the health care industry. Dr Smoot implores physicians to demonstrate their commitment to these efforts, and he proposes two main goals for the medical profession: "to eliminate inappropriate hospital admissions, and . . . to attain the proper length of stay for the hospitalized patient."

To enforce these measures, physicians must adopt a new, more open attitude towards peer review. Peer review is a necessity, states the author, and "is the exclusive, incontrovertible responsibility of the medical profession, and should be funded by the medical profession." He suggests that the medical staff of each hospital establish a fund, to be supported by its members, that could be drawn on to compensate for inappropriate admissions and unreasonably long hospital visits. The need for such a fund could be curtailed, he proposes, if admission reviews and case reviews occurred concurrently with treatment, rather than retrospectively. Any administrative and secretarial costs related to peer review activities should be absorbed by the hospital.

Following an inappropriate admission or unnecessary hospital stay, the medical staff involved should waive all professional fees related to the incident. In addition, the medical staff should consider imposing sanctions on their peers, ranging in severity from requiring approval of the prehospitalization certification by another staff member, to suspending the admitting privileges of the physician.

Dr Smoot envisions several important functions for organized medicine in this scenario. Medical associations could assist in defining the criteria for hospital admission and the guidelines used to regulate length of hospital stay. Institutions such as the American Medical Association and the National Medical Association could set the standards for collecting the data used to set reasonable guidelines around the country. Furthermore, organized medicine could help to arbitrate between hospital staff and third-party payors in controversial cases.

The author details the advantages of his proposals for the health care industry, hospitals, and the medical profession. His plan would virtually eliminate the need for the federal government's Professional Review Organization (PRO). Hospitals would benefit through improved relations between the administration and the medical staff, fewer instances of revenue losses due to inappropriate admissions and unnecessary, extended hospital stays, fewer hospital-based malpractice suits due to ongoing review of patient care by the medical staff, and simplified hospital admission procedures because preadmission reviews by insurance companies would no longer be needed.

"The demonstration of a commitment to the development of an efficiently run health care delivery system, a commitment not yet made clear, would enhance the credibility of our profession and give it a more effective and influential voice," states Dr Smoot. Benefits to patients would include a stabilization, or possibly even a reduction in health insurance premiums, as insurance companies save money due to lower hospital costs.

In the author's opinion, this plan can work and would be in the best interest of all the participants. However, he conjectures, "It is unlikely that this plan will be voluntarily tried by physicians, although they will offer no substantial reasons to the contrary. The usual posture of the medical profession has been one of resisting proposed changes in the practice of medicine."

Limits proposed for blood pressure reduction

Does continual lowering of blood pressure benefit all patients with hypertension? Not according to a study from the University of Goteberg, Sweden (*JAMA* 1987; 258:1768–1776), which examined the relationship between morbidity due to cardiovascular disease and control of blood pressure and serum cholesterol levels in 686 men with hypertension. The authors found that a reduction in both blood pressure and serum cholesterol levels was needed to effect a meaningful change in morbidity.

Without a concurrent drop in serum cholesterol levels, blood pressure levels had little effect on morbidity due to cardiovascular disease or coronary heart disease. The reduction in cholesterol levels reported during the first three years of treatment showed a significant correlation with morbidity due to cardiovascular disease during the following nine years of follow-up. Reducing blood pressure levels below about 150/85 mm Hg appeared to offer no additional advantage to the participants of this study.

Dysphagia may signal recurrent breast cancer

Dysphagia caused by an esophageal obstruction can be the first and only symptom of recurrent breast carcinoma. Goldberg et al recently reported the case of a 62-year-old woman with progressive dysphagia due to an esophageal stricture that represented invasion of the distal esophagus by metastatic breast carcinoma (*Cancer* 1987; 60:1085–1088).

The patient had undergone a modified left radical mastectomy four years earlier to remove an infiltrating ductal carcinoma. She received nine cycles of chemotherapy. Physical examination and laboratory studies performed after the onset of dysphagia showed no signs of recurrent carcinoma. A series of tests, including a chest film, barium swallow test, and flexible esophagoscopy led to the discovery of a concentric esophageal stricture. Biopsies showed squamous epithelium with focal dysplasia. A computed tomographic scan of the chest disclosed thickening

---

NEWS BRIEFS are compiled and written by Vicki Glaser.

of the distal esophagus. Laparotomy revealed adenocarcinoma of the distal esophagus associated with metastatic breast carcinoma. Radiotherapy produced marked improvement of the esophageal narrowing and relief of the dysphagia.

How breast carcinoma metastasizes to the esophagus is not well understood. The authors suggest the following hypotheses: diffuse mediastinal carcinomatosis with periesophageal involvement, dissemination along intercostal lymphatics and veins, and lymphatic diffusion from contiguous metastatic lymph nodes to deep lymphatic channels within the esophageal wall.

Metastasis of breast cancer to the gastrointestinal tract is not uncommon. This study shows that esophageal involvement, presenting as dysphagia, can be the sole manifestation of recurrent breast carcinoma.

Questioning the safety of home medical tests

Claims made by the Food and Drug Administration (FDA) that medical test kits designed for home use are as effective as similar tests performed in clinical settings came under fire in a recent article in the *New York Times* (October 4, 1987, p F2). The author reveals that the FDA has no written standards for the effectiveness of medical tests performed either at home or in a professional setting. Citing a congressional report, the author states that "proof that a medical test will never cause injury or will always be effective is not a requirement for agency approval."

Despite a 1976 law requiring the FDA to guarantee the effectiveness and safety of diagnostic devices prior to their being marketed, the FDA cannot regulate who uses a specific test device and how it is used once it has been sold. The author cites as examples the following potential problems: an incorrectly read x-ray; a false-positive AIDS blood test; and a heart catheter that causes damage to the heart while being used to diagnose a problem. With regard to the safety of diagnostic testing devices, the author recalls that four of 10,000 people who use a treadmill while undergoing an electrocardiogram suffer heart attacks and one person dies as a result of the test.

"It is wrong and even dangerous for the FDA to tell the public that home medical tests are as accurate as their counterparts done in clinical settings," the author concludes, "since neither has any official accuracy standards at this time. This does not mean medical tests are bad. Rather, they are diagnostic adjuncts and should not be considered innocuous."

Do long-distance runners suffer nerve damage in their feet?

This question was the focus of a study by Dyck et al (*Mayo Clinic Proc* 1987; 62:568–572) that evaluated the severity of the injuries most often associated with long distance running. The authors sought to determine whether any potential nerve damage would offset the health benefits attributable to this form of exercise.

Dyck et al based their findings on the following components: questionnaires completed by 25 active long-distance runners; a review of the participants' medical records for evidence of neurologic disease or conditions that would predispose them to neuropathy; interviews aimed at discovering motor, sensory, or autonomic symptoms associated with neuropathy, as well as previous foot injuries; and a neurologic examination. The neurologic examination included measurements of detection thresholds for vibratory and touch-pressure sensations and the determination of motor nerve conduction. Normal levels for motor, sensory, and autonomic criteria were based on data collected from 300 healthy subjects.

The authors found no statistically significant difference between the runners' reports of neuropathy symptoms and those of control subjects. The results of the neurologic examinations were also normal. As a group, the runners exhibited slight but significant differences in nerve conduction (worse) and detection threshold for vibratory sensation (elevated). The authors described these deficits as being "subclinical." They found no signs of overt neuropathy.

In conclusion, state the authors, "the trivial subclinical neuropathic deficits we noted are readily offset by the assumed health and recreational benefits of running."

Is physician dress perceived as an indicator of competence?

The doctor-patient relationship is a crucial component of quality health care. And a patient's confidence in the doctor's abilities is necessary for a strong, trusting relationship. In a recent issue of the *American Journal of Diseases of Children* (1987; 141:426–428), Paul Taylor, MB, reports on a survey aimed at determining whether the mode of dress of pediatric house staff influences parents' initial perception of the physicians' competence.

Taylor showed a random sample of visiting parents photographs of male and female house staff dressed in various outfits typically worn by such hospital personnel. In the formal outfit, the staff wore a long white lab coat over formal street clothes; the operating room (OR) garb consisted of a green OR suit and a short white intern's jacket. For the traditional intern's outfit, the men wore white pants, a casual shirt, and a short white jacket; the women wore casual slacks and shirt, and an intern's jacket.

The findings revealed a significant ($p < 0.001$) preference for formal dress, as compared to the other two styles. Formal dress got the nod of approval 59.4% of the time, with OR attire ranking second at 25.4%, and the intern outfit being preferred in 15.2% of comparisons.

Although the findings are based on comparisons of photographs, which are out of context and solely reflect visual cues—neglecting the effects of training, personality, and verbal skills—the author reports a clear relationship between pediatric house staff dress and parents' initial perceptions of physician competence. This, he concludes, "may reflect a widespread social perception of physicians in which the white coat remains a culturally significant symbol of healing."

1987 legislative accomplishments of the society

The 1987 legislative session in New York State was a particularly successful one for the Medical Society of the State of New York (MSSNY). The society's position prevailed on nine of 11 key issues.

Pension plan protection. The governor has signed into law a bill strongly supported by the MSSNY which protects approved pension plan assets from the reach of judgment creditors.

Free excess insurance coverage. A bill extending the excess layer of $1 million/$3 million liability insurance protection at no cost to physicians has been signed into law. It was supported by the MSSNY.

Immunity from damages for release of medical records. Two MSSNY-supported bills providing physicians with immunity for any damages occurring due to the required release of patients' mental health or general medical records have been signed into law.

No-fault coverage for victims of alcohol-related accidents. Governor Cuomo vetoed a bill strongly supported by the MSSNY and approved by the legislature which would have removed the inequity in the no-fault law barring physicians and hospitals from receiving payment for treatment rendered to victims of alcohol-related auto accidents. The governor did, however, direct the Insurance Department to work with health care providers and insurers to devise a solution to the problem, and said he hoped for legislative action on the issue in the 1988 session.

Eligibility for prenatal care services. A bill expanding the number of women eligible for prenatal care services, strongly supported by the MSSNY, has been signed into law.

Presumptive Medicaid eligibility. The governor has signed into law a bill making it state policy that patients applying for Medicaid and awaiting transfer to a home health care agency or a long-term care program will be presumed eligible for a 60-day period. The bill was strongly endorsed by the MSSNY.

DNR procedure. New law supported by the MSSNY establishes procedures for physicians issuing do-not-resuscitate orders which include consulting with patients or specific surrogates. The law grants physicians immunity from both civil and criminal liability as well as from charges of unprofessional conduct for issuing the DNR orders if they follow the prescribed procedures.

Physician drug dispensing. A bill opposed by the MSSNY which would have banned dispensing of prescribed drugs by physicians passed the Assembly but died on the Senate calendar.

Nurse practitioners. Each house of the legislature passed a different version of a bill to permit nurse practitioners to practice primary care medicine, but they failed to reach agreement, and thus no bill was enacted. The MSSNY opposed the bills because they failed to require what society officials considered the minimally necessary educational level of a master's degree. The Assembly bill would have given nurse practitioners the right to diagnose, treat, and prescribe with almost complete independence from physicians.

Extension of liability study. A bill appropriating funds for a continuation of the study of alternatives to the current tort system for adjudicating medical liability cases has been approved, and extends the study completion date to April 1, 1988. It was supported by the MSSNY.

Medicare/Medicaid crossover payments. A bill which would have reinstated Medicaid payment of the coinsurance and deductible amounts under Medicare passed the Senate but died in the Assembly, despite an extensive lobbying effort by the MSSNY, the New York State Psychiatric Association, and other provider and consumer groups. Legal challenges by the MSSNY to overturn the regulation eliminating such Medicaid payments continue in both the federal and state courts.

Increasing the role of women physicians in the society

The council recently approved a recommendation of the Committee on Membership that House of Delegates Resolution 87-49, Women Physicians, be amended. The amendment calls for the society to set up a leadership conference to identify women physicians within the society who would be willing to assume a more active role. The council also urged that the society through its Committee on Membership continue to focus on strategies to attract more women into membership.

Supervision and working hours of interns and residents

At its September 10, 1987, meeting, the society's council approved four staff recommendations that are the basis of the society's position regarding proposed revisions of Section 405 of the New York State Health Code. These proposed revisions to the code cover working hours, supervision, and credentialing of interns, residents, and medical students. The staff recommendations approved include support of the position of the American Medical Association (AMA) regarding the regulation of working hours and supervision of house staff. They also include support for the position of the Associated Medical Schools of New York, published in the November 1987 issue of the *Journal*, on the proposed regulations affecting medical students. The council went on record as recommending that the issue of resident supervision needed further study and clarification before specific recommendations and regulations are issued and as opposing the credentialing of residents as currently suggested in the proposed revisions to Section 405.4 of the New York State Health Code.

David Benford, MD, elected vice-president

At its meeting of September 10, 1987, the council of the Medical Society elected David Benford, MD, as vice-president to fill the unexpired term of the late Joseph Shanaphy, MD, who died in July. The council also chose Stuart Orsher, MD, to take Dr Benford's place as New York and Bronx Councilor.

MSSNY seeks further limitations on ATVs

The Medical Society of the State of New York (MSSNY) has proposed changes in state law that would prohibit the use of all-terrain vehicles (ATVs) in New York State by children under 14 years of age. The current law bans the use of ATVs by children under 10 years of age and requires registration of these recreational vehicles. MSSNY's move to raise the age limit reinforces an earlier recommendation by the American Academy of Pediatrics.

The Committee on Accident and Injury Prevention of the MSSNY proposed the legislative reform based on its review of a study of 412 accidents involving an ATV that were investigated by the US Consumer Product Safety Commission. In general, children younger than 14 years do not possess the "skill, judgment, and experience" required to operate these vehicles safely, according to committee chairman John States, MD.

Council votes to seek allocations of funds for the Committee for Physicians' Health

At its September 10, 1987, meeting, the council resolved to seek legislation authorizing a $20 increase in the triennial physician licensure fee, to $240, on condition that $30 of the total fee be allocated to support the Committee for Physicians' Health.

FROM THE LIBRARY

REPORTING MEDICAL SOCIETY NEWS

State medical journals are organs of the medical societies of the states and their constituent county societies. They are the natural repositories of the proceedings of those societies, and fulfill their functions best when their records are accessible and understandable. . . .

It is one of the duties of an editor of a state journal to read the journals of the other states in order to learn what the other societies are doing, and to bring new ideas to the attention of the officers, committeemen, and members of his own state society. . . .

An editor is something more than a clerk who takes reports, ties them together end to end like pieces of string, and winds them around the pages of his Journal. One of his great opportunities consists of writing his interpretations of meetings and conferences and recording them in a brief form which the average readers of his Journal can grasp readily and willingly. It may even happen that the average members of a Medical Society may open the pages of their Journal eagerly to see what new ideas have come out of the meetings and conferences.

The cry of every editor of State Medical Journals is "Give me more reports of conferences and meetings of Societies and Committees." He wants first of all to get the reports. Many secretaries and committeemen in New York State send clippings from newspapers. These are nearly always excellent provided the officers and speakers at the meetings have taken pains to tell the newspaper reporters what has happened. A reporter would be fired from a newspaper if he did not record in one concise paragraph the who, what, when and where of the meeting, and give a summary of the important subject that was discussed. A reporter may be brutally frank in what he says; but if he is, the reason is likely to be that the speakers missed constructive points as they made destructive criticisms regarding the subject under discussion.

When an editor writes up a meeting or conference, he follows exactly the same method of an expert physician with a patient.

First he takes a history and makes a physical examination in order to obtain *facts*.

Second he analyzes his facts and compares and weighs them in order to make a *diagnosis*, which in the case of a report of a conference consists largely in determining what the speaker is driving at and what the discussers think of his points.

Third, the editor develops a plan of *therapeutics*, for what the patient wants to know is what the doctor advises him to do. The editor will deduce the action which seems to be the consensus of opinion of the speakers.

The object of the report of a conference is to influcnce the actions of the members. If a short snappy report in a Journal arouses either interest or action in the members, it has fulfilled its object.

(*NY State J Med* 1929; 29:400–401)

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Joseph Peter Arcomano, MD, Huntington. Died September 8, 1987; age 63. He was a 1949 graduate of Pritzker School of Medicine of the University of Chicago, Chicago. Dr Arcomano was a Fellow of the American College of Radiology and a Diplomate of the American Board of Radiology. His memberships included the Radiological Society of New York State, the Radiological Society of North America, Inc, the American Roentgen Ray Society, the Society of Nuclear Medicine, the American Academy of Compensation Medicine, Inc, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Irvin Balensweig, MD, Palm Beach, Florida. Died June 18, 1987; age 93. Dr Balensweig was a 1918 graduate of Cornell University Medical College, New York. He was a Fellow of the American College of Surgeons, a Diplomate of the American Board of Orthopedic Surgery, and a member of the Academy of Medicine, the American Academy of Orthopaedic Surgeons, the New York County Medical Society, and the Medical Society of the State of New York.

Robert Glenn Bertsch, MD, Closter, New Jersey. Died September 6, 1987; age 56. Dr Bertsch was a 1957 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American College of Surgeons, a Diplomate of the American Board of Surgery, and a member of the New York County Medical Society and the Medical Society of the State of New York.

James Leslie Boyd, MD, Nashua, New Hampshire. Died August 7, 1987; age 78. He was a 1937 graduate of University of Western Ontario Faculty of Medicine, London, Ontario, Canada. Dr Boyd was a Diplomate of the American Board of Ophthalmology and a member of the American Academy of Ophthalmology and Otolaryngology, the Pan American Association of Ophthalmology, the Nassau County Medical Society, and the Medical Society of the State of New York.

John Butler Burns, MD, Binghamton. Died September 4, 1987; age 84. Dr Burns was a 1928 graduate of State University of New York at Buffalo School of Medicine. He was a Diplomate of the American Board of Pediatrics and a member of the American Academy of Pediatrics, the Broome County Medical Society, and the Medical Society of the State of New York.

Louis Marius D'Esopo, MD, Tarrytown. Died September 13, 1987; age 87. Dr D'Esopo was a 1928 graduate of Yale University School of Medicine, New Haven. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Vincent A. Doroszka, MD, Riverhead. Died August 8, 1987; age 85. Dr Doroszka was a 1930 graduate of Yale University School of Medicine, New Haven. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Henry Irwin Fineberg, MD, Dix Hills. Died October 4, 1987; age 83. Dr Fineberg was a 1927 graduate of Yale University School of Medicine, New Haven. His memberships included the American Association for Thoracic Surgery, the American Public Health Association, the Academy of Medicine, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Louise Fischer, MD, New York. Died June 28, 1987; age 76. She was a 1937 graduate of New York Medical College, New York. Dr Fischer was a Diplomate of the American Board of Otolaryngology. Her memberships included the American Academy of Otolaryngology, the American Academy of Allergy, the New York County Medical Society, and the Medical Society of the State of New York.

Rudolph E. Fremont, MD, New York. Died September 7, 1987; age 75. He was a 1936 graduate of Universitaet Wien, Medizinische Fakultaet, Wien, Austria. Dr Fremont was a Fellow of the American College of Angiology, the American College of Cardiology, the American College of Physicians, and the American College of Chest Physicians, and a Diplomate of the American Board of Internal Medicine. His memberships included the New York Cardiological Society, the Aerospace Medical Association, the Pan American Medical Association, the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Abel Albert Goodman, MD, Bronx. Died September 2, 1987; age 86. Dr Goodman was a 1926 graduate of New York Medical College, New York. He was a Diplomate of the American Board of Internal Medicine and the American Board of Allergy and Immunology. He was a member of the American Academy of Allergy, the Bronx County Medical Society, and the Medical Society of the State of New York.

Maurice Barnett Gordon, MD, New York. Died July 31, 1987; age 80. Dr Gordon was 1934 graduate of State University of New York at Buffalo. He was a Diplomate of the American Board of Psychiatry and Neurology and a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Kitty Fischer Hahn, MD, New York. Died June 24, 1987; age 73. Dr Hahn was a 1939 graduate of Orvosi Fakultas Tudomanyegyetem, Budapest, Hungary. She was a Diplomate of the American Board of Pediatrics and a member of the American Academy of Pediatrics, the New York County Medical Society, and the Medical Society of the State of New York.

Janet Jakub, MD, New York. Died May 9, 1987; age 78. Dr Jakub was a 1937 graduate of Facolta di Medicina e Chirugia dell'Universita di Bari, Bari, Italy. She was a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Thomas La Barbera, MD, Boynton Beach, Florida. Died August 15, 1987; age 78. He was a 1934 graduate of New York Medical College, New York. Dr La Barbera was a Fellow of the American College of Angiology. His memberships included the New York Cardiological Society, the New York State Society of Internal Medicine, the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of the New York.

Morris Leider, MD, New York. Died June 16, 1987; age 79. Dr Leider was a 1933 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Dermatology, Inc, and a member of the New York State Dermatological Society, the American Academy of Dermatology, the New York County Medical Society, and the Medical Society of the State of New York.

Harold Irving Lepow, MD, New Rochelle. Died August 5, 1987; age 69. He was a 1943 graduate of Faculte de Medicine de l'Universite de Lausanne, Lausanne, Switzerland. Dr Lepow was a Fellow of the American College of Pathologists and a Diplomate of the American Board of Pathology. His memberships included the New York State Society of Pathologists, the New York Pathological Society, the American Association of Pathologists and Bacteriologists, the Bronx County Medical Society, and the Medical Society of the State of New York.

Oscar Merker, MD, Bronx. Died April 19, 1987; age 72. Dr Merker was a 1943 graduate of Middlesex University School of Medicine, Waltham, Massachusetts. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Walter G. Nauenberg, MD, New York. Died June 16, 1987; age 90. Dr Nauenberg was a 1922 graduate of Medizinische Fakultaet der Universitaet Rostock, Rostock, Germany. He was a member of the American Society of Tropical Medicine and Hygiene, the New York County Medical Society, and the Medical Society of the State of New York.

Arthur Quito Penta, MD, Schenectady. Died August 9, 1987; age 90. He was a 1925 graduate of University of Vermont College of Medicine, Burlington. Dr Penta was a Fellow of the American College of Chest Physicians and a Diplomate of the American Board of Otolaryngology. His memberships included the American Academy of Otolaryngology, the American Broncho-Esophagological Association, the Medical Society of the County of Schenectady, and the Medical Society of the State of New York.

Ira Alvin Rashkoff, MD, White Plains. Died September 8, 1987; age 68. Dr Rashkoff was a 1943 graduate of Yale University School of Medicine, New Haven. He was a Diplomate of the American Board of Internal Medicine, and a member of the Academy of Medicine, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Frederick Rath, MD, Woodmere. Died August 29, 1987, age 78. Dr Rath was a 1938 graduate of Medizinische Fakultaet der Universtat Wien, Wien, Austria. He was a Diplomate of the American Board of Psychiatry and Neurology and a member of the American Psychiatric Association, the Nassau County Medical Society, and the Medical Society of the State of New York.

Josephine Rera, MD, Brooklyn. Died July 28, 1987; age 84. Dr Rera was a 1926 graduate of New York Medical College. She was a member of the American Academy of Family Practice, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Boswell Roberts, Jr, MD, Oswego. Died July 22, 1987; age 42. Dr Roberts was a 1973 graduate of the University of Rochester School of Medicine-Dentistry, Rochester. He was a member of the Medical Society of the County of Oswego and the Medical Society of the State of New York.

Martin Joseph Ryan, MD, Conklin. Died August 30, 1987; age 82. He was a 1931 graduate of Columbia University College of Physicians and Surgeons, New York. Dr Ryan was a Fellow of the American College of Surgeons and a member of the Broome County Medical Society and the Medical Society of the State of New York.

Alan Schrenzel, MD, Brookville. Died September 10, 1987; age 61. He was a 1949 graduate of Chicago Medical School, Chicago. Dr Schrenzel was a Fellow of the American College of Gastroenterology and the American Academy of Family Practice, and a Diplomate of the American Board of Family Practice. His memberships included the Academy of Medicine, the New York Academy of Gastroenterology, the Nassau County Medical Society, and the Medical Society of the State of New York.

Bertram H. Seiffer, MD, New York. Died September 8, 1987; age 75. Dr Seiffer was a 1936 graduate of the University of Louisville School of Medicine, Louisville. He was a member of the New York State Society of Occupational Medicine, the Aerospace Medical Association, the American Occupational Medical Association, the Pan American Medical Association, the New York County Medical Society, and the Medical Society of the State of New York.

Murray Sherwin, MD, Scarsdale. Died September 11, 1987; age 80. He was a 1934 graduate of New York Medical College, New York. Dr Sherwin was a member of the New York State Society of Internal Medicine, the American Society of Internal Medicine, the Bronx County Medical Society, and the Medical Society of the State of New York.

Walter F. Stafford, MD, Buffalo. Died August 12, 1987; age 67. He was a 1944 graduate of State University of New York at Buffalo School of Medicine, Buffalo. Dr Stafford was a Diplomate of the American Board of Psychiatry and Neurology and a member of the American Academy of Neurology, the Association for Research in Nervous and Mental Disease, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Roger D. Strauss, MD, New York. Died September 1, 1987; age 38. He was a 1975 graduate of State University of New York Health Science Center at Brooklyn. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

John James Tidona, MD, Brooklyn. Died September 1, 1987; age 76. Dr Tidona was a 1936 graduate of New York Medical College, New York. He was a

Fellow of the American Academy of Family Practice and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Paul M. Traub, MD, Newburgh. Died August 1, 1987; age 81. Dr Traub was a 1931 graduate of the State University of New York Health Science Center at Syracuse. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Otolaryngology. His memberships included the American Academy of Otolaryngology, the Medical Society of the County of Orange, and the Medical Society of the State of New York.

Peter Vogel, MD, New York. Died August 26, 1987; age 86. Dr Vogel was a 1927 graduate of Tufts University School of Medicine, Boston. He was a Diplomate of the American Board of Pathology and a member of the Academy of Medicine, the New York Pathological Society, the American Society of Clinical Pathologists, the American Association of Immunologists, the International Society of Hematology, the American Society of Hematology, the New York County Medical Society, and the Medical Society of the State of New York.

NEW YORK STATE
JOURNAL OF MEDICINE

INDEX
Volume 87
January–December
1987

Editor:
PASCAL JAMES IMPERATO, MD

Published by the
MEDICAL SOCIETY OF THE STATE OF NEW YORK
PO Box 5404
420 Lakeville Road
Lake Success, NY 11042

ISSUE	NUMBER	PAGES
January	1	1-84
February	2	85-138
March	3	139-194
April	4	195-250
May	5	251-318
June	6	319-376
July	7	377-424
August	8	425-486
September	9	487-530
October	10	531-586
November	11	587-636
December	12	637-698

CLASSIFICATION CODES

CC = Clinical Conference
CPC = Clinicopathologic Conference
CR = Case Report
ED = Editorial Commentary

EP = Essay in Pediatrics
ER = Essay Review
HIST = History of Medicine

LE = Letter to the Editor
LEN = Leads from *Epidemiology Notes*
NF = News Feature

SUBJECT INDEX

Abruptio placentae
- Incidence of placenta previa and abruptio placentae in New York State, 594

Abscess
- Amebic liver abscess without obvious risk factors (CR), 662
- Modified radical neck dissections necessitated by a retropharyngeal abscess (CR), 567
- Rare *Streptococcus pneumoniae* soft tissue abscess (LE), 574
- Use of computed tomographic scanning to diagnose sterile gluteal abscesses (CR), 233

Acoustic neuroma
- Acoustic neuromas: Yesterday, today, and tomorrow (ED), 640
- Childhood acoustic neuroma (CR), 665

Acquired immunodeficiency syndrome
- Acquired immunodeficiency syndrome—1987 (ED), 251
- AIDS care in New York City: The comprehensive care alternative, 298
- AIDS definition clarified (NF), 136
- AIDS in adolescents: A rationale for concern, 290
- AIDS in Africa: Evidence for heterosexual transmission of the human immunodeficiency virus, 286
- AIDS in New York City: Moving ahead on effective public health approaches (ED), 257
- AIDS Intervention Management System (LEN), 626
- AIDS risk following therapy with blood-based factor concentrates (NF), 372
- AIDS testing proposed for transfusion recipients (NF), 310
- AIDS: The eleventh year (ED), 255
- AIDS update (NF), 247
- American Medical Association endorses mandatory AIDS testing for two groups (NF), 479
- Azidothymidine and human immunodeficiency virus infection (ED), 7
- AZT approved for sale in Britain (NF), 309
- CDC to expand testing for AIDS (NF), 309
- CDC to study AIDS (NF), 528
- Coagulase-negative staphylococcal sepsis in drug-dependent newborns who may be HIV-positive (CR), 301
- Cyclooxygenase inhibition and improved oxygenation in patients with pulmonary complications of AIDS, 280
- Demography of HIV infections among civilian applicants for military service in four counties in New York City, 262
- Education and contact notification for AIDS prevention, 296
- Effect of group education on improving attitudes about AIDS risk reduction, 272
- Fatal hemoptysis in a patient with AIDS-related complex and pulmonary aspergilloma (CR), 306
- Fear of AIDS (ED), 260
- Fulminant lymphocyte-depleted Hodgkin disease in a homosexual man with HIV infection (CR), 570
- Health care costs of AIDS projected into next decade (NF), 311
- Herpes simplex pericarditis in AIDS (CR), 304
- HIV antibodies in commercial intravenous gammaglobulin products (NF), 63
- HIV screening proposed for international travelers (NF), 310
- Hospital workers and AIDS: Effect of in-service education on knowledge and perceived risks and stresses, 278
- Intravenous drug use and the heterosexual transmission of the human immunodeficiency virus: Current trends in New York City, 283
- Local policy responses to the AIDS epidemic: New York and San Francisco, 264
- Male to female transmission of AIDS (LE), 184
- New AIDS drug to be tested in humans (NF), 311
- News in brief (NF), 420
- Number of AIDS cases expected to rise into the next century (NF), 309
- Oral candidiasis and AIDS (CR), 303
- Peace Corps becomes fourth federal agency to begin screening for AIDS (NF), 309
- Potential AIDS drug derived from egg yolk (NF), 372
- Prevention of AIDS (ED), 259
- Supreme Court ruling could benefit victims of AIDS (NF), 309
- Tuberculosis and AIDS: Guidelines for management of mycobacterial infections in people with HIV infections (LEN), 671
- Unusual presentation of lymphoma in a homosexual man (CR), 302

Acyclovir
- Acyclovir in the treatment of primary varicella pneumonia in non-immunocompromised adults (LE), 237

Adenocarcinoma
- Microscopic bilateral adenocarcinoma arising in ovarian endometrial cysts (CR), 617
- Renal adenocarcinoma (ED), 638
- Renal adenocarcinoma in the pediatric age group (CR), 663

Adolescent health
- AIDS in adolescents: A rationale for concern, 290

Africa
- AIDS in Africa: Evidence for heterosexual transmission of the human immunodeficiency virus, 286

Aging
- Mr Keane's tree, 509

AIDS
- (see **Acquired immunodeficiency syndrome**)

Air pollution
- Health effects of radon exposure (NF), 581
- Indoor air pollution (ED), 196
- Medical students' exposure to formaldehyde in gross anatomy laboratories, 385
- Moses Maimonides and air pollution (LE), 574
- Role of building construction and ventilation in indoor air pollution: Review of a recurring problem, 203

Alzheimer disease
- Genetic defect in Down's syndrome linked to Alzheimer's disease (NF), 312
- News in brief (NF), 420

Amebiasis
- Amebic liver abscess without obvious risk factors (CR), 662

American Medical Association
- AMA addresses key issues at annual meeting (NF), 527
- AMA gains judicial support for peer review activities (NF), 311
- AMA reports continued decline in US medical school enrollments (NF), 634
- AMA's socioeconomic survey (NF), 581
- American Medical Association endorses mandatory AIDS testing for two groups (NF), 479

Amiodarone
- Amiodarone: Therapeutic but toxic (ED), 322
- Serum creatinine increase associated with amiodarone therapy (CR), 358
- Status of amiodarone (ED), 324

Anemia
- Recombinant human erythropoietin used to treat anemia (NF), 136

Aneurysm
- Cystic artery pseudoaneurysm (CR), 47

Angioendotheliomatosis
- Malignant angioendotheliomatosis manifesting as ascending spinal cord dysfunction (CR), 470

Angioplasty
- At issue: The role of angioplasty in acute myocardial infarction (NF), 634

Anticonvulsants
- Hypersensitivity reaction to anticonvulsants following head injury (CR), 571

Apolipoprotein B-100
- Role of apolipoprotein B-100 in cardiovascular disease (NF), 246

Appreciation
- Our referees, 130

Arrhythmia
- Amiodarone: Therapeutic but toxic (ED), 322
- Serum creatinine increase associated with amiodarone therapy (CR), 358
- Status of amiodarone (ED), 324

Asbestos exposure
- Extensive calcified pleural plaques (LE), 236

Aspiration
- Evaluation and management of chronic aspiration, 154

Athletic injury
- Child and adolescent in sports, 116
- Treatment of acute sports injuries, 113

Azidothymidine
- Azidothymidine and human immunodeficiency virus infection (ED), 7
- AZT approved for sale in Britain (NF), 309

Baldness
- Baldness remedy takes a step forward (NF), 312

Beta blockers
- Symposium on beta blockers (NF), 528

Bezoar
- Gastric antacid bezoar in a patient with chronic renal failure (LE), 235

Bicycle accidents
- Preventing bicycle-related head injuries (ED), 319

Bochdalek hernia
- Bochdalek hernia in adulthood (CR), 516

Books received, annotations of
- 61, 313, 481, 633

Books reviewed
- *Advances in Upper Extremity Surgery and Rehabilitation* (Boswick, Jr., ed.), 369
- *American Cancer Society Cancer Book, The. Prevention, Detection, Diagnosis, Treatment, Rehabilitation, Cure* (Holleb, ed.), 676
- *America's Health Care Revolution. Who Lives? Who Dies? Who Pays?* (Califano), 674
- *Angiographic Diagnosis of Supratentorial Cerebral Tumours* (*Compared with CT Scan*) (Pansini and Conti), 58
- *Annual Progress in Child Psychiatry and Child Development 1985* (Chess and Thomas, eds.), 60
- *Anthology of Plastic Surgery, An* (Hayes, Jr, ed.), 189
- *Antimicrobial Drugs, The* (Pratt and Fekety), 417
- *Appleton's Review for FLEX* (Berg), 58
- *Atlas of Stapling Techniques* (Rubio), 370
- *Basic Surgery* (McCredie and Burns), 368
- *Basic Toxicology. Fundamentals, Target Organs, and Risk Assessment* (Lu), 134
- *Beat the Odds* (Solomon), 579
- *Behavioral Counseling in Medicine. Strategies for Modifying At-Risk Behavior* (Russell), 629
- *Biostatistics in Clinical Medicine* (Ingelfinger, Mosteller, Thibodeau and Ware), 577
- *Black Adolescent Pregnancy. Prevention and Management* (Goodwin, ed.), 415
- *Blood Disorders. The Facts* (Callender), 191
- *Cardiac Arrhythmias: Self-Learning* (Chung), 578
- *Cardiology 1986* (Roberts, ed.), 190
- *Case Studies in Pediatric Emergency Medicine* (Haftel, ed.), 525
- *Cervical Adenocarcinoma: A Colposcopic Atlas* (Ueki), 244
- *Clinical Computed Tomography. Illustrated Procedural Guide* (Chiu, Lipcamon, and Yiu-Chiu), 57
- *Clinical Doppler Echocardiography* (Missri), 675
- *Colonic Neoplasms* (Elias, ed.), 242
- *Common Sports Injuries in Youngsters* (Birrer and Brecher), 418
- *Computer in Radiology, The* (Hunter, ed.), 57
- *Consent and Confidentiality in the Health Care of Children and Adolescents. A Legal Guide* (Morrissey, Hofmann and Thrope), 415
- *Continuing Medical Education. A Primer* (Rosof and Felch, eds.), 629
- *Cost Containment and DRGs. A Guide to Prospective Payment* (Spiegel and Kavaler), 243
- *Dachman Diet for Kids, The. A Complete Guide to Healthy Weight Loss* (Dachman), 526
- *Development of American Physiology, The. Scientific Medicine in the Nineteenth Century* (Fye), 577
- *Diabetic Doctor, A, Looks at Diabetes: His and Yours* (Lodewick), 580
- *Doctors in Science and Society. Essays of a Clinical Scientist* (Booth), 669
- *Exercise and Sports Sciences Reviews* (Pandolf, ed.), 132
- *Fifteen Minute Hour, The. Applied Psychotherapy for the Primary Care Physician* (Stuart and Lieberman, III, eds.), 135
- *Fighting the Third Death* (Linksz), 239
- *Goldfrank's Toxicologic Emergencies* (Goldfrank, Flomenbaum, and Lewin), 134
- *Handbook of 1985 Living Will Laws* (Society for the Right to Die), 133
- *Handbook of Pediatric Cardiology* (Rees, Solinger, and Elbl), 524
- *Home Health Care Solution, The* (Nasif), 188
- *Infectious Diseases in Child Day Care: Management and Prevention* (Osterholm, Klein, Aronson, and Pickering, eds.), 523
- *International Textbook of Cardiology, The* (Cheng, ed.), 415
- *Italian Broadsides Concerning Public Health Documents from Bologna and Brescia in the Mortimer and Anna Neinken Collection, New York Academy of Medicine* (Jarcho, ed.), 242
- *Last Taboo, The. Money as Symbol and Reality in Psychotherapy and Psychoanalysis* (Krueger), 630
- *Ligament Injuries and Their Treatment* (Jenkins, ed.), 132
- *Manhattan Country Doctor: A Memoir* (Slocum), 239
- *Medicare System of Prospective Payment, The. Implications for Medical Education and Practice* (Garg and Barzansky, eds.), 579
- *Modern Medicine and Jewish Ethics* (Rosner), 578
- *Night Calls. The Personal Journey of an Ob/Gyn* (Eisenberg, Eisenberg, and Eisenberg), 243
- *1986 Year Book of Family Practice* (Rakel, ed.), 416
- *Ob/Gyn Emergencies. The First 60 Minutes* (Farrell, ed.), 244
- *Once a Doctor, Always a Doctor. The Memoirs of a German-Jewish Immigrant Physician* (Hartmann), 239
- *Pathology of Neoplasia in Children and Adolescents* (Finegold), 523
- *Persisting Osler, The. Selected Transactions of the First Ten Years of the American Osler Society* (Barondess, McGovern, and Roland, eds.), 417
- *Personal Money Management for Physicians* (Farber), 674
- *Physician and the Hopelessly Ill Patient, The. Legal, Medical and Ethical Guidelines* (Society for the Right to Die), 133
- *Physician Assistants: Present and Future. Models of Utilization* (Zarbock and Harbert, eds.), 631
- *Pocket Reference for Medical Intensive Care* (Phillips, ed.), 675
- *Practical Management of Eye, Ear, Nose, Mouth and Throat Emergencies* (Stair, ed.), 370
- *Practical Obstetrical Ultrasound* (Seed and Cefalo), 58
- *Practical Strategies in Outpatient Medicine* (Reilly), 59
- *Practicing Physician's Approach to Headache, The* (Diamond and Dalessio, eds.), 631
- *Primary Anaesthesia* (King, ed.), 368
- *Primer of Water, Electrolyte and Acid-Base Syndromes, A* (Goldberger), 676
- *Principles of Ambulatory Medicine* (Barker, Burton, and Zieve, eds.), 190
- *Principles of Family Systems in Family Medicine* (Henao and Grose, eds.), 188
- *Psychobiological Aspects of Allergic Disorders* (Young, Rubin, and Daman, eds.), 189
- *Rabies, The Facts* (Kaplan, Turner, and Warrell), 417
- *Rejuvenation Strategy, The. A Medically Approved Fitness Program to Reverse the Effects of Aging* (Cailliet and Gross), 677
- *Sexual Aversion, Sexual Phobias, and Panic Disorder* (Kaplan), 630
- *Shoulder Surgery in the Athlete* (Jackson, ed.), 368
- *Special Care. Medical Decisions at the Beginning of Life* (Frohock), 525
- *Spine. Failed Back Surgery Syndrome: Evaluation and Treatment* (White, ed.), 369

  - *Surgeon! A Year in the Life of an Inner City Surgeon* (Caleel and Littell), 239
  - *Taking the World in for Repairs* (Selzer), 579
  - *Three Forms of Sudden Death* (Gonzalez-Crussi), 632
  - *Trauma in Children* (Marcus, ed.), 525
- **Brain tumor**
  - Electrocardiographic anterior wall pseudoinfarction pattern caused by a metastatic brain tumor (CR), 125
- ***Branhamella catarrhalis***
  - Fatal *Branhamella catarrhalis* pneumonia (CR), 359
  - FDA approves drug against lower respiratory infections caused by *B catarrhalis* (NF), 372
- **Breast**
  - Subcutaneous nonparenchymal hemangioma of the breast (LE), 521
- **Breast cancer**
  - Breast carcinoma in a man following local trauma (LE), 186
  - Dysphagia may signal recurrent breast cancer (NF), 678
  - Is breast self-examination an effective means of screening for breast cancer? (NF), 479
  - Oral contraceptives and breast cancer risk (NF), 246
  - Tissue injury and breast carcinoma (LE), 521
- **Breast mass**
  - Subcutaneous nonparenchymal hemangioma of the breast (LE), 521
  - Use of ultrasonography in fine-needle aspiration biopsy of breast masses (NF), 246
- **Breathing support systems**
  - Artificial breathing support systems: Preferred nomenclature for medicine and the courts (ED), 589
- **Bromocriptine**
  - Neuroleptic malignant syndrome after open heart surgery: Successful treatment with bromocriptine (CR), 362
- **Cancer**
  - Breast carcinoma in a man following local trauma (LE), 186
  - Cancer screening and prevention: Knowledge, attitudes, and practices of New York City physicians, 643
  - Dysphagia may signal recurrent breast cancer (NF), 678
  - First human cancer gene sequenced (NF), 312
  - Immune system's response to cancer (NF), 63
  - Is breast self-examination an effective means of screening for breast cancer? (NF), 479
  - Limb salvage with sarcomas (LE), 667
  - Microscopic bilateral adenocarcinoma arising in ovarian endometrial cysts (CR), 617
  - New drug approach against ovarian cancer (NF), 479
  - New hope for treating skin cancer (NF), 528
  - Renal adenocarcinoma (ED), 638
  - Renal adenocarcinoma in the pediatric age group (CR), 663
  - Stomach and colorectal cancers in Puerto Rican-born residents of New York City (LEN), 625
  - Visual loss and deafness associated with meningeal carcinoma (CR), 616
  - "Vocal" antibodies used to track tumor tissue (NF), 372
- **Candidiasis**
  - Oral candidiasis and AIDS (CR), 303
- **Cardiac metastasis**
  - Isolated cardiac metastatic recurrence of epithelioid sarcoma after two and a half disease-free years (CR), 231
- **Cardiac screening**
  - Cardiac evaluation of school sports participants, 120
- **Cardiopulmonary bypass**
  - Bilateral diaphragm paralysis associated with topical cardiac hypothermia (CR), 514
  - Fibronectin deficiency following cardiopulmonary bypass (ED), 487
  - Fibronectin levels during cardiopulmonary bypass, 493
- **Cardiopulmonary resuscitation**
  - Effectiveness of film and videotape in teaching cardiopulmonary resuscitation to the lay public, 382
- **Carpal tunnel syndrome**
  - Carpal tunnel syndrome: cause and treatment (NF), 582
- **Cerebrovascular accident**
  - Relation of stroke to cocaine abuse (LE), 128
- **Chemotherapy**
  - Protecting healthy cells from chemotherapeutic drugs (NF), 419
- **Chicken pox**
  - Acyclovir in the treatment of primary varicella pneumonia in non-immunocompromised adults (LE), 237
- **Children's health**
  - AIDS in adolescents: A rationale for concern, 290
  - Cardiac evaluation of school sports participants, 120
  - Chemical perspectives in the design of a pediatric meningitis vaccine (EP), 226
  - Child and adolescent in sports, 116
  - Childhood acoustic neuroma (CR), 665
  - Educational and legal efforts needed to combat smoking by minors (NF), 527
  - Homelessness in New York City: The youngest victims (ED), 3
  - Immunization guidelines—1987, 476
  - Importance of mandatory mumps vaccination (NF), 420
  - MSSNY seeks further limitations on ATVs (NF), 681
  - New prospects for preventing *Haemophilus influenzae* type b meningitis through immunization (ED), 195
  - Preventing bicycle-related head injuries (ED), 319
  - Renal adenocarcinoma (ED), 638
  - Renal adenocarcinoma in the pediatric age group (CR), 663
- **Choledochal cyst**
  - Choledochal cyst in an adult (CR), 49
- **Cholesterol**
  - Limits proposed for blood pressure reduction (NF), 678
  - New cholesterol-reducing drug announced (NF), 136
  - New drug joins the fight against cholesterol (NF), 634
  - Researchers identify animal model for hypercholesterolemia and atherosclerosis (NF), 192
- **Ciprofloxacin**
  - Oral ciprofloxacin in the treatment of 14 patients with bacterial pneumonia, 330
- **Clinical Conferences (series)**
  - Adult measles, 391
- **Clinicopathologic Conferences (series)**
  - Myelofibrosis, renal vein thrombosis, and pulmonary emboli, 599
- **Cocaine**
  - Relation of stroke to cocaine abuse (LE), 128
- **Colon**
  - Volvulus of the descending colon (CR), 406
- **Commentaries**
  (see Editorial Commentaries)
- **Computed tomography**
  - Computed tomography of Morgagni hernia (LE), 520
  - Use of computed tomographic scanning to diagnose sterile gluteal abscesses (CR), 233
- **Condoms**
  - Condom advertising gains acceptance (NF), 310
  - FDA compliance policy guide for condoms (NF), 479
- **Condyloma acuminata**
  - Carbon dioxide laser surgery of female genital, perineal, and anal condyloma acuminata (LE), 365
- **Corticosteroids**
  - Corticosteroid therapy in sarcoidosis: A five-year, controlled follow-up study, 496
  - Corticosteroid treatment of sarcoidosis—who needs it? (ED), 490
- **Cutaneous polyarteritis nodosa**
  - Cutaneous polyarteritis nodosa (ED), 381
  - Polyarteritis nodosa presenting with migratory soft tissue swelling (CR), 402
- **Cyclosporine A**
  - Adverse effects of cyclosporine A on the microcirculation of the cold preserved kidney (ED), 141
- **Cyst**
  - Choledochal cyst in an adult (CR), 49
  - Mesenteric cyst (CR), 177
  - Primary retroperitoneal hydatid cyst (LE), 52
- **Cystic artery pseudoaneurysm**
  - Cystic artery pseudoaneurysm (CR), 47
- **Dentist**
  - Death of a dentist, 356
  - Responses to "Death of a dentist" (LE), 574
- **Deprenyl**
  - Deprenyl versus placebo in Parkinson disease: A double-blind study, 646
- **Diabetes**
  - Low serum triiodothyronine in diabetic ketoacidosis, 431
- **Diaphragmatic hernia**
  - Bochdalek hernia in adulthood (CR), 516
  - Computed tomography of Morgagni hernia (LE), 520
  - Diaphragmatic hernia (LE), 520
  - Diaphragmatic hernia mimicking pyopneumothorax and colonic mass (CR), 122
  - Diaphragmatic rupture due to blunt trauma: The case for timely intervention (CR), 512
  - Radionuclide imaging in the diagnosis of diaphragmatic hernia associated with neonatal group B streptococcal sepsis (CR), 518

Ruptured diaphragm: A diagnostic dilemma (ED), 87

Diaphragm paralysis

Bilateral diaphragm paralysis associated with topical cardiac hypothermia (CR), 514

Diarrhea

Diarrhea caused by group G streptococcus (LE), 366

Disseminated intravascular coagulation

Acute disseminated intravascular coagulation as a complication of systemic lupus erythematosus (CR), 181

Down syndrome

Genetic defect in Down's syndrome linked to Alzheimer's disease (NF), 312

Drug abuse

Coagulase-negative staphylococcal sepsis in drug-dependent newborns who may be HIV-positive (CR), 301

Intravenous drug use and the heterosexual transmission of the human immunodeficiency virus: Current trends in New York City, 283

Poster contest: The hazards of drug abuse, 623

Relation of stroke to cocaine abuse (LE), 128

Drug prescribing

How safe and effective are generic drugs? (NF), 635

New prescription format (NF), 479

Physicians' attitudes toward and knowledge about generic drug substitution, 539

Drug trials

CPMC seeking participants for three multicenter trials (NF), 582

Early American medicine

Impact of smallpox on the native population of the 18th century South (HIST), 30

Patterns of drug usage in colonial America (HIST), 37

Visual and artifactual materials in the history of early American medicine (HIST), 14

Women healers and domestic remedies in 18th century America: The recipe book of Elizabeth Coates Paschall (HIST), 23

Editorial Commentaries

Acoustic neuromas: Yesterday, today, and tomorrow, 640

Acquired immunodeficiency syndrome—1987, 251

Adrenal medullary grafts transplanted into the brain as a treatment for Parkinson disease, 377

Adverse effects of cyclosporine A on the microcirculation of the cold preserved kidney, 141

AIDS in New York City: Moving ahead on effective public health approaches, 257

AIDS: The eleventh year, 255

Amiodarone: Therapeutic but toxic, 322

Artificial breathing support systems: Preferred nomenclature for medicine and the courts, 589

Azidothymidine and human immunodeficiency virus infection, 7

Corticosteroid treatment of sarcoidosis—who needs it?, 490

Cutaneous polyarteritis nodosa, 381

Diagnostic approaches to Pancoast's syndrome, 320

Epithelioid sarcoma, 197

Ethical dilemmas imposed by the Medicare hospice regulations, 379

Evaluating the impact of the 1986 New York State liability legislation, 85

"Exaggerated death" of peritoneal lavage in the management of trauma patients, 534

Fear of AIDS, 260

Fibronectin deficiency following cardiopulmonary bypass, 487

Group B streptococcal infection in 1987, 428

Homelessness in New York City: The youngest victims, 3

Humanism and medical ethics in a technological age, 427

Implications of the recent clinical trials in hypertension, 5

Indoor air pollution, 196

Mesenteric cysts, 140

Myth called Medicare, The, 531

New prospects for preventing *Haemophilus influenzae* type b meningitis through immunization, 195

New York's homeless, 1

Nonoperative management of blunt abdominal trauma: The cost/benefit margin, 536

Parkinson disease, 139

Preventing bicycle-related head injuries, 319

Prevention of AIDS, 259

Renal adenocarcinoma, 638

Role of the physician as the patient's advocate, 491

Ruptured diaphragm: A diagnostic dilemma, 87

Sleep deprivation in physician training, 637

Status of amiodarone, 324

Supervising interns and residents and limiting their working hours, 425

Tuberculosis of the knee, 88

View of the proposed New York State regulations governing the professional activities of residents, 587

View of the proposed New York State regulations pertaining to the activities of medical students, 533

Withdrawing fluids and nutrition: An alternate view, 591

Electrocardiographic abnormalities

Electrocardiographic anterior wall pseudoinfarction pattern caused by a metastatic brain tumor (CR), 125

Emergency medical services

Emergency medical services (LE), 184

Endometriosis

Isolated endometriosis in a Bartholin gland (LE), 575

Microscopic bilateral adenocarcinoma arising in ovarian endometrial cysts (CR), 617

Entamoeba histolytica

Amebic liver abscess without obvious risk factors (CR), 662

Epidemiology Notes

(see Leads from *Epidemiology Notes* (series))

Epithelioid sarcoma

Epithelioid sarcoma (ED), 197

Isolated cardiac metastatic recurrence of epithelioid sarcoma after two and a half disease-free years (CR), 231

Limb salvage with sarcomas (LE), 667

Epstein-Barr virus

Etiology of infectious mononucleosis: Solving the riddle (EP), 352

Errata, 632

Erythropoietin

Recombinant human erythropoietin used to treat anemia (NF), 136

Essay Reviews

Doctors in Science and Society. Essays of a Clinical Scientist (Booth), 669

Taking stock: Physicians' memoirs, 239

Essays in Pediatrics (series)

Chemical perspectives in the design of a pediatric meningitis vaccine, 226

Discovery of the first drug effective in treating acute lymphoid leukemia in children, 500

Etiology of infectious mononucleosis: Solving the riddle, 352

Ethics

(see Medical ethics)

Ethylene glycol

Ethylene glycol intoxication: A new stage in the clinical syndrome (CR), 179

Etretinate

FDA approval announced for psoriasis drug (NF), 63

Exercise

Do long-distance runners suffer nerve damage in their feet? (NF), 679

Exercise prescription, 106

Variations in energy expenditure during physical exercise (LE), 621

Fiberoptic bronchoscopy

Role of fiberoptic bronchoscopy and transbronchial biopsy in the diagnosis of Pancoast's tumor, 326

Fibronectin

Fibronectin deficiency following cardiopulmonary bypass (ED), 487

Fibronectin levels during cardiopulmonary bypass, 493

Fibrosarcoma

Malpractice by nonphysician healers (LE), 473

Fineberg, Henry Irwin

In Memoriam, 642

Food and Drug Administration

FDA compliance policy guide for condoms (NF), 479

FDA licenses Western blot kit for HIV (NF), 479

FDA revises drug regulations (NF), 528

Questioning the safety of home medical tests (NF), 679

Foreign body

Subglottic foreign body in an adult patient (CR), 46

Formaldehyde

Medical students' exposure to formaldehyde in gross anatomy laboratories, 385

Generic drugs

How safe and effective are generic drugs? (NF), 635

Physicians' attitudes toward and knowledge about generic drug substitution, 539

Haemophilus influenzae

Chemical perspectives in the design of a pediatric meningitis vaccine (EP), 226

New prospects for preventing *Haemophilus influenzae* type b meningitis through immunization (ED), 195

Hamartoma

Endotracheal hamartoma (CR), 408

Mesonephric hamartoma of the seminal vesicle: Rare cause of a retrovesical mass (CR), 48

Head injury

Hypersensitivity reaction to anticonvulsants following head injury (CR), 571

Preventing bicycle-related head injuries (ED), 319

Health care
- Saving the US health care system (NF), 678

Health care costs
- Have prospective reimbursement systems affected physicians' awareness of health care costs? (NF), 371
- Health care costs of AIDS projected into next decade (NF), 311
- Physician payment reform (NF), 371

Hearing loss
- News in brief (NF), 420

Heart disease
- Effects of copper deficiency suggest sex-related differences in vulnerability to heart disease (NF), 246
- New cholesterol-reducing drug announced (NF), 136
- Researchers identify animal model for hypercholesterolemia and atherosclerosis (NF), 192
- Role of apolipoprotein B-100 in cardiovascular disease (NF), 246

Heimlich maneuver
- Aortic valve rupture following the Heimlich maneuver (NF), 480

Hemangioma
- Subcutaneous nonparenchymal hemangioma of the breast (LE), 521

Hemodialysis
- Recombinant human erythropoietin used to treat anemia (NF), 136

Hemoptysis
- Fatal hemoptysis in a patient with AIDS-related complex and pulmonary aspergilloma (CR), 306

Hepatic disease
(see Liver)

Herpes simplex
- Herpes simplex pericarditis in AIDS (CR), 304

History of Medicine (series)
- Arthur Donaldson Smith, MD: Physician, explorer, naturalist, and diplomat—Part I, 90
- Arthur Donaldson Smith, MD: Physician, explorer, naturalist, and diplomat—Part II, 161
- Arthur Donaldson Smith, MD: Physician, explorer, naturalist, and diplomat—Part III, 210
- Arthur Donaldson Smith, MD: Physician, explorer, naturalist, and diplomat—Part IV, 334
- Arthur Donaldson Smith, MD: Physician, explorer, naturalist, and diplomat—Part V, 436
- Impact of smallpox on the native population of the 18th century South, 30
- Medical writings of Moses Maimonides, The, 656
- Patterns of drug usage in colonial America, 37
- Physician framers of the Constitution, 504
- Role of physicians in the life insurance industry in the 19th century, 546
- Visual and artifactual materials in the history of early American medicine, 14
- Women healers and domestic remedies in 18th century America: The recipe book of Elizabeth Coates Paschall, 23

HIV
(see Human immunodeficiency virus (HIV))

Hodgkin disease
- Fulminant lymphocyte-depleted Hodgkin disease in a homosexual man with HIV infection (CR), 570
- Unusual thoracic myelopathy in two patients with Hodgkin disease (CR), 467

Homelessness
- Homelessness in New York City: The youngest victims (ED), 3
- Homelessness in Paris (LE), 411
- New York's homeless (ED), 1
- Reproductive experience of women living in hotels for the homeless in New York City, 10

Home medical tests
- Questioning the safety of home medical tests (NF), 679

Hospice
- Ethical dilemmas imposed by the Medicare hospice regulations (ED), 379

Hospital workers
- Hospital workers and AIDS: Effect of in-service education on knowledge and perceived risks and stresses, 278

Human growth hormone
- Recombinant human growth hormone receives FDA approval (NF), 311

Human immunodeficiency virus (HIV)
- AIDS definition clarified (NF), 136
- AIDS in Africa: Evidence for heterosexual transmission of the human immunodeficiency virus, 286
- Azidothymidine and human immunodeficiency virus infection (ED), 7
- Coagulase-negative staphylococcal sepsis in drug-dependent newborns who may be HIV-positive (CR), 301
- Demography of HIV infections among civilian applicants for military service in four counties in New York City, 262
- FDA licenses Western blot kit for HIV (NF), 479
- Fulminant lymphocyte-depleted Hodgkin disease in a homosexual man with HIV infection (CR), 570
- HIV antibodies in commercial intravenous gammaglobulin products (NF), 63
- HIV screening proposed for international travelers (NF), 310
- Intravenous drug use and the heterosexual transmission of the human immunodeficiency virus: Current trends in New York City, 283
- Tuberculosis and AIDS: Guidelines for management of mycobacterial infections in people with HIV infections (LEN), 671

Hydatid cyst
- Primary retroperitoneal hydatid cyst (LE), 52

Hyperparathyroidism
- Urinary calcium excretion in nonfamilial hyperparathyroidism, 143

Hypertension
- Hypertension and complications of labor/delivery (LEN), 627
- Implications of the recent clinical trials in hypertension (ED), 5
- Limits proposed for blood pressure reduction (NF), 678

Immunization
- Chemical perspectives in the design of a pediatric meningitis vaccine (EP), 226
- Immunization guidelines—1987, 476
- Importance of mandatory mumps vaccination (NF), 420
- New prospects for preventing *Haemophilus influenzae* type b meningitis through immunization (ED), 195

Infectious mononucleosis
- Etiology of infectious mononucleosis: Solving the riddle (EP), 352

Injury control
- Injury control program (LEN), 628

Insulin
- Tetracycline reduces the need for insulin (LE), 576

Kidney
- Renal adenocarcinoma (ED), 638
- Renal adenocarcinoma in the pediatric age group (CR), 663

Kidney transplant
- Adverse effects of cyclosporine A on the microcirculation of the cold preserved kidney (ED), 141

Koplik spots
- Koplik spots and echo 9 virus (LE), 667

Lacunes
- Lacunes: The pervasive strokes, 650

Laryngeal foreign body
- Subglottic foreign body in an adult patient (CR), 46

Laser surgery
- Carbon dioxide laser surgery of female genital, perineal, and anal condyloma acuminata (LE), 365
- Laser device is first approved for treatment of vascular disorder (NF), 419

Leads from *Epidemiology Notes* (series)
- AIDS Intervention Management System, 626
- Available publications, 673
- Changing patterns in cause specific mortality, 672
- Hypertension and complications of labor/delivery, 627
- Injury control program, 628
- Stomach and colorectal cancers in Puerto Rican-born residents of New York City, 625
- Tuberculosis and AIDS: Guidelines for management of mycobacterial infections in people with HIV infections, 671
- Yersiniosis, 625

Letters to the Editor
- Acyclovir in the treatment of primary varicella pneumonia in non-immunocompromised adults, 237
- A "pack of trouble" for smoking drivers, 184
- Breast carcinoma in a man following local trauma, 186
- Carbon dioxide laser surgery of female genital, perineal, and anal condyloma acuminata, 365
- Central phlebothrombosis with two venous irritants, 236
- Cigarette advertising, 52
- Colostomy bag simulating bony metastases, 474
- Computed tomography of Morgagni hernia, 520
- Continuing use of x-ray pelvimetry for pregnant women, 411
- Diaphragmatic hernia, 520
- Diarrhea caused by group G streptococcus, 366
- Dr Bluestone's essay review, 473
- Emergency medical services, 184
- Extensive calcified pleural plaques, 236
- Gastric antacid bezoar in a patient with chronic renal failure, 235
- Homelessness in Paris, 411
- House staff supervision and working hours, 621

  Isolated endometriosis in a Bartholin gland, 575
  Jury is a crowd, 128
  Koplik spots and echo 9 virus, 667
  Limb salvage with sarcomas, 667
  Male to female transmission of AIDS, 184
  Malpractice by nonphysician healers, 473
  Massive pulmonary embolism with right atrial thrombus, 413
  Measles rash, 667
  Medicaid billing, 365
  Medicaid recordkeeping requirements and professional misconduct, 410
  Medical liability premiums in Great Britain, 52
  Minilaparotomy: An approach to postpartum tubal sterilization, 622
  Moses Maimonides and air pollution, 574
  Obturator hernias, 414
  Patient-physician relationship, 364
  Physicians and access to health care, 128
  Primary retroperitoneal hydatid cyst, 52
  Rare *Streptococcus pneumoniae* soft tissue abscess, 574
  Relation of stroke to cocaine abuse, 128
  Responses to "Death of a dentist", 574
  Review of *Once a Doctor, Always a Doctor*, 473
  Review of physicians' memoirs, 473
  Strategic planning for medical societies, 667
  Subcutaneous nonparenchymal hemangioma of the breast, 521
  Tetracycline reduces the need for insulin, 576
  Tissue injury and breast carcinoma, 521
  Variations in energy expenditure during physical exercise, 621

Leukemia
  Discovery of the first drug effective in treating acute lymphoid leukemia in children (EP), 500

Life insurance
  Role of physicians in the life insurance industry in the 19th century (HIST), 546

Lithium
  Lithium poisoning (CR), 230

Liver
  Amebic liver abscess without obvious risk factors (CR), 662
  Bile peritonitis following expectant management of liver fracture (CR), 565
  "Exaggerated death" of peritoneal lavage in the management of trauma patients (ED), 534

Lovastatin
  New cholesterol-reducing drug announced (NF), 136
  New drug joins the fight against cholesterol (NF), 634

Lyme disease
  Stillbirth following maternal Lyme disease (CR), 615

Lymphoma
  Unusual presentation of lymphoma in a homosexual man (CR), 302

Maimonides
  Medical writings of Moses Maimonides, The (HIST), 656
  Moses Maimonides and air pollution (LE), 574

Malpractice
  Increasing power of medical malpractice insurers (NF), 581
  Malpractice by nonphysician healers (LE), 473

Measles
  Adult measles (CC), 391
  Koplik spots and echo 9 virus (LE), 667
  Measles rash (LE), 667

Medicaid
  Medicaid billing (LE), 365
  Medicaid recordkeeping requirements and professional misconduct (LE), 410

Medical education
  AMA reports continued decline in US medical school enrollments (NF), 634
  Clinical clerkships approved for Caribbean medical school (NF), 312
  House staff supervision and working hours (LE), 621
  Medical students' exposure to formaldehyde in gross anatomy laboratories, 385
  Response to the recommendations of the New York State Commission on Graduate Medical Education concerning teaching in ambulatory care settings, 613
  Sleep deprivation in physician training (ED), 637
  Supervising interns and residents and limiting their working hours (ED), 425
  Supervision and working hours of interns and residents (NF), 680
  View of the proposed New York State regulations governing the professional activities of residents (ED), 587
  View of the proposed New York State regulations pertaining to the activities of medical students (ED), 533

Medical ethics
  Ethical considerations of reproductive technologies (committee report), 398
  Ethical dilemmas imposed by the Medicare hospice regulations (ED), 379
  Humanism and medical ethics in a technological age (ED), 427
  Withdrawing fluids and nutrition: An alternate view (ED), 591

Medical liability
  Evaluating the impact of the 1986 New York State liability legislation (ED), 85
  Medical liability premiums in Great Britain (LE), 52

Medical liability insurance
  Evaluating the impact of the 1986 New York State liability legislation (ED), 85
  Increasing power of medical malpractice insurers (NF), 581
  Medical liability premiums in Great Britain (LE), 52

Medical Liability Reform Act of 1986
  Evaluating the impact of the 1986 New York State liability legislation (ED), 85

Medical Society of the State of New York
  Council approves report on ethical considerations of reproductive technologies (NF), 373
  Council votes to seek allocations of funds for the Committee for Physicians' Health (NF), 681
  David Benford, MD, elected vice-president (NF), 680
  Ethical considerations of reproductive technologies (committee report), 398
  Facilitating enrollment of nonmember physicians (NF), 529
  Increasing the role of women physicians in the society (NF), 680
  In Memoriam: Henry Irwin Fineberg, MD, 642
  In Memoriam: Joseph F. Shanaphy, MD, 538
  Mandatory Medicare assignment (MSSNY white paper), 560
  MSSNY House of Delegates convenes for annual meeting (NF), 373
  MSSNY seeks further limitations on ATVs (NF), 681
  1987 legislative accomplishments of the society (NF), 680
  Readership survey of the *New York State Journal of Medicine*, 606
  Supervision and working hours of interns and residents (NF), 680
  Voluntary testing for HIV infection (NF), 529

Medical Society of the State of New York, House of Delegates
  Board of Trustees 1986–1987, 72
  Councilors 1986–1987, 72
  House Committee on Bylaws, 81
  1987 House of Delegates Members, 74
  Nominating Committee, 80
  Officers 1986–1987, 71
  Schedule of Sessions, 69

Medical specialties
  Information on medical specialties (NF), 311

Medicare
  Ethical dilemmas imposed by the Medicare hospice regulations (ED), 379
  Mandatory Medicare assignment (MSSNY white paper), 560
  Myth called Medicare, The (ED), 531

Memoriam
  Fineberg, Henry Irwin, MD, 642
  Shanaphy, Joseph F., MD, 538

Meningeal carcinoma
  Visual loss and deafness associated with meningeal carcinoma (CR), 616

Meningitis
  Chemical perspectives in the design of a pediatric meningitis vaccine (EP), 226
  New prospects for preventing *Haemophilus influenzae* type b meningitis through immunization (ED), 195

Mesenteric cyst
  Mesenteric cyst (CR), 177
  Mesenteric cysts (ED), 140

Mesonephric hamartoma
  Mesonephric hamartoma of the seminal vesicle: Rare cause of a retrovesical mass (CR), 48

Morgagni hernia
  Computed tomography of Morgagni hernia (LE), 520

Mortality
  Changing patterns in cause specific mortality (LEN), 672

Mumps
  Importance of mandatory mumps vaccination (NF), 420

Myelofibrosis
  Myelofibrosis, renal vein thrombosis, and pulmonary emboli (CPC), 599

Myocardial infarction
  Electrocardiographic anterior wall pseudoinfarction pattern caused by a metastatic brain tumor (CR), 125

Neuroleptic malignant syndrome
- Neuroleptic malignant syndrome after open heart surgery: Successful treatment with bromocriptine (CR), 362

New York City
- AIDS care in New York City: The comprehensive care alternative, 298
- AIDS in New York City: Moving ahead on effective public health approaches (ED), 257
- Cancer screening and prevention: Knowledge, attitudes, and practices of New York City physicians, 643
- Demography of HIV infections among civilian applicants for military service in four counties in New York City, 262
- Homelessness in New York City: The youngest victims (ED), 3
- Intravenous drug use and the heterosexual transmission of the human immunodeficiency virus: Current trends in New York City, 283
- Local policy responses to the AIDS epidemic: New York and San Francisco, 264
- Reproductive experience of women living in hotels for the homeless in New York City, 10

New York State
- Evaluating the impact of the 1986 New York State liability legislation (ED), 85
- Incidence of placenta previa and abruptio placentae in New York State, 594
- New York's homeless (ED), 1
- Response to the recommendations of the New York State Commission on Graduate Medical Education concerning teaching in ambulatory care settings, 613
- View of the proposed New York State regulations governing the professional activities of residents (ED), 587
- View of the proposed New York State regulations pertaining to the activities of medical students (ED), 533

Nurse shortage
- Nursing shortages in hospitals (NF), 529

Obituaries
- Abel, Louis, 314
- Ackernecht, Clarence F., 248
- Adler, Herbert, 248
- Ainsworth, Roy Chapman, 314
- Allen, Charles Leslie, 137
- Alley, John Lamont, 314
- Altieri, Anthony Joseph, 137
- Amoury, Emanuel Joseph, 374
- Ancona, Leonard Emanuel, 248
- Apton, Adolph A., 314
- Aquilino, Armand U., 314
- Arcomano, Joseph Peter, 682
- Argue, John Frederick, 65
- Ariola, Dominic S., 636
- Atkin, Samuel, 374
- Auerbach, Ludwig, 483
- Avnet, Samuel, 137
- Bahlke, Anne Magdalen, 65
- Baker, John Percy, 65
- Balensweig, Irvin, 682
- Balk, Kamila Czeslawa, 248
- Bannon, John Arthur, 636
- Barbanti, Benjamin John, 314
- Barbu, Valer, 65
- Barra, Alfred Joseph, 65
- Begner, Jacob Anthony, 374
- Benton, Leon, 583
- Berkowitz, Maxwell, 483
- Berkwits, Abner, 137
- Bertsch, Robert Glenn, 682
- Bien, Morris, 314
- Blau, Saul, 314
- Blinick, George, 583
- Bloom, Bernard, 583
- Boeck, Virgil Henry F., 636
- Bogatko, Frances L. Hacker, 583
- Bokhair, Labeeb Najib, 137
- Bonin, Jean Pierre, 583
- Bosshold, Erich P., 137
- Boyd, James Leslie, 682
- Braude, Eugene, 583
- Braun, Manfred, 483
- Bridge, Frederick David, 137
- Brikates, Peter, 314
- Brinitzer, Walter, 483
- Broad, Monroe M., 483
- Brockman, Louis, 421
- Bronson, Winifred, 374
- Brown, Clara Adele, 248
- Brucker, Mitchell A., 483
- Bullwinkel, George A., 421
- Burke, F. Vincent, 421
- Burns, John Butler, 682
- Bylow, Lubomyr, 583
- Cacioppo, Lawrence Thomas, 583
- Callerame, Salvatore J., 314
- Cameron, Alexander, 583
- Cannon, George Dows, 65
- Carlino, Lawrence L., 248
- Carroll, Gerard Ross, 583
- Carson, Arthur Henry, 65
- Cash, George Colling, 248
- Casper, Donald Jack, 421
- Castroviejo, Ramon, 248
- Chester, Morris, 248
- Clahr, Jacob, 483
- Cohan, Morris Harris, 421
- Cohen, Albert A., 248
- Coles, Jerome S., 314
- Collins, Robert Joseph, 421
- Corey, June G., 583
- Cornell, Albert, 421
- Costigan, Thomas Joseph, 65
- Cote, Marie Leonore, 583
- Dance, Clifton Louis, 65
- Deal, C. Pinckney, 65
- De Cicco, Louis Marius, 314
- De La Chapelle, Clarence, 583
- DeLuca, Frank Joseph, 421
- Demeri, Joseph P., 65
- D'Esopo, Louis Marius, 682
- De Stefano, Felice J., 583
- Dexter, Fred Ennis, 314
- Dickler, Emanuel, 374
- Dinnerstein, Morris, 248
- Dombeck, Sol, 248
- Donovan, Donald Edward, 248
- Doroszka, Vincent A., 682
- Dresner, Anczel, 374
- Drews, Ralph Clarence, 483
- Drislane, Thomas Murray, 248
- Eder, John, 421
- Egand, Maurice Abraham, 483
- Ehrlich, Herman, 248
- Elia, Dominic R., 374
- Elias, Miguel Grausman, 65
- Eller, Milton, 314
- Escher, George C., 314
- Fein, Harry David, 583
- Feit, Louis Joel, 584
- Feldman, Fred, 421
- Feldmann, Pauline, 137
- Feuerstein, Gerald, 421
- Fineberg, Henry Irwin, 682
- Fischer, Louise, 682
- Fisch, Mayer, 584
- Fisher, Joseph Osias, 584
- Fisher, Wilbur Jerome, 374
- Florin, Abraham, 137
- Fox, Louis, 248
- Frankel, Jesse Morris, 584
- Fremont, Rudolph E., 682
- Freydberg, Leo David, 483
- Friedenson, Meyer, 584
- Friedmann, Asa Ben, 483
- Fries, Margaret Evelyn, 374
- Fronczak-Bukowska, E. L., 421
- Galbraith, John M., 248
- Galvin, Eugene Frederick, 137
- Geiger, Ferdinand, 374
- Gensini, Goffredo G., 315
- George, Almer Tristan, 65
- Gerbasi, M. J., 374
- Gershman, Harry, 374
- Ginorio, Antonio R., 584
- Giuffre, Joseph, 315
- Glasser, Joseph G., 584
- Goldberger, Morris, 483
- Goldberg, Leo G., 249
- Goldberg, Leon, 374
- Goldfarb, Leopold, 421
- Goldman, Louis, 137
- Goldner, Martin Gerhard, 483
- Goldstein, Louis, 315
- Golob, Meyer E., 584
- Goodman, Abel Albert, 682
- Gordon, Maurice Barnett, 682
- Gordon, Nathan, 65
- Gottlieb, Israel, 66
- Gottlieb, Solon H., 483
- Gozan, Henry Abraham, 584
- Granet, Emil, 483
- Hagedorn, June Helene, 374
- Hageman, Herbert Corle, 315
- Hahn, Kitty Fischer, 682
- Haider, Zafar, 66
- Hamm, Frank Coleman, 374
- Hanania, Anwar J., 66
- Harris, Joseph William, 249
- Hatch, Merton Charles, 315
- Hauer, Julius, 584
- Hellreich, Emanuel, 315
- Herman, Morris, 315
- Hershey, Thomas Shetrond, 249
- Herz, Stefan, 421
- Hetrick, Emery Sylvester, 315
- Higgins, Frank Manley, 584
- Higinbotham, Norman L., 137
- Hinse, Eleonore, 249
- Hiss, John George Fred, 249
- Hoffman, Margot, 584
- Holden, Robert A., 249
- Holmberg, Frank Frederick, 315
- Horn, John E., 484
- Horowitz, Albert W., 584
- Hosmer, Harriet, 584
- Imberman, Renee Koehler, 66
- Immergut, Sidney, 315
- Israel, Martin, 315
- Jaffe, Matthew H., 584
- Jakub, Janet, 682
- Jenkins, Samuel F., 584
- Johnson, Samuel C., 484
- Kaminester, Sanford, 584
- Kampf, Samuel, 66
- Kantor, George J., 421
- Kantor, Mark G., 137
- Karver, Abraham, 375
- Kasnetz, Joseph P., 484
- Katz, Robert, 249
- Kaye, Martin L., 315

Keese, Thurston L., 375
Kelman, Aaron, 585
Kidder, James H., 375
Kissin, Milton, 421
Kleiger, Barnard, 484
Klein, Lionel, 585
Klinger, Louis H., 484
Koblentz, Aaron E., 375
Kodaman, Hilmi I., 422
Kopp, Abraham, 137
Koulack, Isidore, 315
Kovachev, Danica N., 422
Kroo, Adalbert, 585
La Barbera, Thomas, 683
Ladany, Emory, 137
LaForge, Harry George, 585
Lampka, Victor B., 249
Landsberger, Max, 249
Lara, Gustav A., 636
Lasky, Mortimer A., 66
Laster, Merny M., 66
Lauer, Dolor John, 422
Lawler, John A., 315
Lawyer, Tiffany, Jr., 66
Lebow, Harry, 138
Lecker, Sidney, 66
Leibowitz, Dudley, 484
Leider, Morris, 683
Lepow, Harold Irving, 683
Lerner, Charles, 484
Leslie, Charles Joseph, 375
Lester, Garra Levant, 422
Levin, Samuel G., 484
Levy, Samuel Benjamin, 315
Lewandowski, Tadeusz J., 585
Lieberman, Irving Edwin, 484
Liebmann, H. George, 66
Loesch, John, 66
Logue, Joseph Bruce, 66
Loiacono, Felix Peter, 66
Lorand, Sandor, 636
Lord, Alvah Leon, 249
Lourie, Herbert, 375
Lowry, John Edward, 249
Lusskin, Harold, 249
Lyon, Donald Rich, 422
Magovern, John Joseph, 249
Magovern, Malcolm James, 484
Mahoney, Jesse Waring, 315
Malko, Michael Gregory, 315
Mandel, Edward H., 66
Marek, Frederick M., 422
Marmar, William W., 585
Matthews, Benjamin L., 316
Mattikow, Bernard, 316
Medine, Myer Michael, 66
Meinhardt, William F., 585
Melomo, Francis P., 585
Merker, Oscar, 683
Merrill, Malcolm M., 316
Messina, Richard Philip, 249
Metzler, Gerald P., 138
Meylackson, Saul L., 585
Mezz, David, 422
Michaels, Joseph, 316
Miller, J. Lowry, 249
Mitchell, Alfred Adam, 585
Mitchell, Raymond W., Jr., 484
Monaco, Robert N., 375
Montgomery, James R., 585
Morton, Paul Colhoun, 422
Munzesheimer, Josef, 316
Murphy, George E., 585
Myers, Frank Daniel, 249
Nauenberg, Walter G., 683
Neidengard, Theodore H., 636
Nesbitt, Albert E., 316
Neuberg, Babette, 484
Newman, Saul Charles, 375
Ney, Imre, 585
Nigro, Bartholomew A., 316
Nittis, Savas T., 422
O'Brien, John Joseph, 375
O'Connor, Daniel James, 636
Oderkirk, Francis V., 249
Oliveri, Robert Andrew, 316
Oppenheimer, Hans, 636
Paganelli, Americus J., 66
Palmer, Milton Alwood, 422
Parnell, Vincent A., 585
Paul, George James, 422
Pelzer, Rudolph Henry, 375
Penta, Arthur Quito, 683
Perfetto, Angelo J., 250
Perlmutter, Isidore I., 484
Perman, Abraham Harold, 585
Petry, William Alfred, 585
Pincus, Jerome, 585
Popovits, Charles Joseph, 316
Primeau, Daniel F., 586
Princer, Robert Reuben, 316
Rabiner, Abraham Max, 250
Rabinowitz, Jay Robert, 375
Rapaport, Howard G., 422
Rashkoff, Ira Alvin, 683
Rath, Frederick, 683
Reid, Henry Nowell, 138
Reitz, Russell Elsworth, 586
Rera, Josephine, 683
Rescigno, Joseph, 375
Righter, George J., 316
Riley, Eugene B. F., 316
Riordan, Daniel Joseph, 484
Rizzo, Peter-Cyrus, III, 316
Roberts, Boswell, Jr, 683
Rogachefsky, Hymen, 422
Roodenburg, Andries I., 586
Rosenberg, Henry, 250
Rosenwasser, Harry, 316
Ross, Nathaniel, 250
Rubin, Arthur, 636
Rudorfer, Leon, 250
Russell, Edwin Philip, 422
Rutecki, Joseph Edmund, 316
Ryan, Martin Joseph, 683
Salisbury, Edward I., 484
Saltzman, Edward Aaron, 422
Samwick, A. Allen, 316
Sanchez, Filomeno, 250
Sanford, Clarence E., 375
Saxl, Stephan F., 586
Schiffman, Emanuel A., 250
Schmitt, Roswell L., 375
Schrenzel, Alan, 683
Schultze, Paul, Jr., 66
Schwartzwald, Joseph, 67
Schwarz, Helga Amalie, 422
Schwarz, Leonard, 250
Scott, William Doughty, 636
Scribner, Archie, 138
Seiffer, Bertram H., 683
Sengupta, Amarendra, 250
Shanaphy, Joseph F., 586
Shapiro, Harry, 484
Sharkey, William Francis, 250
Sherman, William T., 250
Sherwin, Murray, 683
Shomstein, Morton William, 250
Sichell, Jane Henny, 423
Siegelbaum, Harold L., 586
Simonetti, Amalia Julia, 636
Siris, Joseph H., 316
Skodnick, Augusta, 484
Smith, Charles Aloysius, 636
Smith, Jacob, 317
Smith, Wilbur Kenneth, 375
Smitonick, Michael, 375
Snow, Herman Bernard, 423
Sonnenschein, Harry, 67
Spaeth, Frank, 484
Speer, Francis Davis, 317
Speigelman, Anna R., 317
Sporn, Harry, 317
Stafford, Walter F., 683
Stalkus, Anthony J., 423
Star, Leon David, 317
Steinberg, Charles LeRoy, 636
Steinbrocker, Otto, 317
Stern, Victor, 586
Stirt, Sonia Sara, 317
Stix, Louis, 67
Stock, Robert, 376
Stone, Frederick Engels, 423
Stone, Maurice Joel, 67
Strauss, Roger D., 683
Strobos, Robert Julius, 376
Teichmann, Mark, 376
Thaler, S. Henry, 586
Thompson, T. Campbell, 317
Thorne, Irving J., 67
Tidona, John James, 683
Todoro, Joseph C., 586
Townsend, Alden J., 250
Traub, Paul M., 684
Traver, Ralph F., 484
Trillo, Raul Arturo, 67
Twinem, Francis P., 376
Tyson, Mary C., 138
Valt, Renato Paul, 250
Vieta, John O., 485
Vincent, Myrtle Wilcox, 636
Viola, Laurence E., 485
Viti, Gaetano Thomas A., 423
Vogel, Peter, 684
Voorhees, Charles H., 376
Wachen, Robert, 67
Walker, Roland J., 317
Wallace, Roy E., 250
Watters, Hyla S., 636
Wehl, Selma L., 485
Weinrauch, Paul A., 485
Weintraub, Frederick, 423
Weiss, Steven H., 317
Weshta, William H., 138
Wexler, Louis, 317
Wilcke, Burton W., 586
Williams, Kent D., 250
Willis, Willard H., 586
Wolcott, Charles C., 586
Wormer, Duncan L., 317
Yu, Woon Sang, 250
Zeiger-Prozan, Rebecca, 317
Zimring, Joseph George, 138

Obturator hernia
  Obturator hernias (LE), 414
Organized medicine
  Strategic planning for medical societies (LE), 667
Osteomyelitis
  Group B streptococcal osteomyelitis in an adult (CR), 466
Ovarian cancer
  New drug approach against ovarian cancer (NF), 479
Pancoast syndrome
  Diagnostic approaches to Pancoast's syndrome (ED), 320
  Role of fiberoptic bronchoscopy and transbronchial biopsy in the diagnosis of Pancoast's tumor, 326

Parkinson disease
  Adrenal medullary grafts transplanted into the brain as a treatment for Parkinson disease (ED), 377
  Autograft procedure seen as new mode of therapy for Parkinson disease (NF), 371
  Battling the effects of Parkinson disease (NF), 192
  Deprenyl versus placebo in Parkinson disease: A double-blind study, 646
  Parkinson disease (ED), 139
  Update on Parkinson disease, 147
Patient-physician relationship
  Death of a dentist, 356
  Is physician dress perceived as an indicator of competence? (NF), 679
  Patient-physician relationship (LE), 364
  Physician-patient relationship in interesting times, 553
  Responses to "Death of a dentist" (LE), 574
  Role of the physician as the patient's advocate (ED), 491
Pediatrics
  (see Children's health; Essays in Pediatrics (series))
Peer review
  AMA gains judicial support for peer review activities (NF), 311
Peptide T
  New AIDS drug to be tested in humans (NF), 311
Pericarditis
  Herpes simplex pericarditis in AIDS (CR), 304
Peritoneal lavage
  "Exaggerated death" of peritoneal lavage in the management of trauma patients (ED), 534
Pharmacology
  Patterns of drug usage in colonial America (HIST), 37
Phlebothrombosis
  Central phlebothrombosis with two venous irritants (LE), 236
Physicians' memoirs
  Dr Bluestone's essay review (LE), 473
  Review of *Once a Doctor, Always a Doctor* (LE), 473
  Review of physicians' memoirs (LE), 473
  Taking stock: Physicians' memoirs, 239
Placenta previa
  Incidence of placenta previa and abruptio placentae in New York State, 594
Pleural plaques
  Extensive calcified pleural plaques (LE), 236
Plumbism
  Diagnostic significance of free erythrocyte protoporphyrin and the FEP/hemoglobin ratio in plumbism, 542
Pneumonia
  Acyclovir in the treatment of primary varicella pneumonia in non-immunocompromised adults (LE), 237
  Fatal *Branhamella catarrhalis* pneumonia (CR), 359
  FDA approves drug against lower respiratory infections caused by *B catarrhalis* (NF), 372
  Nosocomial pneumonias in a community teaching hospital, 199
  Oral ciprofloxacin in the treatment of 14 patients with bacterial pneumonia, 330
  Tuberculosis mimicking acute pyogenic bacterial pneumonia (CR), 404
Polyarteritis nodosa
  Cutaneous polyarteritis nodosa (ED), 381
  Polyarteritis nodosa presenting with migratory soft tissue swelling (CR), 402
Pregnancy
  Bilateral pregnancy in a uterus didelphys (CR), 361
  Continuing use of x-ray pelvimetry for pregnant women (LE), 411
  Hypertension and complications of labor/delivery (LEN), 627
  Incidence of placenta previa and abruptio placentae in New York State, 594
  Stillbirth following maternal Lyme disease (CR), 615
Premature infants
  Gentler treatment reduces risk of chronic lung disease in premature infants (NF), 192
Preventive medicine
  Cancer screening and prevention: Knowledge, attitudes, and practices of New York City physicians, 643
Prospective reimbursement
  Have prospective reimbursement systems affected physicians' awareness of health care costs? (NF), 371
Pseudoaneurysm
  Cystic artery pseudoaneurysm (CR), 47
Psoriasis
  FDA approval announced for psoriasis drug (NF), 63
Public Health Research Institute
  Public Health Research Institute commemorates 45th anniversary (NF), 193
Pulmonary aspergilloma
  Fatal hemoptysis in a patient with AIDS-related complex and pulmonary aspergilloma (CR), 306
Pulmonary disease
  Cyclooxygenase inhibition and improved oxygenation in patients with pulmonary complications of AIDS, 280
  Evaluation and management of chronic aspiration, 154
  Gentler treatment reduces risk of chronic lung disease in premature infants (NF), 192
Pulmonary embolism
  Massive pulmonary embolism with right atrial thrombus (LE), 413
  Myelofibrosis, renal vein thrombosis, and pulmonary emboli (CPC), 599
Pyopneumothorax
  Diaphragmatic hernia mimicking pyopneumothorax and colonic mass (CR), 122
Radiographic abnormalities
  Colostomy bag simulating bony metastases (LE), 474
Radionuclide imaging
  Radionuclide imaging in the diagnosis of diaphragmatic hernia associated with neonatal group B streptococcal sepsis (CR), 518
Radon
  Health effects of radon exposure (NF), 581
Readership survey
  Readership survey of the *New York State Journal of Medicine*, 606
Renal disease
  (see Kidney)
Renal vein thrombosis
  Myelofibrosis, renal vein thrombosis, and pulmonary emboli (CPC), 599
Reproductive experience
  Reproductive experience of women living in hotels for the homeless in New York City, 10
Reproductive technology
  Council approves report on ethical considerations of reproductive technologies (NF), 373
  Ethical considerations of reproductive technologies (committee report), 398
Retropharyngeal abscess
  Modified radical neck dissections necessitated by a retropharyngeal abscess (CR), 567
Reye syndrome
  News in brief (NF), 420
San Francisco
  Local policy responses to the AIDS epidemic: New York and San Francisco, 264
Sarcoidosis
  Corticosteroid therapy in sarcoidosis: A five-year, controlled follow-up study, 496
  Corticosteroid treatment of sarcoidosis—who needs it? (ED), 490
Sarcoma
  Limb salvage with sarcomas (LE), 667
Seminal vesicle
  Mesonephric hamartoma of the seminal vesicle: Rare cause of a retrovesical mass (CR), 48
Shanaphy, Joseph F.
  In Memoriam, 538
Skin cancer
  New hope for treating skin cancer (NF), 528
Smallpox
  Impact of smallpox on the native population of the 18th century South (HIST), 30
Smith, Arthur Donaldson
  Arthur Donaldson Smith, MD: Physician, explorer, naturalist, and diplomat—Part I (HIST), 90
  Arthur Donaldson Smith, MD: Physician, explorer, naturalist, and diplomat—Part II (HIST), 161
  Arthur Donaldson Smith, MD: Physician, explorer, naturalist, and diplomat—Part III (HIST), 210
  Arthur Donaldson Smith, MD: Physician, explorer, naturalist, and diplomat—Part IV (HIST), 334
  Arthur Donaldson Smith, MD: Physician, explorer, naturalist, and diplomat—Part V (HIST), 436
Smoking
  A "pack of trouble" for smoking drivers (LE), 184
  Cigarette advertising (LE), 52
  Cigarette smoking among Ethiopian health professionals and students, 433
  Educational and legal efforts needed to combat smoking by minors (NF), 527
  Elimination of tobacco use is focus of New York Academy of Medicine report (NF), 192
  Politics of smoking (NF), 527
Soft tissue mass
  Use of computed tomographic scanning to diagnose sterile gluteal abscesses (CR), 233

Sports medicine
  Cardiac evaluation of school sports participants, 120
  Child and adolescent in sports, 116
  Do long-distance runners suffer nerve damage in their feet? (NF), 679
  Exercise prescription, 106
  Treatment of acute sports injuries, 113
Sterilization
  Minilaparotomy: An approach to postpartum tubal sterilization (LE), 622
Streptococcus
  Diarrhea caused by group G streptococcus (LE), 366
  Group B streptococcal infection in 1987 (ED), 428
  Group B streptococcal osteomyelitis in an adult (CR), 466
  Radionuclide imaging in the diagnosis of diaphragmatic hernia associated with neonatal group B streptococcal sepsis (CR), 518
  Rare *Streptococcus pneumoniae* soft tissue abscess (LE), 574
Suture substitute
  New glue could do job of stitches (NF), 419
Syphilis
  CDC reports increase in syphilis in US (NF), 528
Systemic lupus erythematosus
  Acute disseminated intravascular coagulation as a complication of systemic lupus erythematosus (CR), 181
Tax Reform Act of 1986
  How the new tax law will affect physicians (NF), 63
Tetracycline
  Tetracycline reduces the need for insulin (LE), 576
Tobacco
  Elimination of tobacco use is focus of New York Academy of Medicine report (NF), 192
Toxic epidermal necrolysis
  New treatment approach reduces morbidity and mortality associated with toxic epidermal necrolysis (NF), 480
Trauma
  Bile peritonitis following expectant management of liver fracture (CR), 565
  Breast carcinoma in a man following local trauma (LE), 186
  Diaphragmatic hernia mimicking pyopneumothorax and colonic mass (CR), 122
  Diaphragmatic rupture due to blunt trauma: The case for timely intervention (CR), 512
  "Exaggerated death" of peritoneal lavage in the management of trauma patients (ED), 534
  Hypersensitivity reaction to anticonvulsants following head injury (CR), 571
  Nonoperative management of blunt abdominal trauma: The cost/benefit margin (ED), 536
  Ruptured diaphragm: A diagnostic dilemma (ED), 87
Trinidad
  Nobel laureates attend medical update in Trinidad (NF), 193
Tuberculosis
  Current management of tuberculosis, 389
  Tuberculosis and AIDS: Guidelines for management of mycobacterial infections in people with HIV infections (LEN), 671
  Tuberculosis mimicking acute pyogenic bacterial pneumonia (CR), 404
  Tuberculosis of the knee (ED), 88
  Tuberculosis of the knee presenting as internal derangement (CR), 123
Ulcers
  Ulcers linked to bacterial infection (NF), 528
Ultrasonography
  Use of ultrasonography in fine-needle aspiration biopsy of breast masses (NF), 246
Uterus didelphys
  Bilateral pregnancy in a uterus didelphys (CR), 361
Vancomycin
  Vancomycin resistance documented (NF), 419
Volvulus
  Volvulus of the descending colon (CR), 406
Withdrawal of treatment
  Withdrawing fluids and nutrition: An alternate view (ED), 591
Women physicians
  Expanding role of women in medicine (NF), 527
  Increasing the role of women physicians in the society (NF), 680
  Women healers and domestic remedies in 18th century America: The recipe book of Elizabeth Coates Paschall (HIST), 23
Women's health
  Reproductive experience of women living in hotels for the homeless in New York City, 10
X-ray pelvimetry
  Continuing use of x-ray pelvimetry for pregnant women (LE), 411
Yersiniosis
  Yersiniosis (LEN), 625

AUTHOR INDEX

Abbate, Andre, 306
Abrams, Lawrence, 493
Agarwal, Nanakram, 49
Aledort, Louis M., 181, 613
Alexander, Fakhry Y., 361
Alexander, Jennifer E., 408
Ambre, John J., 134
Amstott, Richard, 135
Anaise, David, 141
Anandarao, Nanarao, 306
Annunziato, David, 667
Aranda, Conrado P., 326
Arno, Peter S., 264
Arnold, Angelo N., 141
Associated Medical Schools of New York, 533, 587

Ball, Gene V., 381
Ballambattu, Subrahmanya, 236
Balz, Thomas, 417
Bard, Gregory, 366
Barker, Stephen L., 565
Barzel, Uriel S., 143
Baum, John, 113
Becker, Norwin H., 663
Becker, William H., 306
Belmar, Roberto, 3
Benach, Jorge L., 615
Bennett, Stephen L., 60
Berkowitz, Dov J., 123
Bernier, William, 262
Berson, Burton L., 123
Bhatt, B., 404
Biliouris, Timothy, 184
Birrer, Richard B., 106, 132, 190, 621
Blackman, Norman S., 364
Blitzer, Andrew, 154
Bloomfield, Randall D., 128, 244, 415, 667
Bluestone, Naomi R., 239, 356
Bonnheim, David C., 47
Borrero, Edgar, 516
Brady, Elizabeth, 283
Brandstetter, Robert D., 184, 359, 589
Brecher, David, 580
Brodman, Richard, 408
Brooks, Chandler McC., 577
Brown, Audrey K., 542
Bruce-Chwatt, Leonard Jan, 242
Brundage, John F., 262
Buchalter, Ira H., 370
Burgdorfer, Willy, 615
Burke, Donald S., 262
Burstin, Harris E., 402
Bykofsky, Michael, 616

Cabrera, Manuel R., 514
Caglar, Haluk, 365
Caldarola, Vincent, 177
Campbell, Cheryl A., 674
Cardamone, Mary, 579
Caropreso, Philip, 140
Carsons, Steven, 493
Casarona, Joseph, 616
Cassell, Eric J., 398
Chaudary, Riffat R., 233
Chavkin, Wendy, 10
Checola, Robert, 518
Chen, Patrick, 470
Chiarello, Linda, 199
Chitkara, Rajinder, 237
Cho, Nan-Hwa, 470
Chu, Benjamin, 59
Cirillo, Leonard, 184
Cohen, Ann B., 504
Colbourn, Donald, 570
Coli, Lorraine, 359
Colvin, Stephen, 362
Conetta, Rick, 125
Cook, Albert W., 128
Cregler, Louis L., 128
Cuadrado, Mary, 283
Curran, John P., 366
Curry, Steven C., 134

Dalayeun, Jean, 411
Daller, John A., 106, 621
Davern, Paul F., 236
De Caprariis, Pascal J., 366
Decina, Paolo, 230
DeCosse, Jerome, 370
DeHovitz, Jack A., 7, 298, 523
Desiderio, Diane, 466
Des Jarlais, Don C., 283
Devanagondi, Bhaskar, 542
Dhar, Santi R., 404
Dickinson, P. C. T., 574
Dieterich, Douglas T., 304
DiFranza, Joseph R., 184
DiLorenzo, James C., 406
DiMaio, Mary F. A. C., 617
DiNardo, James A., 368
Disney, Frank A., 473
Dobkin, Jay, 574
Donovan, Virginia, 570
Doolittle, Thomas, 571
Drucker, Ernest, 3, 255
Dubner, Sanford, 302

Echenberg, Dean F., 296
Edsall, John R., 514
Edson, John N., 190, 415
Elinson, Jack, 643
Ellman, Herman, 675
El-Sadr, Wafaa, 283
Engel, Milton C., 123
Engler, Alan M., 189
Erk, Mehmet, 365
Esposito, John M., 622
Estes, J. Worth, 37
Exelby, Philip R., 231

Factor, Stewart A., 179
Feiner, Helen, 521
Fenton, A., 617
Fernandez, Kimberly Hall, 184
Fieve, Ronald R., 230
Fine, Edward J., 650
Fine, Richard, 516
Finn, William E., 379
Fishman, Sylvia, 382
Fleisher, Jay M., 385
Flynn, John T., 599, 667
Fogoros, Richard N., 324
Foo, Son Hoo, 646
Foster, Steven H., 280
Fox, Arthur C., 382
Foy, Donald F., 85
Freedberg, Robin S., 304
Friedland, Gerald, 260
Friedland, Michael L., 398
Friedman, Daniel, 539
Friedman, Joseph H., 139
Friedman, Samuel R., 283
Fromm, David, 565
Fulop, Milford, 431

Gaetjens, Eric, 470
Garbitelli, Vincent P., 576
Garcia, Emanuel E., 621, 637
Gardner, Lytt I., 262
Garibaldi, Richard A., 236
Garofalo, Michael, Jr., 616
Garrett, Robert C., 280
Garrick, James G., 418
Gartrell, Ellen G., 23
Geibel, Valerie, 542
Gellhorn, Alfred, 411
Gelwan, Jeffrey S., 303
Gemson, Donald H., 643
Gerdes, John, 629
Ghattas, George M., 413
Gilbert, Marvin S., 123
Gindea, Aaron J., 304
Gingell, Robert L., 524
Ginsberg-Fellner, Fredda, 526
Giorlando, Stephen W., 361
Gitler, Bernard, 125
Glass, Walter M., 665
Glebatis, Donna M., 594
Glidewell, Oliver, 577
Gold, Burton M., 235, 303
Goldberg, Robert J., 184
Goldenberg, Steven, 570
Goldfarb, Richard, 518
Goler, Robert I., 14
Gopinathan, Govindan, 646
Gordon, Gerald, 417
Gordon, Richard E., 237
Gordon, Stanley L., 369, 621
Gotta, Alexander W., 493
Graham, Peter E., 186
Green, Robert P., 567
Greene, Jeffrey B., 304
Guigli, Pamela E., 10
Gunzenhauser, Jeffrey, 262

Haller, J. Alexander, 525
Haller, John S., Jr., 546
Hanna, Bruce, 466
Harner, Stephen G., 640
Harris, Matthew, 521
Harris, Raymond, 677
Harris, Tommy, 575
Hartmann, Heinz, 473
Hassett, James M., Jr., 571
Hayes, Joseph G., 382
Heiberg, Elisabeth, 87
Hein, Karen, 290
Heller, Keith S., 302
Hemminki, Elina, 594
Hena, Muhammad Abu, 52, 667
Herbold, John R., 262
Hertz, Alan, 630
Hinkle, Lawrence E., Jr., 196
Hirsch, Charles S., 574
Ho, Monto, 272
Holzman, Robert, 466
How, Shu-Wen, 470
Hreshchyshyn, Myroslaw M., 365
Huang, Chin Tang, 496
Huggins, James, 272
Hughes, Robert G., 264
Huvos, Andrew, 231

Iaquinta, Frank E., 359
Imperato, Pascal James, 1, 90, 161, 210, 251, 334, 425, 436, 509, 606, 669
Ingegno, Alfred P., 574
Israel, Harold L., 490
Iwan, Laura, 411

Jacobs, Michael B., 358
Jaffe, Arnold, 539
Jakobovitz, Julian, 578
Jamal, Nasiruddin M., 413
Janerich, Dwight T., 594
Johnson, Nancy, 199
Joseph, Stephen C., 257

Kahn, Ellen I., 242, 617
Kandall, Stephen R., 301, 518
Karakousis, Constantine P., 197
Kaufman, Gerald, 411
Kaufmann, Thomas, 663
Kaul, Balakrishna, 542
Kavaler, Florence, 504
Keany, Ann E., 493
Kelley, Joseph F., 189
Kerr, Leslie Dubin, 181
Khan, Faroque A., 237, 330
Khurana, B., 662
Kinas, Helen, 48
King, Marjorie L., 120
King, Steven J., 520
Kingsley, Lawrence A., 272
Kirschenbaum, Abram E., 632
Klarsfeld, Jay H., 567
Koslow, Max, 377
Kramer, Charles, 364
Kriplani, Ajay K., 52
Kristal, Alan, 10
Krumpe, Peter E., 320
Ku, Yuan Kuang, 578
Kuhn, Matthew J., 48
Kumar, Kusum L., 474

LaDuca, John, 177
Lalka, David, 571
Landolt, Allison B., 398
Las, Murray, 143
Laude, Teresita A., 525
Lava, Neil S., 179
Lee, Ti-Kai, 470
Leilop, Linda, 496
Levensohn, Alan, 411
Levere, Richard, 613
Levin, William, 665
Leviton, Laura C., 272
Lieberman, Abraham N., 147, 362, 377, 646
Lin, Ching-Shen, 306
Lingenfelter, Bruce A., 674

Lister, Joan E., 243
Loeb, Laurence, 398
Lombardo, Gerard T., 306
Lucas, Linda M., 474
Lutwick, Larry I., 417
Lyons, Harold A., 496
Lyter, David W., 272

MacDonald, Alan B., 615
MacMillan, Roger W., 512
Maestrone, Paul, 377
Manning, Reginald, 368
Marchlinski, Francis E., 322
Mark, Herbert, 128
Martin, Carolyn, 616
Marx, John A., 536
Maskiell, Charles D., 520
Mastellone, Aniell T., 622
Matseoane, Stephen, 575
Mattucci, Kenneth F., 665
Maxfield, Roger A., 326
May, John J., 199
McCunney, Robert J., 203
McMeeking, Alexander A., 466
Mejia, Francisco Medina, 301
Menghini, Mario V., 560
Meredith, Gary S., 402
Metz, David, 504
Meyer, Gregg S., 621
Mifsud, Anthony, 631
Mikk, Helmut A., 667
Mildvan, Donna, 283
Militello, Philip R., 534
Miller, Steven N., 184
Mitnick, Hal J., 402
Mitnick, Julie S., 521
Moh, Paul, 235
Molloy, Michael, 571
Moore, Ernest E., 536
Morrissey, James, 133
Moscowitz, Edward, 575
Moser, Marvin, 5
Moy, Donald R., 133, 365, 410
Murray, Latham B., 512
Mushlin, Alvin I., 613
Mustalish, Anthony C., 184

Nenna, Andre D., 473
Neophytides, Andreas, 646
Nocks, Barry N., 638
Nordlicht, Stephen, 427
Nores, Jean-Marc, 411, 473
Numann, Patricia J., 398

O'Connor, Michael K., 57
O'Donnell, Carl R., 278
O'Donnell, Lydia, 278
Oh, Man S., 676
Olson, John E., 512
Ona, Fernando V., 398
Ongseng, Fukiat, 518
O'Shea, John S., 406
Osler, Turner, 512

Padmanabhan, Krishnan, 404
Papaioannou, A. N., 521
Paroski, Margaret W., 650
Pasternack, Peter, 362
Patel, Ashok, 662
Patel, Rameshbhai, 46
Payne, Franklin E., 416
Pellecchia, Crescens, 303
Pellegrino, Virginia, 298
Penney, Christopher L., 226
Perera, Charles A., 473, 574
Peters, Dawn, 236
Pochedly, Carl, 195, 352, 500
Poe, Robert H., 520
Powell, Steven, 236
Pratt, David S., 199
Prior, John T., 531

Quinn, Thomas C., 286

Raio, Frank, 570
Rajkumar, Solomon, 542
Ramanathan, Kumudha, 233
Raoof, Suhail, 330
Rapaport, Felix T., 141
Raviglione, Mario C., 186
Reddy, Sudhakar A., 662
Redfield, Robert R., 262
Remy, Jean-Michel, 473
Renshaw, Domeena C., 630
Requena, Ramiro, 46
Reygagne, Pascal, 473
Reynolds, Benedict M., 49
Reynolds, Vernon, 621
Rhee, John W., 47
Rieder, Ronald F., 191
Rinaldo, Charles R., 272
Risemberg, Herman M., 398
Robins, June, 231
Rose, Richard H., 414
Rosen, Gerald, 231
Rosenberg, Stephen, 243
Rosenblum, Lawrence J., 57
Rosner, Fred, 398, 574, 591, 656
Rubino, George, 622
Ruff, Robert L., 467
Ruymann, Frederick B., 523

Saba, Thomas M., 487
Sacks, Steven H., 567
Sadovsky, Richard, 188, 674
Sathyavagiswaran, Lakshmanan, 574
Schapira, Hans E., 123
Schell, Norman B., 120
Schell, Robert, 467
Schlegel, Anne M., 230
Schluger, Joseph, 382
Schneiderman, Henry, 236
Schofield, Janet W., 272
Schraft, William, 359
Seabron, Cheryl, 10
Self, Donnie J., 525
Setzen, Michael, 665
Shenker, I. Ronald, 415
Sher, Joanna, 470
Sherman, Lawrence M., 379
Shih, Henry J., 303
Sidel, Victor W., 259
Sillman, Frederick H., 244
Singh, Inderjit, 46
Smith, David L., 474
Soderstrom, Carl A., 534
Somasundaram, Mahendra, 470, 631
Sordillo, Peter P., 398
Soria, Emilio D., 650
Sperber, Robert G., 122
Spiegel, Allen D., 188, 579
Spiera, Harry, 181
Splain, Shepard H., 132
Stallones, Lorann, 199
Stankaitis, Joseph A., 553
Staszewski, Harry, 570
Stehney, Michael, 676
Steinhardt, Susan, 539
Steinheber, Francis U., 662
Steinherz, Laurel J., 231
Steinherz, Peter G., 231
Stevens, Dennis L., 428
Stillman, Richard M., 58, 368
Stolfi, Julius E., 184, 417, 579, 629
Stone, Alex, 516
Stoneburner, Rand, 283
Sutro, William H., 520

Tamarin, Frank M., 184, 589
Tenner, Michael S., 58
Therriault, Gene D., 594
Thomas, Henry M., III, 280
Tulin, Maurice, 574
Turino, Gerard M., 382
Turro, James, 431
Tyner, J. Richard, 52

Upson, James, 47

Valdiserri, Ronald O., 272
Vennema, Alje, 88, 389
Vermund, Sten H., 3
Visconti, Ernest B., 366
Voskovitch, James, 262

Wadler, Dennis, 414
Wallace, Eleanor Z., 613
Wallerson, Donald C., 675
Waltzer, Wayne C., 141
Warshaw, Leon J., 491
Weinberger, Sylvain M., 518
Weiss, Barry D., 319
Wickiewicz, Thomas L., 116
Wilkinson, Harold Arthur, 369
Winters, Thomas H., 184
Wish, Eric, 283
Wollschlager, Christine M., 330
Wong, Sunny, 359
Wood, Peter H., 30

Yancovitz, Stanley R., 283

Zaki, Mahfouz H., 496
Zein, Zein Ahmed, 433
Zide, Barry M., 574
Zimmerman, Sol S., 402
Zubowski, Robert, 406
Zusmer, Noel Robert, 58

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1983; 83:1089-1094). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith^2 reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al*. Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83: 925-927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337-338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, P.O. Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

Arthur Donaldson Smith
and
the Exploration of Lake Rudolf

By
Pascal James Imperato, MD

104 pages ***illustrated*** ***$5.00***

Arthur Donaldson Smith, MD was an American physician who also has an unusual career as an explorer, naturalist and diplomat.

The five part series on Dr. Smith, published during 1987 in the *New York State Journal of Medicine*, has now been reprinted as a softcover book on acid free paper by the Medical Society of the State of New York.

ORDER FORM Clip and mail

Arthur Donaldson Smith and the Exploration of Lake Rudolf.
Please send me ______ copies at $5.00 per copy.

Circulation Department
New York State Journal of Medicine
420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042

Name________________________________

Address______________________________

____

Payment required with order.

AIM HIGH

A SPECIAL PRACTICE FOR SPECIALISTS.

If you're a **Surgeon or OB/GYN or Other Medical Specialist,** the Air Force may have a special practice for you. What makes it special? You'll enjoy an excellent pay and benefits package. There'll be more time to spend with your family. You'll receive 30 days of vacation with pay each year. And you will work with modern equipment and some of the most highly trained professionals in the world, serving your country and your patients. Now that's special! Find out just how special your practice can be. Call

1-800-423-USAF

AIR FORCE

REAL ESTATE FOR SALE OR RENT

CUSTOM BUILD superbly designed professional home located in the East Northport area on highly visible 1/2 acre, just minutes from local hospitals, parkways and schools. Reception room plus 900 sq. ft. useable space on lower level. Upper level features wonderful contemporary living area with highhat lighting, skylights and cathedral ceilings. Asking $425,000. The Daniel Gale Agency, Camille Sonesen (516) 754-3400 or Joan Gannon (516) 692-6770.

MANHATTAN'S GREENWICH VILLAGE AREA, prime medical space for rent/sale. Private street entrance with 2500 sq. ft. of ground floor space and 1300 sq. ft. of lower level space. Ideal for labs, x-ray facility and/or suite doctor's office. Immediate occupancy. Will build to suit. No brokers. No fee. Call owner (516) 487-1714.

161 MADISON AVENUE (Madison Medical Building) between 32nd and 33rd streets, approximately 1,400 sq. ft. on the 5th floor. Ideal for Ophthalmologist or Optometrist (complete eye diagnostic unit on this floor). State-of-the Art telecommunications, 24 hour concierge. Convenient access to mass transit. Negotiable term or lease. Call Jane Rosenthal at Cross & Brown Company, (212) 642-9406.

REAL ESTATE FOR SALE OR RENT—CONT'D

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

ATTENTION PROFESSIONALS! Ideal home/office, best location Nassau County, 4 bedrooms, formal living and dining rooms, library, Florida room. Plus 650 square feet of office space, plenty of parking, close to main highways, flexible spring occupancy. Owner may finance for qualified practitioner! Contact Denise Glozek, Merrill Lynch Realty, (516) 681-2600.

PARK AVENUE AND 72nd STREET doctor's office for sale or lease. 1,500 square feet, newly renovated, doctor's office for any specialty with full operating room, 2 recovery rooms, consultation rooms, 3 examining rooms, nurse's station, etc. 660 square feet also available at same location. Call (212) 879-0409.

REAL ESTATE FOR SALE OR RENT—CONT'D

EAST SIXTIES, PROFESSIONAL BUILDING. The preferred location for internists, cardiologist, rheumatologist. 7 treatment rooms, 2 reception areas to share with three other internists. Share NYC licensed lab, radiology, thermography. Full or part time. (212) 838-2860.

PROFESSIONAL CONDOMINIUM conversion. Maximize the profit potential of your professional building and create flexibility for yourself (and your partners). For information and free article "Doctors Go Condo", contact: Paul Gellert, President, Gelco Realty Corp., 155 West 68th Street, New York, NY 10023. Phone (212) 272-7900.

MISCELLANEOUS

FIRM OF GERALD GOLDBERG, CPA-ATTORNEY, formerly with IRS, announces his availability to prepare taxes and plan under changing tax environment, accounting, tax appeals, legal representation or practice transfers. (212) 967-1404 and (516) 565-6260.

(*continued from p 4A*)

COLORADO

Jan 23–29. **The Fourth Annual Decisions in Surgery.** Snowmass. Contact: University of California, Extended Programs in Medical Education, Room U-569, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

Jan 30–Feb 6. **14th Annual Vail Conference in Anesthesiology.** Marriott's Mark Resort, Vail. Contact: Professional Seminars/University of Miami, PO Box 012318, Miami, FL 33101. Tel: (305) 547-6411.

FLORIDA

Jan 15–17. **Mammography and the Search for Breast Cancer.** 24 Cat 1 Credits. Hyatt Orlando, Orlando. Contact: Theresa Wade, 1351 Mt Hope Ave, Rochester, NY 14620. Tel: (716) 442-8432.

AROUND THE WORLD

MEXICO

Jan 12–17. **17th Annual Pediatric Postgraduate Course—Pediatric Update 1988.** 18 Cat 1 Credits. Camino Real, Puerto Vallarta. Contact: Ann J. Boehme, Associate Director for Continuing Education, Schneider Children's Hospital of Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

Jan 16–23. **Advances in Anesthesiology.** 22 Cat 1 Credits. Cancun. Contact: Ms Francine Kurth, Department of Anesthesiology, Mount Sinai Medical Center, One Gustave L. Levy Place, New York, NY 10029. Tel: (212) 650-5954. ☐

SARANTOS & COMPANY

S. Randy Sarantos, D.D.S., President

THE DOCTORS' MONEY DOCTOR

1. ***Investment Management Services***
   Pension, Personal and Childrens' Educational Asset Management Programs
2. ***Total Financial Planning Services***
   Wide selection of 10 different financial plans for every budget level
3. ***Insurance Advisory Services***
   Custom-designed Insurance and Risk Management programs
4. ***Practice Management Services***
   Buy/Sell, Associate Advisory Services, Practice Evaluations, Cash-Flow Analysis
5. ***Seminar Education Services***
   Special Financial Seminar programs for professional societies, groups & institutions

Exclusive Marketing Agents For:

Pro-Tech Practice Evaluations	***National Association of Doctors (NAD)***
Low-cost Expanded Option Analysis, including 180 values for your practice.	Exclusive agents in Eastern PA, NJ and CT, providing low-cost, high quality financial services.

YES: I would like to know more about Professional Financial Services. Please send me a free brochure.

Name ____________________

Address ____________________

City ____________ State ______ Zip ______

Sarantos & Company, Inc. 240 Cedar Knolls, Rd., Suite 310
Cedar Knolls, NJ 07927 (201) 539-4000

— *OR CALL 1-800-223-0164* —

The principals of Sarantos & Company, Inc., a Registered Investment Advisor, are also representatives of Integrated Resources Equity Corp Member NASD & SIPC, and a registered broker dealer

ROLLS ROYCES & BENTLEYS
UNUSUALLY LOW PRICES!

1958 Rolls Royce Silver Cloud I	$27,900
1965 Rolls Royce-Bentley S3	$23,900
1967 Rolls Royce-Bentley T	$21,900
1965 Rolls Royce Silver Cloud III	$37,900
1971 Rolls Royce Corniche Coupe	$31,900

IMPORTANT! WE SERVICE WHAT WE SELL
Call Rudy Turner N.J. (609) 655-8350

MISCELLANEOUS—CONT'D

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuarial certification and employee statements of participation—partnership agreements and professionals incorporation—No insurance required—references upon request—Wachstock and Dienstag Attorneys at Law, 122 Cutter Mill Road, Great Neck, NY 11021 (516) 773-3322.

EB TRANSCRIPTION SPECIALISTS offering expert work with professional service to fit your needs. All medical specialties. Letters and correspondence, patients charts and manuscript typing. Fast turn around time, free pick up and delivery in the New York area and competitive rates. For additional information call Emily at (718) 894-1987.

SIGNATURE LOANS—$5,000 to $60,000 doctors only. No collateral, simple interest, 4, 5, or 6 year terms. No points, no fees, no prepay penalties. No use restrictions. Residents, D.O.'s, new practitioners welcome. For application and info call: (NY) Northstar Funding (212) 323-8076 or Mediversal 1-800-331-4952 Dept. #135.

Have Your Journals Bound

- Title, Vol. & Year
- Matched Samples
- Choice of Colors
- $17.50 per Volume
- Pick-Up and Delivery Services for Nominal Fee

L.E.C. BOOKBINDERS
292 Broadway, Lynbrook, NY 11563
(516) 593-1199

"SELLING PRACTICES IS OUR BUSINESS"
Want to maximize the return on your investment? Before you buy or sell, call for an appraisal to assure the best financial and transfer terms. We are a full service practice broker, not simply a listing service. Since 1981, Countrywide has been instrumental in helping hundreds of doctors like yourself buy and sell their practices. We guide you through the entire sales process from initial meeting to closing. Our offices serve New York, New Jersey and New England.
Countrywide Business Brokerage, Inc.
319 East 24th Street, Suite 23-G, New York, NY 10010
(212) 686-7902 (203) 868-3666 (617) 232-1075

SIGNATURE LOANS—$5,000 to $60,000
DOCTORS ONLY. No collateral, simple interest, 6 years to repay. No points, no fees, no prepay penalties. Use for any reason. Residents, D.O.'s, start-up welcome. For appointment and info call:
(NY) NORTHSTAR FUNDING (212) 323-8076
or MEDIVERSAL 1-800-331-4952 Dept. #135
EQUIPMENT LEASING—$10K-$10 MILLION
NORTHSTAR FUNDING (212) 323-8076 Apply by Phone

MISCELLANEOUS—CONT'D

HAVE YOU RECEIVED ANY COMMUNICATION FROM THE OFFICE OF PROFESSIONAL MEDICAL CONDUCT? If affirmative, contact Susan Kaplan, Attorney-at-Law, (212) 877-5998. Practice limited to the crucial legal problems directly affecting your license to practice medicine—with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professions), and as an Assistant District Attorney in Nassau County. Susan Kaplan, Esq., 165 West End Avenue, Suite 27P, New York, N.Y. 10023.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta, GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

ADVICE, COUNSEL & REPRESENTATION of physicians and other health care professionals in licensure and professional conduct matters, audit preparations, reimbursement issues and practice-related litigation. David E. Ruck, Esq., former Chief, Criminal Division, Office of the New York State Special Prosecutor for Medicaid Fraud Control and Alain M. Bourgeois, Esq., former Acting Justice, Supreme Court of the State of New York. Bourgeois & Ruck, 101 Park Avenue, New York, New York 10178. Telephone 212-661-8070.

PORTABLE RADIOLOGY SERVICE: abdominal + pelvic ultrasound, cardiac echos including 2D + m-mode portable x-rays and portable osteoporosis bone densitometry screening. NYC and suburbs. Schedule weekly/bi-weekly in your office or visits to your patient's home. New portable hi-tech equipment. Hi-quality like standard office images, far superior to hospital portable images. No lead shielding needed for x-rays. University Radiology Service, (212) 265-7392 or (718) 339-5363.

MISCELLANEOUS—CONT'D

MANHATTAN FEB. 1-5. Review Course for the 1988 ABFP/ABIM Geriatric Medicine Examination. Cat 1 AMA Credit 39 hours AFFP prescribed hours 39. Information and registration: Page and William Black Post Graduate School of Medicine of the Mount Sinai School of Medicine, 1 Gustave L. Levy Place, New York, New York 10029, (212) 241-6737.

PROFESSIONAL MISCONDUCT ATTORNEYS. William L. Wood, Jr., formerly Executive Director of the New York State Office of Professional Discipline and Anthony Z. Scher, formerly Director of Prosecutions. Our recent tenure as chief enforcement officers for the regulation of New York State's one-half million licensed professionals has given us experience which allows us to represent physicians in professional misconduct proceedings, malpractice, license restoration, controlled drug proceedings, insurance company reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practitioners. Wood & Scher, Attorneys at Law, One Chase Road, Scarsdale, New York 10583. Telephone (914) 723-3500.

COMPUTER BILLING—SAVE TIME. "The Billing Assistant"—Remarkably easy-to-use software automatically prints insurance forms, bills, complete reports. Menu-driven. Only $399. Free information. Demonstration disk and manual $22. IBM & compatibles. Call or write REM Systems, Inc., Dept. N, 70 Haven Ave., NY 10032, (212) 740-0391. VISA, MC accepted.

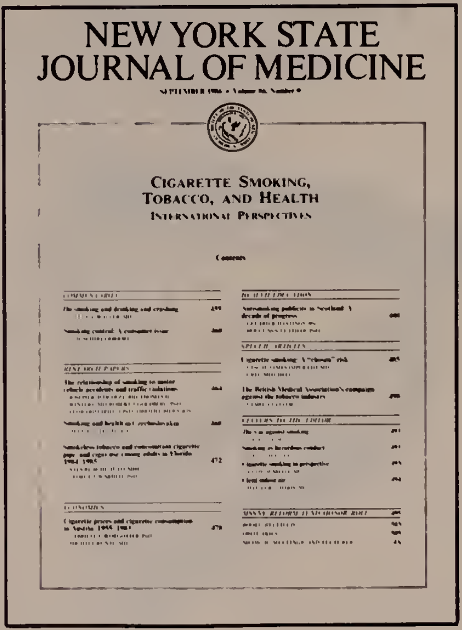
PROFESSIONAL MISCONDUCT ADVOCACY with dignity and concern. George Weinbaum, Esq. former deputy director of the New York State Attorney General's office for Medicaid Fraud Control and also former Director of Medicare and private insurance investigations for Empire Blue Cross/Blue Shield is pleased to announce the opening of his office for the practice of health law. Attention is devoted to misconduct avoidance, disciplinary proceedings, reimbursement maximization, controlled drug issues and medical staff representation among other physician needs. George Weinbaum, Esq., 3 Barker Avenue, White Plains, New York 10601. Telephone (914) 686-9310 office hours, (212) 621-7776-24 hours.

Cover to cover . . . . always good reading.

NEW YORK STATE
JOURNAL OF MEDICINE

MEDICAL MALPRACTICE
AN OVERVIEW

Contents



NEW YORK STATE
JOURNAL OF MEDICINE

CIGARETTE SMOKING,
TOBACCO, AND HEALTH
INTERNATIONAL PERSPECTIVES

Contents

NEW YORK STATE
JOURNAL OF MEDICINE

Contents

NEW YORK STATE
JOURNAL OF MEDICINE

Contents

In 1988, the *New York State Journal of Medicine* promises to provide timely and stimulating commentary, challenging research papers, analysis of clinical trends, debate on topical issues, and in-depth coverage of medical news. The journal will make a welcome gift to a colleague or student in the United States or abroad.

A free copy of the July 1985 issue, a special issue on smoking, will be included with each subscription while the supply lasts.

SUBSCRIPTION ORDER FORM   Clip and mail

Circulation Department
New York State Journal of Medicine
420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042

Name____

Address______________________________________

____

	1 Year	2 Years	3 Years
U.S. & Canada	☐ $30	☐ $55	☐ $75
Foreign	☐ $35	☐ $65	☐ $85

Payment required with order.

As American as...

It's true, our Consumer Information Catalog is filled with booklets that can answer the questions American consumers ask most.

To satisfy every appetite, the Consumer Information Center puts together this helpful Catalog quarterly containing more than 200 federal publications you can order. It's free, and so are almost half of the booklets it lists. Subjects like nutrition, money management, health and federal benefits help you make the right choices and decisions.

So get a slice of American opportunity. Write today for your free Catalog:

Consumer
Information Center
Department AP
Pueblo, Colorado 81009

A public service of this publication
and the Consumer Information Center
of the U S General Services Administration

Doctor . .

Do you, or a physician you know, have a problem with alcohol, drugs, or emotional illness?

If so, please contact the Medical Society of the State of New York's Committee for Physicians' Health, whose function is to assist, *confidentially.*

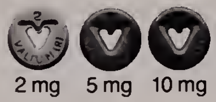
Telephone: 516-488-7777.

Index to Advertisers

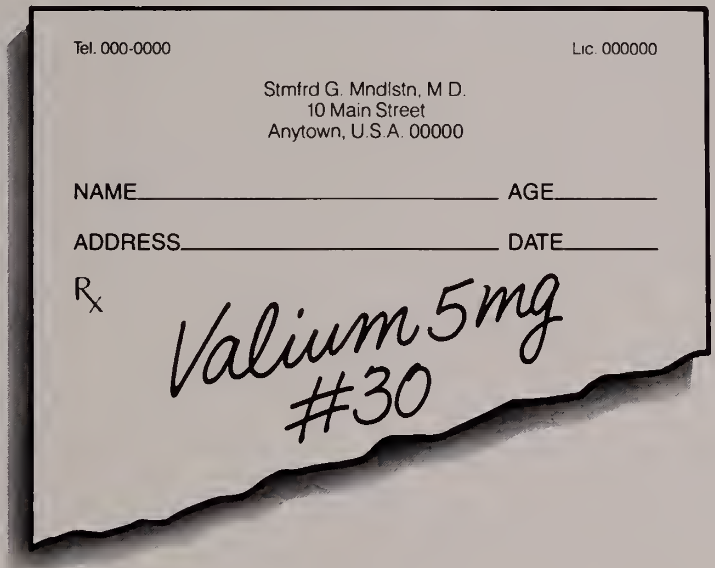
Classified Advertising	6A, 8A, 14A, 16A
Family Practice Recertification Magazine	4A
Eli Lilly and Company	1A
Milstein Properties Corp.	8A
Roche Laboratories	6A, 7A, 3rd & 4th Cover
Sarantos and Company, Inc.	15A
SK&F Company	3A
Upjohn Company	2nd Cover
U.S. Air Force	14A

Do Not Substitute

VALIUM®
brand of
diazepam/Roche
(IV)



The One You Know Best


Tel. 000-0000 Lic. 000000

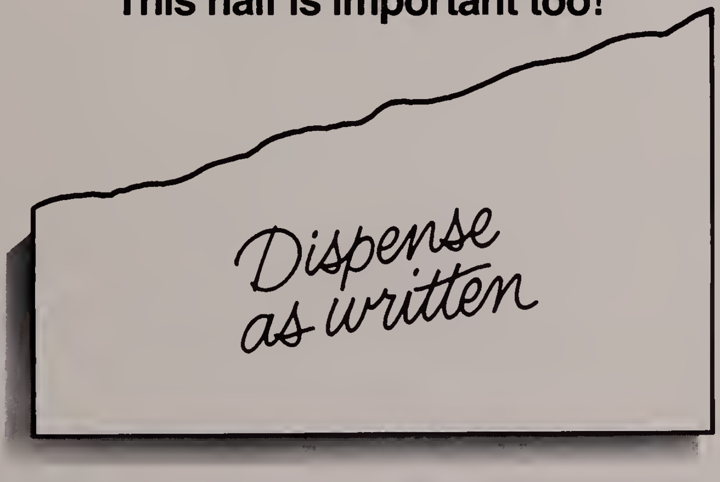
Stmfrd G. Mndlstn, M.D.
10 Main Street
Anytown, U.S.A. 00000

NAME______________________ AGE________
ADDRESS___________________ DATE_______

℞
Valium 5mg
#30

Are you writing only half a prescription for it?

This half is important too!


Dispense
as written

Be sure to write a complete prescription.

Specify "Dispense as written."

ROCHE®
Roche Products Inc.
Manati, Puerto Rico 00701

Copyright © 1987 by Roche Products Inc.
All rights reserved.

State flag of New York

Flag It.

To complete your prescription,
be sure to specify
"Dispense As Written."
This "flags" both pharmacist and patient
that you want the *brand* to be dispensed.
And it protects your decision.

VALIUM®
brand of
diazepam/Roche Ⓘⓥ

scored tablets

The one you know best.

The cut out "V" design is a registered trademark
of Roche Products Inc.

ROCHE®